International Journal of Pharmaceutical Science and Research
www.pharmacyjournal.net
ISSN: 2455-4685
Received: 07-08-2023, Accepted: 22-08-2023, Published: 09-09-2023
Volume 8, Issue 3, 2023, Page No. 21-26

An overview on lumpy skin disease

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Abstract
The lumpy skin disease virus, also known as LSDV, is a member of the Poxviridae family and is responsible for lumpy skin disease (LSD). It is a global disease that affects cattle and water buffaloes and has significant economic ramifications. The disease, which is carried by arthropod vectors, has a high morbidity and a low mortality rate. With a morbidity incidence of 7.1% in cattle, LSD first appeared in India. Fever, anorexia, and unique nodules on the skin and mucous membranes of the mouth, nostrils, udder, genital, and rectum are the usual clinical manifestations of the illness. Infertility, abortion, and occasionally death are other possibilities. Africa and the Middle East are home to the disease, but it has only just started to spread to extended to other continents, including Asia. It has recently been covered in Chinese media, and Bangladesh and India share a border. We have created a list of LSD epidemics that have happened in Asian countries over the previous ten years for the first time. In India, the epidemiological condition of the disease is still unknown. By immunizing people, enforcing strict quarantine guidelines, and taking vector management precautions, it would be able to stop the disease from spreading. This study attempts to describe the most recent developments in epidemiology with a focus on transboundary dispersion, aetiology and transmission, clinical symptoms, diagnostics, and sickness treatment.

Keywords: Transboundary, aetiolgy, itrogenic, collinerity

Introduction
Lumpy skin disease is an infectious viral illness brought on by the Lumpy skin disease virus (LSDV), which belongs to the family Poxviridae, subfamily Chordopoxvirniae, and genus Capripoxvirus. According to Al-Salihi (2014) [16] and Tuppurainen *et al*. (2017) [9], the illness is also referred to as "LSD," "Pseudo-urticarial ia," "Neethling virus disease," "exanthema nodularis bovis," and "knopvelsiekte." The host range of LSD, a non-zoonotic, vector-borne, and transboundary illness, is now only restricted to ruminants, specifically cattle and water buffaloes. Biting flies, mosquitoes, and ticks are the arthropod vectors that carry disease (Tuppurainen *et al*. 2011; Lubinga *et al*. 2013a, b) [15]. Despite being in close proximity to sick cattle and buffaloes, natural sheep and goat infection has not been observed; nonetheless, skin lesions have appeared following experimental infection in sheep, goats, giraffes, impalas, and giant gazelles (Davies, 1991). According to Butarbush *et al*. (2013), LSD is associated with substantial morbidity but low death. Fever, swollen lymph nodes, small nodules on the skin that cause severe emaciation, decreased milk production, and infertility are the disease's hallmarks. Overall, it has an impact on the economic value of animals because it will have an impact on their ability to reproduce effectively (via abortion and (Irregular Pregnancy; RGBE 2014). It is a disease that must be reported and has a terrible impact on international cattle trade as well. Although the illness is indigenous to African nations, it has recently been discovered in previously undiscovered regions of the globe. Morris (1931) said that the first LSD case originated in Zambia and spread to other nations in southern and northern Africa after that. Israel, Kuwait, Oman, and Yemen afterwards caught the virus (Wainwright *et al*. 2013). The OIE reports that this disease is currently widespread in a number of African, European, and Asian nations (Tuppurainen *et al*. 2015). The disease may have reached India due to the movement of animals across international boundaries or the movement of disease-carrying vectors from the surrounding nations. LSD has recently been reported from nations bordering India, including China and Bangladesh. For appropriate planning of the effective illness management, an understanding of the epidemiology of otic diseases becomes essential. The most recent LSD developments are summarized in this overview.

