Review Article

Trends in Agriculutre Science

Vol.2, Issue 1, January, 2023, Page: 13-19

Lumpy Skin Disease: A Review

Shubham Kumar[1], Dhaval J Kamothi[2]*, Manju Gari[3], Anshuk Sharma[4], Meemansha Sharma[5], Thakur Uttam Singh[6], Dinesh Kumar[7]

[1]Ph.D. Scholar, Division of Pharmacology and Toxicology, ICAR-Indian Veterinary Research Institute, Izatnagar, Bareilly, U.P., 243122
[2]Ph.D. Scholar, Division of Pharmacology and Toxicology, ICAR-Indian Veterinary Research Institute, Izatnagar, Bareilly, U.P., 243122
[3]Ph.D. Scholar, Division of Pharmacology and Toxicology, ICAR-Indian Veterinary Research Institute, Izatnagar, Bareilly, U.P., 243122
[4]Scientist, Division of Pharmacology and Toxicology, ICAR-Indian Veterinary Research Institute, Izatnagar, Bareilly, U.P., 243122
[5]Scientist, Division of Pharmacology and Toxicology, ICAR-Indian Veterinary Research Institute, Izatnagar, Bareilly, U.P., 243122
[6]Senior Scientist, Division of Pharmacology and Toxicology, ICAR-Indian Veterinary Research Institute, Izatnagar, Bareilly, U.P., 243122
[7]Principal Scientist & Head, Division of Pharmacology & Toxicology, ICAR-Indian Veterinary Research Institute, Izatnagar, 243122, U.P, India
https://doi.org/10.5281/zenodo.7548336

Abstract

Lumpy skin disease is a disease caused by the capri pox virus belonging to the family pox viridae. The disease has recently created havoc in the livestock sector by causing high mortality in cattle. This disease was first reported in India on 18th of November, 2019 in Odisha. The disease was responsible for great economic loss in terms of mortality, decreased milk production and other dairy products. The diagnosis of the disease was carried out by viral isolation and confirmation was made using different molecular techniques such as the fluorescent antibody test, polymerase chain reaction, ELISA and other techniques. The treatment protocol followed consisted of the conventional and ethno-veterinary approach as per the guidelines of the national dairy development board (NDDB). Through different approaches, efforts were made to control the transmission of the virus and improve the health of the livestock.

Keyword: Lumpy skin disease, Capri pox virus, Fluorescence antibody test, Sit fast.

Introduction

The diseases such as lumpy skin disease virus (LSDV), sheep pox, goat pox are all caused by Capri pox virus belonging to the family Poxviridae. Lumpy skin disease virus causes great economic loss in the livestock sector due to decreased weight gain, permanent damage to hides, decreased milk production, and infertility, usually associated with high morbidity and low mortality (Coetzer, 2004; Hunter and Wallace, 2001). The prototype strain of LSD is known as the Neethling poxvirus (OIE, 2002 and Coetzer *et al*., 2018). Large variability can be seen in the clinical presentation of LSDV infection, which can range from sub-clinical illness to death. Common signs include fever, development of skin nodules on the neck, back, perineum, tail, hind legs, and reproductive organs, growth of superficial lymph nodes, oedema of the limbs and brisket along with lameness.

LSD in India

On 18th of November, 2019, Office International des Epizooties (OIE) received a report of the first LSD case in India (OIE, 2019). According to reports, LSD was first identified in the Indian state of Odisha in August 2019. (FAO, 2020). LSD affected 182 of the 2539 animals that were presented in 2020 (Sudhakar *et al*., 2020), and Madhya Pradesh and Maharashtra were the two states where the disease was most common in the previous year. LSD cases were also reported in the Thrissur and Pallakad in October 2019, and the disease was first confirmed in Kerala (Goud and Vijaykumar, 2020). LSD has expanded to various Indian states, including Kerala, Tamil Nadu, Andhra Pradesh, Telangana, Odisha, Jharkhand, West Bengal, Assam, Chhattisgarh, Maharashtra, and Madhya Pradesh (Kumar *et al*., 2021). Lumpy skin disease has been found in local cattle and Asian water buffaloes near tiger reserves in Madhya Pradesh's central Indian highlands (Pandey *et al*., 2021)

Etiology

Lumpy skin disease (LSD) caused by lumpy skin disease virus (LSDV) belongs to the genus Capripoxvirus, subfamily Chordo poxvirinae, and family Poxviridae. Cattle of all ages are vulnerable to LSDV infection (Weiss, 1968), while cows have been moderately affected while their calves acquired more characteristic and severe lesions 24 to 48 hours sooner than the adult cattle (Le Roux, 1945). Exotic cattle, particularly those with thin skin like Friesians, as well as other high-yielding European dairy breeds have been documented to have a more severe symptom. High milk-yielding cattle appear to be more severely infected (Coetzer *et al*., 2018)

