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A Review: Lumpy Skin Disease

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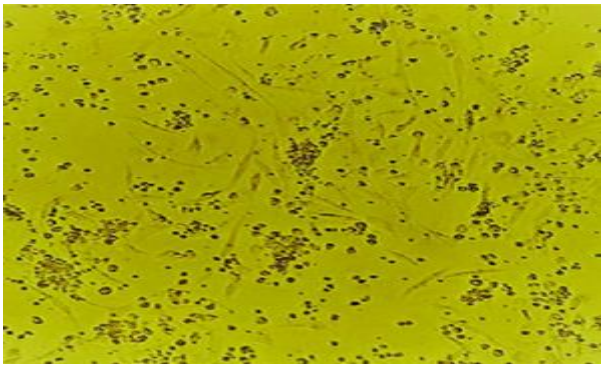
ABSTRACT:
The higher danger of the disease spreading throughout the rest of Asia and Europe should be taken into account. Lumpy skin disease is a developing bovine viral disease that is endemic in most African countries and some Middle Eastern ones. It is critical to comprehend the constraints and channels of dispersion, as evidenced by the recent rapid spread of disease in once disease-free nations. Goatpox and sheeppox can both be brought on by the same agent, Capripoxvirus. Given that they pose a risk to global trade and could be exploited as tools of economic bioterrorism, the economic impact of these diseases is a major issue. Because of the scarcity of reliable vaccines and the widespread poverty in farming communities, capripoxviruses appear to be spreading more widely. This is mainly as a result of the economic consequences of the Global climate change, the COVID-19 pandemic, devastating sanctions imposed in areas where the disease is endemic, as well as an increase in the trade in live animals and animal products, both legally and illegally, are all factors. The goal of this study is to offer current knowledge on the different elements of the illness, including its clinicopathology, transmission, epidemiology, diagnosis, prevention and control strategies, and the probable contribution of wildlife to the disease's spread.
KEYWORDS:
capripox, epidemiology, lumpy skin disease, transboundary disease.

I. INTRODUCTION :

The LSDV, a member of the Poxviridae family of viruses, is the cause of the infectious disease lumpy skin disease (LSD) in cattle. Large domestic ruminants in Asia, Europe, and the Middle East are currently under grave threat from the disease [1]. Due of its ability for rapid cross-border spread, the it has designated the illness [2,3].

Among small-scale and courtyard farmers in endemic locations, LSD is a resurgent transmissible pathogen that significantly harms their socioeconomic situation [4]. When comparing cattle to buffalos and other ruminants, it is discovered that they are more susceptible to the disease in terms of morbidity and mortality [5]. The husbandry of mixed herds of cattle, sheep and goats is a common practise in many nations, although there is currently no proof thatWith the exception of a few laboratory experimental inoculation studies, small ruminants serve as LSDV reservoirs [2,5]. In contrast to native Asian and African ruminants, high producing cattle and Asian water buffaloes frequently exhibit nodular dermatitis [2,6]. The illness is fatal because it causes a sharp decrease in milk production, abortions, poor coat health, and sterility in bulls [7]. If infected animals are transported between farms and quarantine procedures are relaxed, LSDV can spread widely, even across continents [8]. It is noteworthy that there is no epidemiological proof that the illness is zoonotic [9]. The disease remained confined to larger Africa until 1988, when it began to spread gradually to the Middle East, then Eastern Europe, and finally the Russian Federation [10]. TheNew cases were also discovered in South and East Asia in 2019 [1,11], as the outbreak continued to spread. Bangladesh was noted as the first hotspot in South Asia in an OIE study, with the first event occuring on July 14, 2019 [12]. Although no scientific case report of LSD use in buffalo was found in the nation at the time of the investigation. Numerous LSD cases have now been documented in China, India, Nepal, Bhutan, Vietnam, Hong Kong, and Myanmar [13].

Despite the economic significance of LSD, there aren't many studies available on this incredibly deadly arthropod-borne illness in South and East Asian countries [11,14]. The disease has often broken out and reappeared in different places of the world, highlighting theUpdated preventive and adaptive control methods are crucial, as is a reevaluation of the biology of the disease and the viral transmission mechanism. A thorough analysis of LSD has been done, mostly focusing on South-Eastern Asia, taking into account the aforementioned facts.