Lumpy skin disease
Aetiology
Cause of lumpy skin condition, the LSDV Lumpy skin disease is a member of the Poxviridae family, which also includes viruses that cause disease in all domestic animals except dogs. According to Quinn *et al*. (2016), the family is divided into two subfamilies: Entomopoxvirinae, which infects invertebrate hosts, and Chordopoxvirinae, which infects vertebrate hosts. Ten genera, including the Capripoxvirus genus, are included in the Chordopoxvirinae subfamily. According to King *et al*. (2012), this genus contains viruses from three different species, including lumpy skin disease virus (LSDV), goatpox virus, and sheeppox virus. These viruses affect sheep, goats, and cattle, respectively. LSDV is a brick-shaped encapsulated virus with double-stranded DNA that has complex symmetry and replicates in the cytoplasm of the host cell. It is 320 260 nm in size. The 151 kbp-long LSDV genome contains a core coding region and two 156 putative genes are located in a region surrounded by identical 2.4 kbp-inverted terminal repeats. LSDV has 30 structural and non-structural genes that are 97% nucleotide identical to sheeppox and goatpox viruses (Tulman *et al*. 2001, 2002). In later evolution, gene loss restricts the host range of poxviruses, and the same pattern has been seen within Capripoxviruses when

comparing SPPV, GTPV, and LSDV. Nine genes are encoded by the terminal portions of the LSDV virus, including the IL-1 receptor, the vaccinia virus F11L, N2L, and K7L genes, the myxoma virus M003.2 and M004.1, and the LSDV132 unique gene, whose host range and virulence functions are likely compromised by accumulating mutations in both SPPV and GTPV. The three viruses' genome sequence length is unaffected by this disruption, but the absence of these genes does In SPPV and GTPV, the host restriction to only cattle is suggested (Tulman *et al*. 2002; Biswas *et al*. 2019). Comparatively speaking to other Chordopoxviruses, the LSD virus has 146 considered genes that encode proteins involved in DNA replication, transcription, mRNA synthesis, nucleotide metabolism, structural creation and stability, virulence, and host range. The core area genes, particularly those from Suipox, Leporipox, and Yatapox viruses, have an average 65% collinearity and amino acid identity with genes from other poxviruses. With either absence or disruption, the genes implicated in viral virulence and host range differ, sharing a reduced percentage of amino acid identity—only 43% on average—in the terminal region. LSDV has homologous genes for interleukin-10 (IL 10), IL-1 binding proteins, and other molecules. Other poxvirus genera have the G protein-coupled CC chemokine receptor (GPCR) and an epidermal growth factor-like protein (Tulman *et al*. 2001).

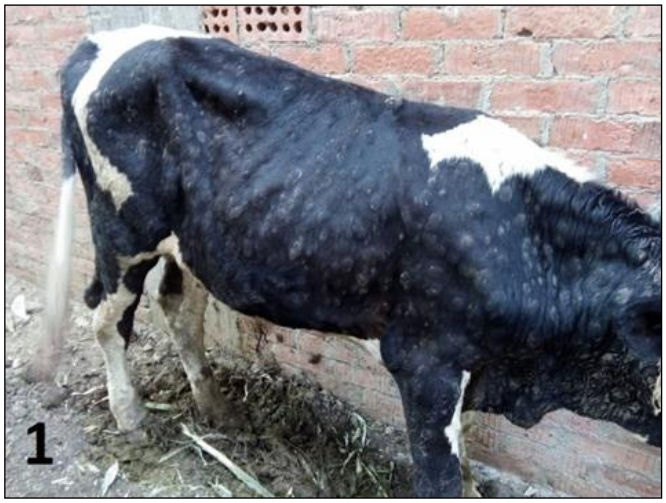

Fig 1: Infected cattle

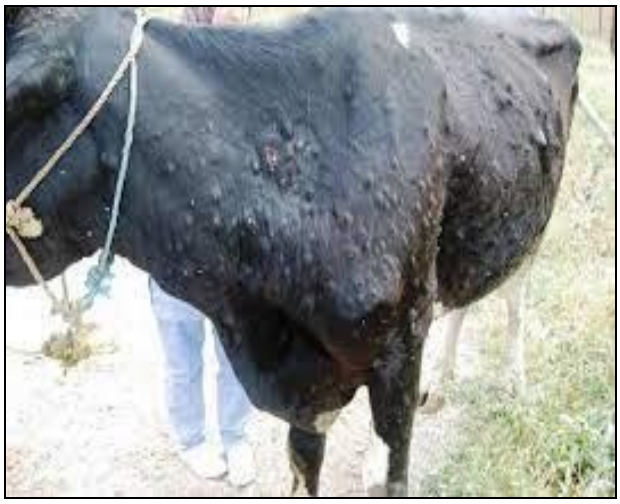

Fig 2: Infected cattle

Stability of virus

The virus can last a long time under ambient circumstances. It can survive for 35 days in dry skin crusts, 33 days in necrotic nodules, and at least 18 days in air-dried hides.