Transmission

LSDV's precise route of transmission is unknown, but it has been suggested that arthropod vectors may spread the disease (Gupta *et al*., 2020). In lab testing, it has been demonstrated that three

African hard tick species *Rhipicephalus (Boophilus) decoloratus* (blue tick), *R. appendiculatus* (brown ear tick) and *Amblyomma hebraeum* transmit the virus. Given that the illness has been experimentally transmitted through contaminated saliva, contact infection is seen as a different way to contract the virus. LSDV can be mechanically transmitted from infected to susceptible cattle by female *Aedes aegypti* mosquitoes and sheep head flies. Mosquitoes feeding on LSDV-infected bovine lesions were able to transmit virus to susceptible cattle for 2-6 days after infection (Wallace *et al*., 2006). Similar to this, it was discovered that female *R. decoloratus* were able to infect cattle by passing the virus to their larvae through their eggs (Coetzer *et al*., 2018). The lumpy skin disease virus and viral antigen were found in tick saliva as well as other organs such as haemocytes, salivary glands, and midgut (Lubinga *et al*., 2014). The LSDV has been isolated from the semen of inapparent affected bulls (Weiss, 1968, Sudhakar *et al*., 2020 and Tuppurainen *et al*., 2012). Experimental evidence of LSDV transmission by artificial insemination has also been found (Annandale *et al*., 2014). LSDV transmission has been observed when common drinking and feeding troughs were used, confirming the notion that infected saliva may contribute to spread of disease (Coetzer *et al*., 2018). The disease is transmitted to suckling calves through contaminated milk, and affected cows have been known to give birth to calves with skin lesions (Coetzer *et al*., 2018). The transmission is largely by indirect contact, most likely by flying, blood-sucking insects, and has significant implications for LSD management (Magori-Cohen *et al*., 2012). Since most LSD outbreaks occur in the summer, when arthro pod activity is at its peak, it is possible that a variety of vector species, primarily blood-feeding insects, are involved in the spread of the virus.

Clinical signs

The incubation time for a disease varies from 4 to 14 days under experimental circumstances, while it is 2 to 5 weeks under natural infection (Carn and Kitching, 1995; Tuppuraine *et al*., 2005). Lacrimation, increased nasal and pharyngeal secretions, anorexia, dysagalactia, general depression, and a disinclination to move is observed in infected animals. The severity of the initial clinical indications of LSD varies depending on the herd management system but is unrelated to animal sex or age. In the skin of the animals, several firms circumscribed nodules appears. Within 1-2 days, these nodules explode unexpectedly. The erupting nodules could be widespread or limited to a few lesions. Skin lesions on the head, neck, udder, genitalia, perineum, and legs are prevalent. The affected skin is hyperaemic, and there may be beads of serum exuding from it. Skin nodules all over the body, temperature (which may surpass 41°C) that lasts 6 to 72 hours, lacrimal discharge, nasal discharge, anorexia, decreased milk yield, emaciation, depression, and a reluctance to move are some symptoms

that may accompany the illness. Skin nodules on the muzzle, nares, back, limbs, scrotum, udder, perineum, eyelids, ears, nasal mucosa, oral mucosa, and tail are observed (Salib and Osman, 2011). Movement may be hampered by edematous and inflamed swellings of the face, brisket, and one or more limbs. Infected animals may occasionally display unilateral or bilateral keratitis **(**Jameel, 2016). Sloughing lesions resulted in "sitfast" hole-shaped lesions, which are the typical lesions is invaded screwworm fly invasion and bacterial invasion, both of which can worsen the condition and lead to septicaemia (Constable *et al*., 2017). Females infected with LSD may experience anestrus for months or become sterile as a result of poor physical condition and other stressors. Pregnant animals that are impacted may abort in some situations. Infected bulls may develop orchitis, which can progress to sterility.