VIRUS CLASSIFICATION :
Realm: Varidnaviria
Kingdom: Bamfordvirae
Phylum: Nucleocytoviricota
Class: Pokkesviricetes
Order: Chitovirales
Family: Poxviridae
Genus: Capripoxvirus
Species: Lumpy skin disease virus

HISTORY :

In Zambia, a 1929 epidemic of lumpy skin condition was first reported. At first, poisoning or an extreme sensitivity to bug bites were assumed to be the cause. In Botswana, Zimbabwe, and the Republic of South Africa, more cases were reported between 1943 and 1945. In 1949, a panzootic infection in South Africa afflicted about 8 million cattle, which resulted in significant financial losses. Affecting livestock in Kenya, Sudan, Tanzania, Somalia, and Cameroon, LSD expanded throughout Africa between the 1950s and 1980s.[Required Citation]

A LSD epidemic occurred in Israel in 1989. LSD was first detected in this epidemic outside of the African continent and north of the Sahara desert.[15] According to speculation, infected Stomoxys calcitrans was the cause of this outbreak.

coming from Egypt's Ismailiya on the breeze. In Peduyim, fourteen of the seventeen dairy herds contracted the LSD virus during the course of 37 days in August and September 1989.[16] The village's cattle, together with a few small flocks of sheep and goats, were all butchered.[16]

throughout the past ten years, incidents involving LSD have been documented throughout the Middle East, Europe, and west Asia.[15]

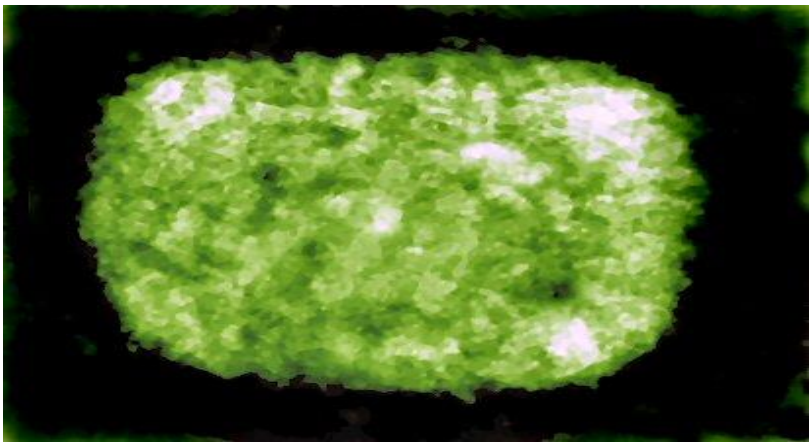
In July 2019, the Bangladesh livestock department received its initial report of LSD.[17] Over time, it was estimated that 500,000 people were affected by this disease.The Food and Agriculture Organisation (FAO) advised widespread immunisation.[17] As a result of the emergence of the autumn armyworm and this cattle disease a short time apart, the FAO, the World Food Programme, Officials from the Bangladeshi government and other parties reached an agreement to start enhancing Bangladesh's emergency preparedness and livestock disease surveillance capacities.[17] The virus's mode of entrance into Bangladesh is yet unknown.[18]

Over 7000 cattle were slaughtered by a lumpy skin disease epidemic in Pakistan in 2022.[19] Over 80,000 cattle died as a result of the lumpy skin disease epidemic in India between July and September 2022.[20]Most of the deaths have occurred in the state of Rajasthan [21].[22] Many states have placed restrictions on the movement of livestock between states and districts.[23][24][25]The development of an indigenous vaccine has been taken on by Indian Council of Agricultural Research laboratories.[26] There is a goat pox vaccine being used; by September 2022, 15 million doses had been given.[27] At least three facilities produce the goat pox vaccinei India.[28][29][30] More institutions now have the ability to provide tests.[31]

CLASSIFICATION OF LUMPY SKIN DISEASE VIRUS :