Viruses can be promptly destroyed by sunlight and lipid detergents, but they can survive for several months in dim environments like feed warehouses and animal shelters. The virus becomes inactive at temperatures of 65oC for 30 minutes and 55oC for 2 hours. Although it is sensitive to extremely alkaline or acidic pH, it can maintain pH 6.6 to 8.6 for five days at 37°C without suffering a major loss in titres. Ether (20%), chloroform (1%), formalin (1%), phenol (2% for 15 minutes), sodium hypochlorite (2-4%), iodine compounds (1:33 dilution), and quaternary ammonium compounds (0.5%) are all capable of killing the virus (OIE 2013). According to Mulatu and Feyisa (2018), LSDV is extremely stable and can be retrieved even after 10 years from skin nodules preserved at -80°C and after 6 months from infected tissue culture fluid held at 4°C.

Transmission

The main method of disease transmission is by mechanical transfer by vectors. The arrival of the seasonal rains and the summer months, which coincide with the peak activity of the vectors, dramatically increase disease rates in the majority of the endemic countries, such as sub-Saharan Africa, Egypt, and Ethiopia (Mulatu and Feyisa 2018). With the coming of winter, incidents significantly drop before resuming in the spring and summer. The virus was found to spread to Israel, 80 to 200 km away, through air movement of biting insects, despite Egypt's restrictions on animal migration (AU-IBAR 2013). The decrease in incidence during dry conditions with no insects or a low insect density has established the involvement of insect vectors in disease transmission rather than via direct or indirect contact with infected individuals. These are regarded as inefficient pathways (Gumbe 2018; Carn and Kitching 1995) [6] because they involve indirect touch (Nawathe *et al*. 1982; Kondela *et al*. 1984). As mechanical vectors and reservoirs of virus, the tick Amblyomma spp., Rhipicephalus decoloratus, Rhipicephalus appendiculatus, and Amblyomma hebraeum have been identified (Ali and Obeid 1977; Lubinga *et al*. 2013a, b; Lubinga 2014; Tuppurainen *et al*. 2013a, b) [13, 15]. Additionally, mosquitoes like Culex mirificens and Aedes natrionus as well as biting flies like Stomoxy calictrans and Biomyia fasciata are implicated in the mechanical transmission of disease. Although there is little concrete evidence of LSDV direct transmission, laboratory research and field observations by Weiss in 1968 support a low rate of direct transmission. While studies have found that direct cruelty to animals has no viral transmission (Magori-Cohen *et al*. 2012; Carn and Kitching 1995). A virus As an indirect source of infection for animals sharing feeding and watering troughs, bacteria are released through milk, nasal secretions, saliva, blood, and lachrymal secretions (Ali *et al*. 2012) [13]. The literature has described LSD virus transmission via the intrauterine pathway (Rouby and Aboulsoud 2016) [14]. According to Tupurainen *et al*. (2017), the infection is thought to be passed from an infected mother to her calf by milk secretions and skin abrasions. The virus has been proven by experimental infection (Annandale *et al*. 2013) [10], and it can stay in the

semen for up to 42 days after infection (Irons *et al*. 2005). When a single needle is used for mass vaccination and the virus is picked up from skin crusts or scabs, this is known as the iatrogenic pathway (Mulatu and Feyisa 2018). Depicts a summary of virus transmission. As a result, it implies that isolation may not be the primary strategy to stop the spread of LSD because vector movement can transmit the illness (EFSA 2015).