Diagnosis

The diagnosis of LSD is based on typical clinical signs combined with laboratory confirmation of the presence of the virus or antigen. Field presumptive diagnosis of LSD can be based upon the morbidity, mortality and clinical signs that reflect LSD such as contagious illness characterized by widespread cutaneous nodules, inverted conical necrosis of skin nodules (sitfast), as well as enlargement of lymph nodes draining from affected areas. Mucous membranes of the mouth, pharynx, epiglottis, tongue, and throughout the digestive tract, nasal cavity, trachea, and lungs are all affected. Oedema with localized lobular atelectasis in the lungs is observed. In extreme cases, pleuritis with expansion of the mediastinal lymph nodes is found. Pox lesions in the testicles and urinary bladder are possibly found. Histopathological features exhibit congestion, hemorrhage, oedema, vasculitis, and necrosis coupled with nodules encompassing all skin layers, subcutaneous tissue, and, in some cases, surrounding musculature is observed. Intracytoplasmic eosinophilic inclusions may be seen in different cells. Confirmative diagnosis of LSD involves isolation of the virus using primary or secondary bovine dermis cells. Fluorescent antibody test has been used to detect the Capripoxvirus antigen. The classic poxvirus virion is identified using electron microscopy, but it cannot be differentiated to the genus or species level (OIE, 2010), and virus isolation using cell culture followed by PCR is required to validate the viral identity (Bowden *et al*., 2009). The precipitating antigen of capripoxvirus has been detected using agar gel immunodiffusion (AGID) technique. Polymerase chain reaction (PCR) and loop-mediated isothermal amplification (LAMP) assay have been used for detection of capripoxviruses with higher sensitivity (Bowden *et al*., 2009; Balinsky *et al*., 2008). LSD is also diagnosed using serological assays

such as the indirect enzyme linked immunosorbent assay (iELISA), western blotting, and the indirect fluorescent antibody test.

Differential diagnosis

There are disorders that cause symptoms similar to LSD. It is critical to establish a definitive diagnosis in order to implement the most effective preventative and control strategies for sensitive herds. The following disorders can be confused with LSD such as Bovine virus diarrhoea/mucosal disease, Pseudo-lumpy-skin disease, Rinderpest, Demodicosis (Demodex), Insect bite allergies, Bovine malignant catarrhal fever, Besnoitiosis, Oncocercariasis

Treatment

Treatment for LSD is carried out by combining both conventional and ethnoveterinary medicine. Conventional therapy includes the use of broad-spectrum antibiotic (Inj. Amoxirum forte @ 10 mg/kg Body weight, I/v); inj. Antipyretic and analgesic (Meloxicam @ 0.1 mg/kg body weight), I/m;); Inj. multivitamin (VIT A @ 1.5 million units I/m; Inj. Tribivet @ 15 ml, I/v;) fluid therapy (Inj. DNS-1000ml, I/v) for 5 days.

Ethnoveterinary medicinal approach involve use of both oral and topical preparations based on the guidelines proposed by NDDB which include development of oral preparations where betel Leaves, black pepper, salt, and jaggery are blended to form a paste and mixed. The preparation is orally administered, one dose every three-hour first day and from second day three doses daily for three weeks. Another preparation consists of garlic, Coriander, Cumin, Tulsi, Bay leaves, Black pepper, Betel leaves, Shallots, Turmeric powder, Chirata leaf powder, sweet basil, Neem leaves, Aegle marmolos leaves and Jaggery which are blended to form a paste.

For the treatment of external wound, Acalypha Indica leaves, Garlic, Neem leaves, Coconut or Sesame oil, turmeric powder, Mehendi leaves and Tulsi leaves are blended and mixed with Coconut or Sesame oil and boiled and allowed to cool. The preparation is applied topically.

Conclusion

Lumpy skin disease caused by the capri pox virus has been responsible for the great economic loss to the country. In order to control and prevent the disease, number of diagnostic techniques have been used and developed. Different treatment strategies have been carried out for the treatment of animals suffering from the disease.

References

Annandale, C. H., Holm, D. E., Ebersohn, K., & Venter, E. H. (2014). Seminal transmission of lumpy skin disease virus in heifers. Transboundary and emerging diseases, 61(5), 443-448.

Balinsky, C. A., Delhon, G. U. S. T. A. V. O., Smoliga, G., Prarat, M. E. L. A. N. I. E., French, R. A., Geary, S. J., ... & Rodriguez, L. L. (2008). Rapid preclinical detection of sheeppox virus by a real-time PCR assay. Journal of clinical microbiology, 46(2), 438-442.

Bowden, T. R., Coupar, B. E., Babiuk, S. L., White, J. R., Boyd, V., Duch, C. J., ... & Boyle, D. B. (2009). Detection of antibodies specific for sheeppox and goatpox viruses using recombinant capripoxvirus antigens in an indirect enzyme-linked immunosorbent assay. Journal of virological methods, 161(1), 19-29.

Carn, V. M., Kitching, R. P., Hammond, J. M., & Chand, P. (1994). Use of a recombinant antigen in an indirect ELISA for detecting bovine antibody to capripoxvirus. Journal of virological methods, 49(3), 285-294.