The virus that causes lumpy skin disease (LSDV) has double-stranded DNA. It belongs to the family Poxviridae and the genus Capripoxvirus. The subfamily of Chordopoxviruses (ChPVs), which consists of eight genera, includes Capripoxviruses (CaPVs).The genus Capripoxvirus includes the LSDV, sheeppox virus, and goatpox virus. Even while CaPV infections are serologically identical, they typically have host specificity within particular geographic distributions.[65]

STRUCTURE



Typical Poxviridae Viruses Brick-Like Structure
Capripoxviruses have a brick-like form like other members of the Poxviridae family. In addition to having a more oval profile and larger lateral bodies, capripoxvirus virions differ from orthopoxvirus virions in this regard. Capripoxvirions typically measure 320 by 260 nanometers.[Needs citation]

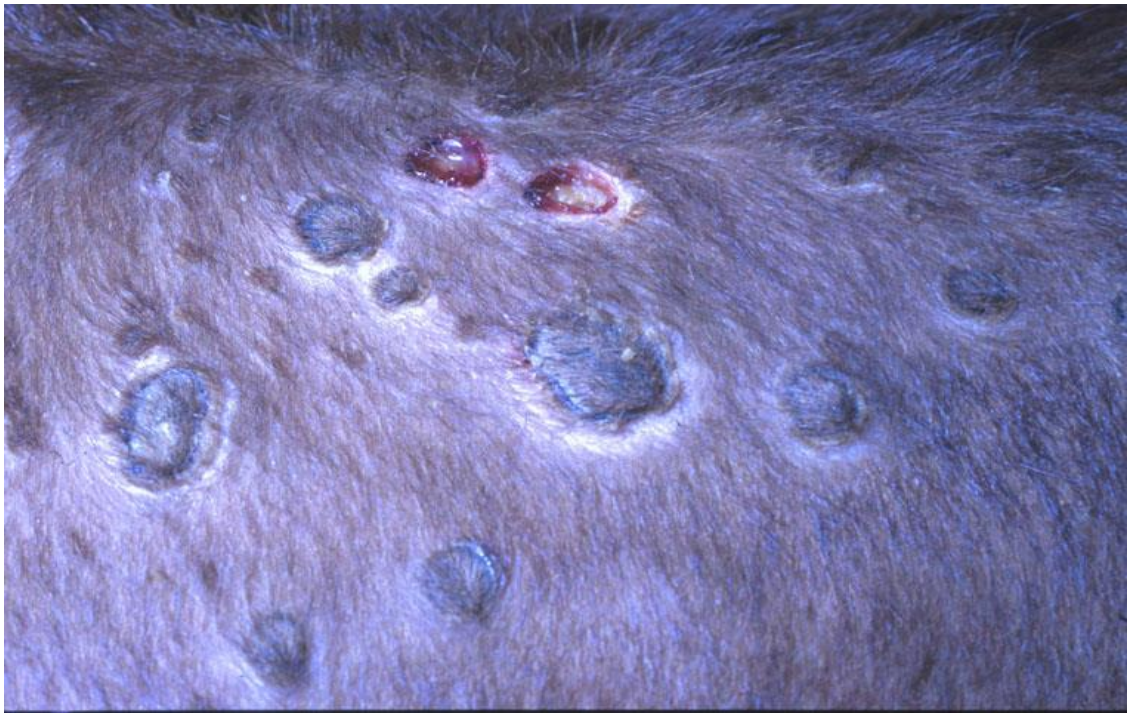
Genome

The 151-kbp virus genome is made up of 156 genes and a core coding region that is surrounded by identical 2.4-kbp-inverted terminal repeats.When comparing LSDV with chordopoxviruses from other genera, there are 146 conserved genes. These genes produce proteins that are involved in the synthesis of mRNA and transcription, DNA replication, nucleotide metabolism, protein processing, virion structure and assembly, viral pathogenicity, and host range.The LSDV genes exhibit extensive collinearity and amino acid identity with the genes of other mammalian poxviruses in the core genomic region.Suipoxvirus, Yatapoxvirus, and Leporipoxvirus are a few examples of viruses with a similar amino acid identity.However, collinearity is broken in terminal regions .Poxvirus homologues are either absent or present at reduced levels in these areas identify an amino acid. The majority of these variations involve genes that are probably connected to viral virulence and host range. In addition to homologues of other poxvirus genera' interleukin-10 (IL-10), IL-1 binding proteins, G protein-coupled CC chemokine receptor, and epidermal growth factor-like protein, LSDV is exclusive to the Chordopoxviridae family.[65]