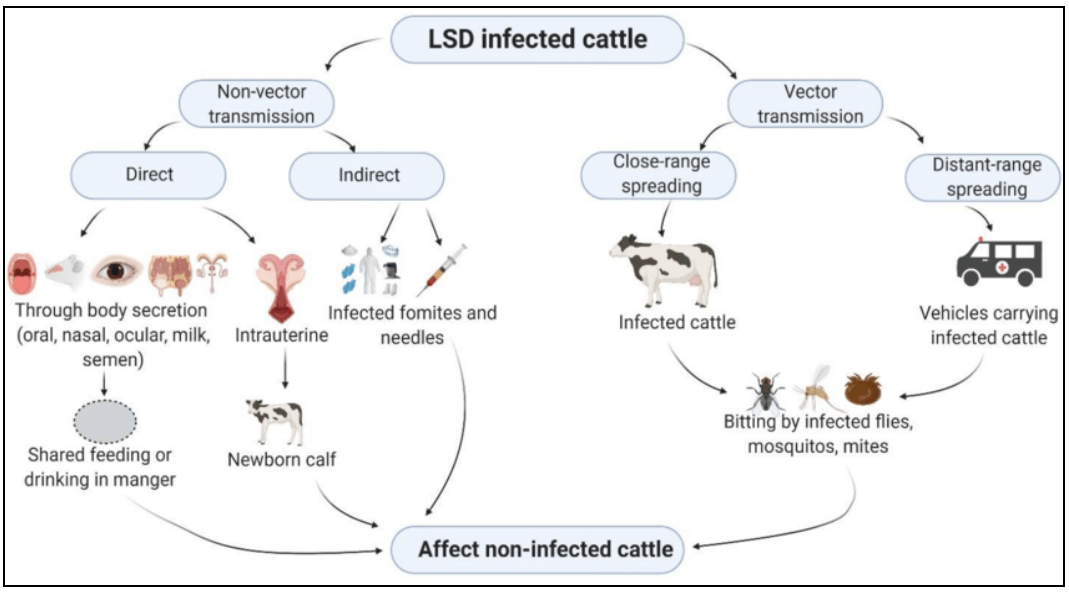

Fig 3

Host range

Cattle (Bos indicus and Bos taurus) and buffalo (Bubalus bubalis) are susceptible hosts. Compared to local breeds of cattle, the bos taurus is more vulnerable. All animals are sensitive, however calves are more so and develop lesions in 24 to 48 hours than other animals (Al-Salihi 2014). Wild animals are naturally immune to infection, but in experimental settings, oryx (Oryx gazelle), springbok (Antidorcas marsupialis), impala (Aepyceros melampus), and giraffe (Giraffe camelopardalis) as well as oryx (Oryx gazelle) and Thomson's gazelle all developed clinical lesions. (Davies 1991; Padilla *et al*. 2005). Typically, it has been discovered that wildlife plays a very small part in the transmission and maintenance of LSDV. The virus does not affect humans (OIE, 2013).

Transboundary spread

According to Tupurainen and Oura (2012), the disease originally appeared in Zambia in 1929 before spreading to the rest of Africa with the exception of Libya, Algeria, Morocco, and Tunisia. Middle Eastern nations have boosted the shipping of animals from their neighbors in response to the region's rising food needs. Due to the transportation of infected animals from afflicted African nations, Egypt contracted LSD in 1988. Again in 2006, the disease reappeared as a result of unrestricted cattle movement from nations producing African horn (Ali *et al*. 1990; Fayez and Ahmed 2011). According to Yaruham *et al*. (1995), the migration of infected Stomoxy calcitrans from Egypt was assumed to be the cause of the first outbreak in Israel in 1989. The information regarding reported outbreaks as listed in the OIE disease outbreak The report for the years 2010 to 2019 is depicted in Fig. 2. Syria, Jordan, and Lebanon saw the disease for the first time in 2012 and 2013. As evidence of the spread of disease across borders, an outbreak in Jordan appeared close to the borders with Israel and Syria (Abutarbush *et al*. 2013). In 2013 and 2014, the illness also spread to neighboring countries including Iran and Turkey. Later, reports of LSD came from Turkey, Cyprus, and Azerbaijan (data not available in OIE; BY-ND 2016). According to the OIE study, LSD returned to Israel in 2019 after 6 years due to a decline in animal vaccinations, which were previously required (European Food Safety Authority *et al*. 2020). Table 2 (Data source: OIE Disease Information) displays the temporal distribution of the number of outbreaks in Asian countries from 2010 to 2019. LSD outbreaks were initially noted in countries bordering each other: Bangladesh, China, and India. China reported the first case of the disease in August 2019; 65 animals were infected in the village of Illi Kazakh Autonomous Prefecture, which is close to the Kazakhstani border. Kazakhstan experienced the most recent epidemic in 2016. A similar illness epidemic that afflicted 66 animals out of 360 exposed animals was reported in Bangladesh in the months of July and September of this year. According to the European Food Safety Authority and others (2020), diseases have returned in the east and south provinces of Russia and Turkey, respectively, in 2019.