Coetzer, J. A. W., 2004: Lumpy skin disease. In: Coetzer, J. A. W. and R. C. Tustin, eds, Infectious Diseases of Livestock, pp. 1268–1276. Oxford University Press, Cape Town.

Coetzer, J.A.W, Tuppurainen, E., Babiuk, S. and Wallace, D. (2018) Lumpy Skin Disease. In book: Infectious Diseases of Livestock, Part II Publisher: Anipedia.

Constable, P. D., Hinchcliff, K. W., Done, S. H., & Grünberg, W. (2016). Veterinary medicine: a textbook of the diseases of cattle, horses, sheep, pigs and goats. Elsevier Health Sciences.

Goud, K. S., & Vijayakumar, K. (2020). Lumpy Skin Disease in Large Ruminants-An Emerging Threat. Intas Polivet, 21(1), 1-8.

Gupta, T., Patial, V., Bali, D., Angaria, S., Sharma, M., & Chahota, R. (2020). A review: Lumpy skin disease and its emergence in India. Veterinary research communications, 44(3), 111-118.

Hunter, P., & Wallace, D. (2001). Lumpy skin disease in southern Africa: a review of the disease and aspects of control. Journal of the South African Veterinary Association, 72(2), 68-71.

Jameel, G. H. (2016). Determination of complications decrease the risk factor in Cattle infected by lumpy skin disease virus in diyala province, Iraq. International Journal of Micro Biology, Genetics and Monocular Biology Research, 2, 1-9.

Kitching, R. P., & Mellor, P. S. (1986). Insect transmission of capripoxvirus. Research in Veterinary Science, 40(2), 255-258.

Kumar, N., Chander, Y., Kumar, R., Khandelwal, N., Riyesh, T., Chaudhary, K., ... & Tripathi, B. N. (2021). Isolation and characterization of lumpy skin disease virus from cattle in India. PLoS One, 16(1), e0241022.

Le Roux, P. L. (1945). Notes on the probable cause, prevention and treatments of pseudo-urticaria and associated septic conditions in cattle. Northern Rhodesia Department of Animal Health, Newsletter, 1-4.

Lubinga, J. C. (2013). The role of Rhipicephalus (Boophilus) decoloratus, Rhipicephalus appendiculatus and Amblyomma hebraeum in the transmission of lumpy skin disease virus (Doctoral dissertation, University of Pretoria).

Magori-Cohen, R., Louzoun, Y., Herziger, Y., Oron, E., Arazi, A., Tuppurainen, E., ... & Klement, E. (2012). Mathematical modelling and evaluation of the different routes of transmission of lumpy skin disease virus. Veterinary research, 43(1), 1-13.

OIE Terrestrial Manual. (2010). Lumpy Skin Disease. Chapter 2.4.14. (Available at http: //www.oie. www.oie.int/fileadmin/Home/eng/Health_standards/tahm/2.

OIE, (2002) LUMPY SKIN DISEASE – Aetiology Epidemiology Diagnosis Prevention and Control References. P. 5.

Pandey, N., Hopker, A., Prajapati, G., Rahangdale, N., Gore, K., & Sargison, N. (2022). Observations on presumptive lumpy skin disease in native cattle and Asian water buffaloes around the tiger reserves of the central Indian highlands. New Zealand Veterinary Journal, 70(2), 101-108.

Salib, F. A., & Osman, A. H. (2011). Incidence of lumpy skin disease among Egyptian cattle in Giza Governorate, Egypt. Veterinary world, 4(4).

Sudhakar, S. B., Mishra, N., Kalaiyarasu, S., Jhade, S. K., Hemadri, D., Sood, R., ... & Singh, V. P. (2020). Lumpy skin disease (LSD) outbreaks in cattle in Odisha state, India in August 2019: Epidemiological features and molecular studies. Transboundary and Emerging Diseases, 67(6), 2408-2422.

Tuppurainen, E. S. M., & Oura, C. A. L. (2012). lumpy skin disease: an emerging threat to Europe, the Middle East and Asia. Transboundary and emerging diseases, 59(1), 40-48.

Wallace, D. B., Ellis, C. E., Espach, A., Smith, S. J., Greyling, R. R., & Viljoen, G. J. (2006). Protective immune responses induced by different recombinant vaccine regimes to Rift Valley fever. Vaccine, 24(49-50), 7181-7189.

Weiss KE, (1968). Lumpy skin disease. In: Virology Monographs, Vol. 3. Vienna, Austria; New York, USA: Springer-Verlag, 111-131.