ETIOLOGY:

The lumpy skin disease (LSD) virus, which is connected to the sheep pox virus, causes LSD. In the subfamily Chordopoxvirinae of the family Poxviridae, LSDV belongs to the genus Capripoxvirus. According to the OIE[42] and the Neethling poxvirus, the original strain of LSD. All age groups and genders of cattle are vulnerable to LSDV infection [43] albeit on occasion cows have only been weakly affected while their calves acquired more characteristic and severe lesions 24 to 48 hours sooner than their dams [44]. Exotic cattle breeds like the Holstein Friesian, particularly those with thin skin like Friesians, and other high-yielding European dairy cattle have been observed to suffer from more severe disease.

breeds. According to[45], cows who are lactating at their peak appear to be more severely impacted. According to research, Holstein Friesian cattle or crossbred cattle are more vulnerable to LSD than zebu cattle [42]



EPIDEMIOLOGY OF LUMPY SKIN DISEASE:

The majority of African nations, especially those in the Sub-Saharan region, are affected by LSD, which is a common disease [32][33]. After 2012, it was extended to South-East Europe, the Balkans, the Caucasus, Russia, and Kazakhstan [34]. Depending on the outbreak, there may be many infected animals, high rates of illness and mortality, or only a small number of reported deaths. However, in general, outbreaks are worse when the infection is first introduced to a place and then they get better after that, probably because a lot of people have developed a resistance to it. In endemic locations, morbidity rates are closer to 20% during epizootics and exceed 80% overall [35].

Hosting and vulnerability

Except for few strains that can reproduce in sheep, LSDV is extremely host-specific (infecting domestic cattle and water buffaloes).

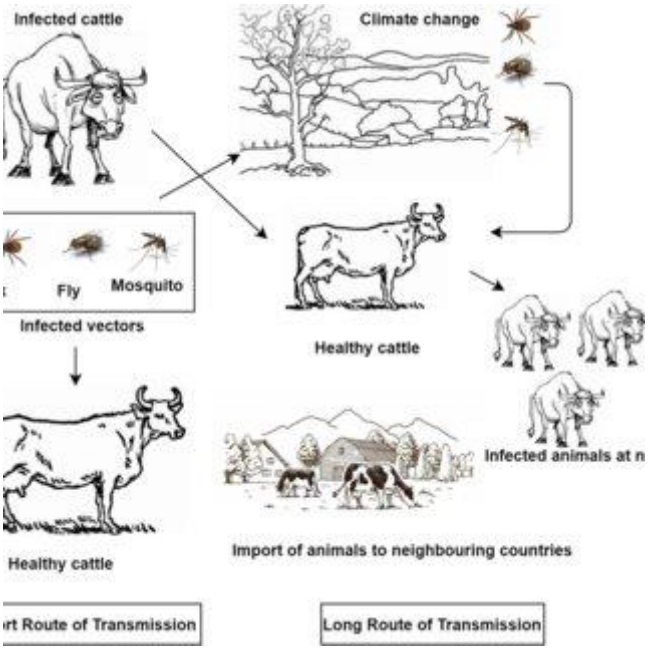
even goats. Small ruminants are virus reservoirs, although epidemiological studies have not yet discovered this [32][36]. The two species most susceptible to LSD epidemics in the wild are domestic cattle and Asian water buffalo [32][37]. Aepyceros melampus and giraffe (Giraffe Camelopardalis) are two examples of wild species that are susceptible to experimental infection, though it is unclear what function they play [38]. The vulnerability of the host animal is governed by its immunological condition, age, and breed rather than the virulence of the virus [39].

The virus's sources

Nodules on the mucous membranes of the mouth, udder, rectum, and genitalia can also ulcerate and release enough viruses to act as sources of the virus. In around half of the infected animals, clinical symptoms may appear; Most experimentally infected animals will go on to contract the virus and become viremic sources of it. Cattle that had been experimentally infected with LSD virus had saliva present for 11 days, semen for 22 days, and skin nodules on 33 days, but not urine or faces [40]. Capripoxviruses have a strong affinity for cutaneous tissues and are particularly resistant to physical and chemical conditions, which allows them to live for a long time in lesions or scabs [41].

MECHANISM:

The systemic illnesses sheeppox, goatpox, and lumpy skin disease all start with cell-associated viremia before lesions and obvious lymphadenopathy occur. Blood monocytes are probably crucial in the virus's ability to disseminate to secondary infection sites.



SIGNS AND SYMPTOMS:

- Four to fourteen days following infection are the incubation period for lumpy skin condition.
- The animal may experience an initial stage of high fever (41°C) and enlarged lymph nodes before forming big, firm nodules in the skin that can measure up to 5 cm in diameter.
- The body can be found with them all over, although they are most noticeable on the:head neck udder scrotum perineum.
- An elevated risk of flystrike may result from the nodules becoming necrotic and ulcerating.
- Cow with a black coat and neck-area skin lumps.a young, brown cow with bumps all over it.
- When milk production is drastically reduced, dairy animals at their peak of production are frequently the most badly impacted. Additional symptoms that could be seen include rhinitis, conjunctivitis, depression, anorexia, and excessive salivation.
- Infected animals exhibit necrotic tissue.
- The most severely impacted dairy cattle typically have a considerable reduction in milk production throughout their peak periods of production. There may also be signs of increased salivation, anorexia, rhinitis, and conjunctivitis.
- Necrotic lesions can form in the gastrointestinal and respiratory tracts of animals who are really ill.
- Up to 50% of cases in an outbreak may be subclinical, and the illness can also be quite serious or even fatal. Mortality rates typically range from 5% to 45%, and morbidity rates range from 5 to 45%, although both rates can be significantly higher when an outbreak affects a community of ignorant cattle.
- Other ailments in cattle that resemble lumpy skin disease
- Other dermatophytes and ringworm infection
- Dermatophilus contamination
- Leukaemia of the skin
- Parapox (popular stomatitis in cattle)
- Herpes mumps in cattle
- Skin with false lumps
- Bovine herpesvirus 2 (fake lumpy skin condition)
- Photosensitisation
- Virus bites
- Demodectic mange, urticaria, burns, and other injuries
- Actinobacillosis (a skin illness caused by Actinobacillus lignieresi)

PATHOLOGY:

Virus replication, viremia, fever, cutaneous localisation of the virus, and nodule growth happen after LSDV infection [45] The following incidents were recorded experimentally following intradermal inoculation of the virus:

Localised swelling at the inoculation site as 1-3 cm nodules or plaques occurs 4–7 days after infection (DPI).

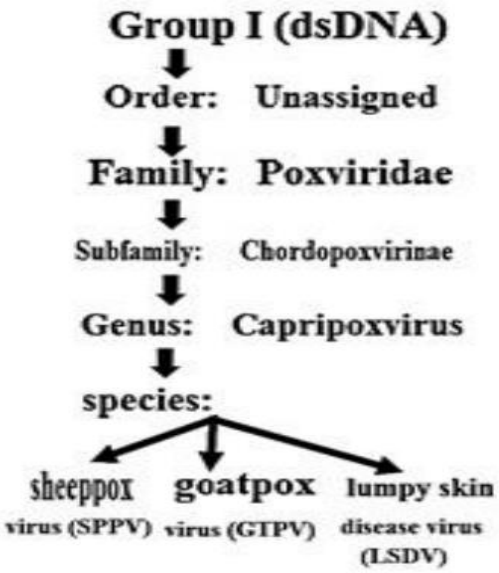
Viral shedding by oral and nasal secretions and viremia between 6 and 18 DP

Regional lymphadenopathy and the emergence of widespread skin nodules from 7 to 19 DPI
There was a virus in the semen 42 days after the illness [46]
Infected tissues experience vasculitis and lymphangitis as a result of the virus' intracellular reproduction in fibroblasts, macrophages, pericytes, and endothelial cells [47]
Young calves, breastfeeding cows, and animals that are overweight appear to be more sensitive
spontaneous infections, most likely as a result of humoral immunity being compromised [48]Animals that have recovered after contracting the virus naturally have displayed permanent immunity. Due to the maternal antibodies they have acquired, calves from infected dams are resistant to clinical illness for about 6 months [49]According to [50]affected animals recover from the infection, and there are currently no known LSDV carrier states.

TRANSMISSION:

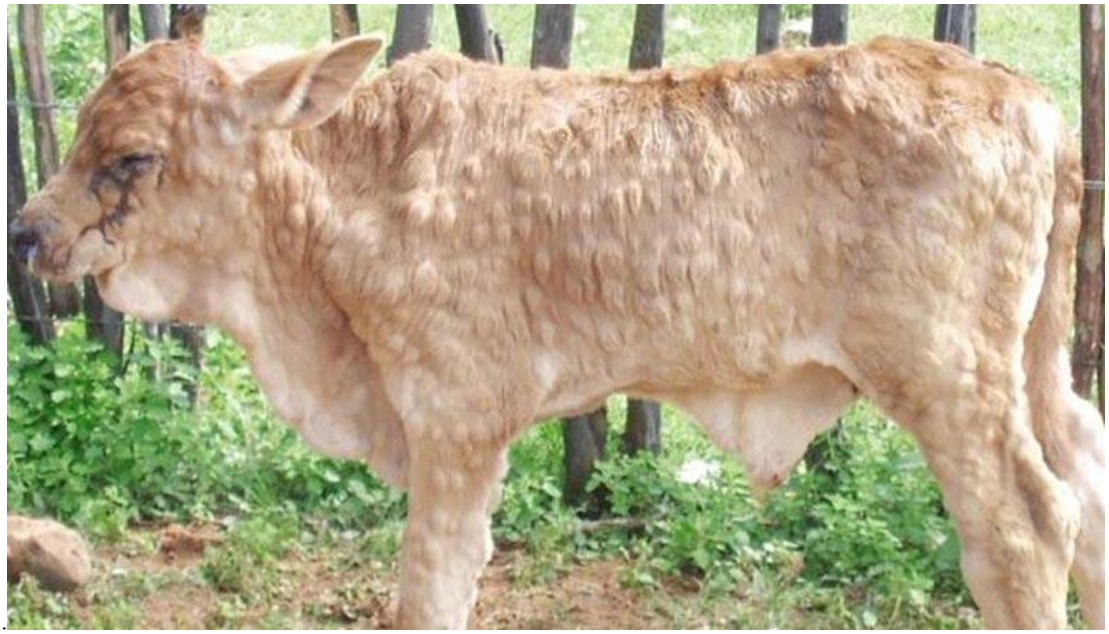
The traditional system of production and seasonal nomadism, in which herds of cattle roam far in search of food and water in dry and semi-arid environments, contribute to the transboundary spread of LSD. The spread of infected animals is most likely how the disease spreads quickly, often appearing hundreds of kilometres from the initial outbreak sites. In the past, it was understood that LSD was primarily spread mechanically through biting arthropods—a sort of vector-borne transmission in which the virus does not replicate within the vector. A typical method of LSDV transmission, according to some researchers, is direct contact without the need of a vector [51]. In recent years, researchers in Russia have researched non-vector-borne transmission. Contact transmission reduces the effect of seasonality, which, in accordance with Aleksandr et al. Regardless of the existence of biting insects, is related to insect activity and increases the likelihood of dissemination. There have also been reports of transmission by tainted feed and water and direct transmission through saliva, nasal secretions, and semen in the later stages of the illness [52][53][54][55] Because the virus stays long in lesions or scabs, skin lesions are the main source of infection. Additionally, the virus is expelled through the blood, saliva, milk, semen, and nasal and lachrymal secretions [53].

Stable flies (Stomoxys calcitrans), mosquitoes (Aedes aegypti), and hard ticks (Rhipicephalus and Amblyomma species) are the blood-sucking arthropods most likely to transmit the LSDV [53]. Horse flies, biting flies S. calcitrans, Stomoxys sitiens, and other Haematopota species have all been used in experiments. Cattle can also catch the sickness from indica [56]. A house fly, Musca domestica, may also contribute to the transmission of LSDV, claim Sprygin et al. [57].



TYPES OF LUMPY SKIN DISEASE:

Ipomas are fatty, squishy lumps that develop beneath the skin.
Cysts: fluid-filled sacs that can form on the body wherever
Glands that are swollen: enlarged lymph nodes that may be signs of infection or inflammation
Hard, reddish, or brownish nodules that develop at the site of a small injury are known as dermatofibromas.
A cherry angioma is a tiny, blood vessel-filled red patch.
Small, flesh-colored growths that hang from the skin are known as skin tags.
Warts are hard, infectious lumps brought on by a virus.
Keratoacanthomas are quickly expanding, dome-shaped tumours that typically appear on sun-exposed skin



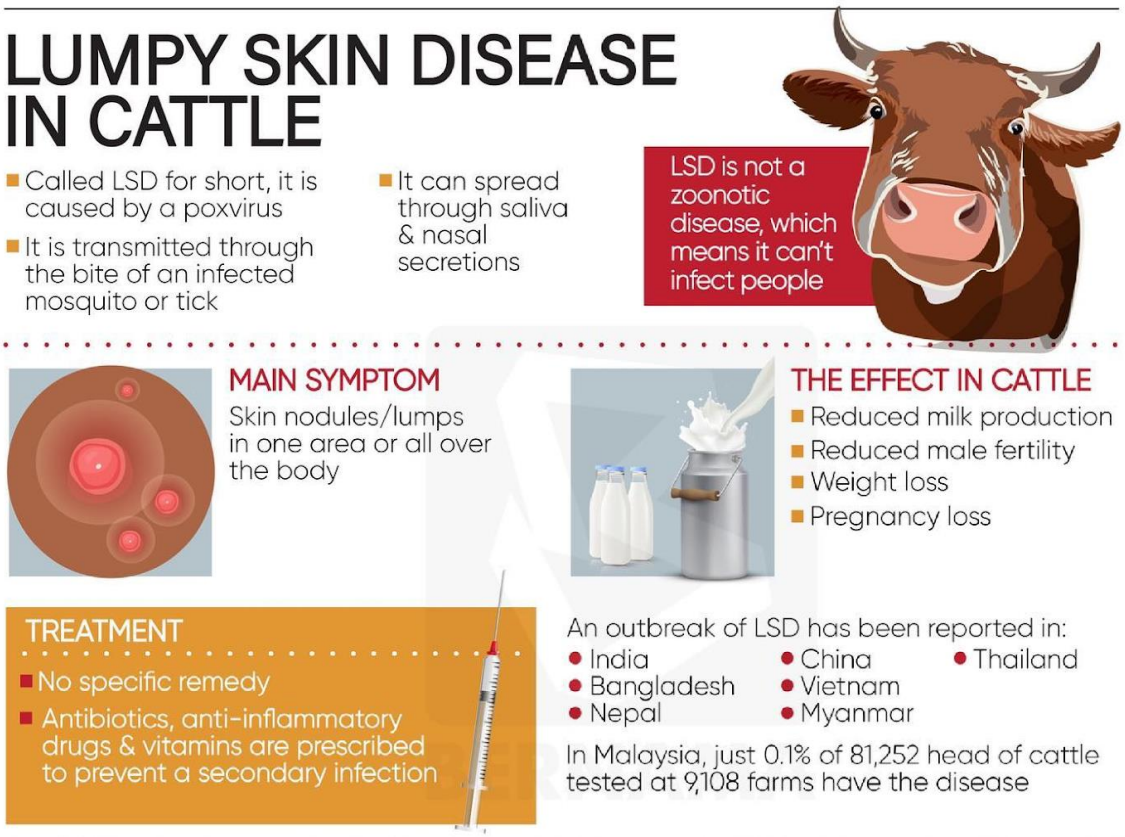
DIAGNOSIS AND TESTING:
A comparison of various diagnostic techniques for the detection of cows with lumpy skin condition In clinically sick, fevered, and otherwise seemingly healthy dairy cows, lumpy skin disease was diagnosed using viral isolation,
-Polymerase chain reaction (PCR)
-Dot blot hybridization (DBH)
-Indirect enzyme-linked immunosorbent assay (iELISA).

TREATMENT:

For cattle infected with LSD, there is no specific antiviral therapy available. In order to reduce fly anxiety and avoid subsequent infections, sick animals may be separated from the herd and provided supportive care, such as local wound dressing. Skin infections, cellulitis, or pneumonia may be treated with systemic antibiotics, and food and drink should always be provided. In an effort to stop future transmission, local insecticide sprays have been made to afflicted animals, but with no apparent success.

All sick and contact cattle should be slaughtered very once, and the carcasses should be destroyed in an effort to get rid of the infection focus, should LSD show up in cattle in a country outside of its prior range. a 25 to 50 km radius immunisation zoneAfterward, a zone could be created around the point of emphasis, and all cattle movements could be stopped there. Alternately, it can be decided to leave all cattle in the area uninfected, enabling any leftover virus to present itself. The vaccination schedule can let the virus hang around in some cattle.

Slaughter laws are improper and large immunisation campaigns are advised when an epizootic occurs in an enzootic area and LSD has already spread widely. Although stringent movement restrictions do not stop LSD outbreaks, they do stop new foci from forming at a certain distance, hence its implementation is advised. Vaccination will significantly lessen the economic and morbid costs of an epizootic, but it may not fully stop LSD from spreading. calves' and lambs' follow-up immunization Revaccination campaigns conducted over a two- to three-year period will significantly lower the incidence of clinical illness.



For the purpose of preventing LSD in African herds of cattle, two different vaccinations have been used extensively and successfully. In tissue cultures made of lamb kidney cells, the Neethling strain of LSD was passed through 50 times before being passed through 20 times in fertilised eggs in southern Africa. Although local reactions do happen in a significant portion of animals at the immunisation site, the strain was shown to be immune- and non-immunogenic for cattle. Its usage has never been followed by a widespread infection. Having been created in tissue culture, [60]

Recently, two different sheep pox vaccination strains have been utilised as a defence against LSD. The Romanian strain, which was used to treat sheep pox in the skin of lambs, was given to several million cattle in Egypt and showed signs of immunogenicity. In Israel, the tissue culture-prepared RM 65 sheep pox strain was employed. The usage of these strains in cattle has not resulted in any difficulties.

An immunising dose of 103.5 TCID50 is preferred for field vaccination campaigns, according to studies with both the Neethling and the Kenya SGPV strains. In the face of an epizootic, 102 has provided good protection, yet thereis evidence that this might not apply to all strains.[61]

Numerous animals resist being challenged with virulent LSD when they lack detectable fluorescence or neutralising antibodies to the virus, according to serological tests with immunised cattle. However, following LSD field infections, the majority of animals do have a serological reaction. Similar to other pox viruses, the immunological response to LSD in cattle has a significant cellular component. and [62]used this as the foundation for the creation of a hypersensitivity test to ascertain the susceptibility of cattle to LSD for use in vaccination research. Determine the reactions to vaccination with this test.

LSD, the Kenya SGPV, the Romanian sheep pox, and the Iraqi Gorgon goat pox haveBy use of fluorescent antibody and serum virus neutralisation assays, it has been established that each one is serologically identical [63] It is therefore conceivable that many of the sheep or goat pox vaccine strains that are readily available around the world would prove effective for the prophylaxis of LSD. However, it is advised to use ten to fifty times the dose given to sheep for immunisation.

The Kenya SGPV and LSD appear to be identical, according on recent restriction enzyme analysis of the genomes of these virus strains. The DNA of the other sheep or goat pox strains does differ from that of LSD [64]but these variations are not reflected in the serological and immunological characteristics of the other strains.

II. CONCLUSION:

Currently, the illness poses a serious threat to small-scale farmers. Greater Africa was the main region of the disease's dissemination up until the eighteenth century. The Middle East, Eastern Europe, the Russian Federation, and most recently Asia were all affected by its migration from there. The scientific community has taken note of the LSDV attacks that occur often in open spaces. In order to prevent the significant spread of this trans-boundary disease, it should be obvious that now is the best time to get ready for emergencies. For the purpose of preventing the invasion and spread of the disease, attention should be given to vector control, movement restrictions, strict quarantines, improved vaccination programmes, proper veterinary treatment, and overall farm sanitary management. Thus, the work promotes additional research on ecology andThe molecular characterisation and determination of the causal agent, as well as the epidemiology of LSDV in India.

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