In India, the first case of the disease was discovered in the state of Odisha in August of this year, during the monsoon season, when there was a high density of mosquitoes. The first event occurred on August 12, 2019, in the Orissan districts of Khairbani, Betnoti, and Mayurbhanj, where 9 instances were reported on a farm with 135 animals. Then, a few days later, a second outbreak from the same area was discovered at a different location called Patalipura, where 20 LSD cases were discovered in a farm with 441 vulnerable animals. On August 20, 2019, a farm in Rajendrapur, Bhandaripokhari, Bhadrak, Odisha reported a third case outbreak with 50 cases and 356 animals (https://www.oie.int/). In the first LSD report released in India, it was discovered that 182 animals out of 2539 tested positive, with no mortality and a 7.1% morbidity rate. The strain found in India was genetically more similar to South African NI2490/KSGP-like strains than to European strains, according to phylogenetic research (Sudhakar *et al*. 2020).

The causative organism

The responsible party the genus Capripoxvirus, one of the Poxviridae family, is what causes lumpy skin disease. These three viruses are distinct and cannot be separated using conventional serological assays. From 55°C/2 hours and 65°C/30 minutes, LSDV is in danger. It can be removed from pores and skin nodules and kept at 80°C for ten years. Fluid from inflamed tissue subcultures can be kept at 4°C for six months. The virus may have a pH that is unusually alkaline or acidic. P> Held for five days at a pH range of 6.6 to 8.6 and 37 °C. Ether (20%), chloroform (10%), formalin (1%), certain detergents, and sodium lauryl sulfate are all toxic to LSDV. Additionally, phenol (2%/15 minutes), sodium hypochlorite (2-4%), iodine compounds (1:33 dilution), Virkon® (2%), and quaternary ammonium (3%), among others, pose risks to your health. compounding (0.5%). LSDV is very stable and can persist at ambient temperature for extended durations, especially in dry crusts. The LSDV has a high level of inactivation resistance. survived for at least 33 days necrotic skin nodules, up to 35 days for dry scabs, and at least 18 days for skins that have been air-dried. It might hang around the neighborhood for a while. The virus may persist for many months in dark conditions, including infected animal quarters, but it is susceptible to exposure to daylight and detergents that contain grease solvents. It is discovered that there is a group of LSDV (Tulman *et al*. 2001). The LSDV genome (151 kbp) has 156 putative genes and a significant coding region surrounded by 2. 4 kbp inverted terminal repeats. However, 146 conserved genes encoding proteins involved in transcription and mRNA biogenesis, nucleotide metabolism, DNA replication, protein processing, virion shape and assembly, viral pathogenicity and host range, are present in all known genera of cordopoxviruses. The genomic area of the LSDV genes has substantial collinearity and amino acid identity (65% shared) with genes from other recognized mammalian poxviruses, including suipoxvirus, yatapoxvirus, and leporipoxvirus. Poxvirus homologues are lacking or have a lower level of amino acid identity (on average 43%) at the terminal sections, and collinearity is broken. The distinct complement of genes responsible for viral host diversity and virulence includes interleukin 10 (IL-10), IL-1 binding proteins, G protein-coupled CC chemokine receptor, and epidermal growth factor-like protein seen in other poxvirus genera. Several capripoxviruses, including LSDV (Tulman *et al*. 2001), varicella virus, and goatpox virus (Tulman *et al*. 2002), have had their whole genomic sequences published.

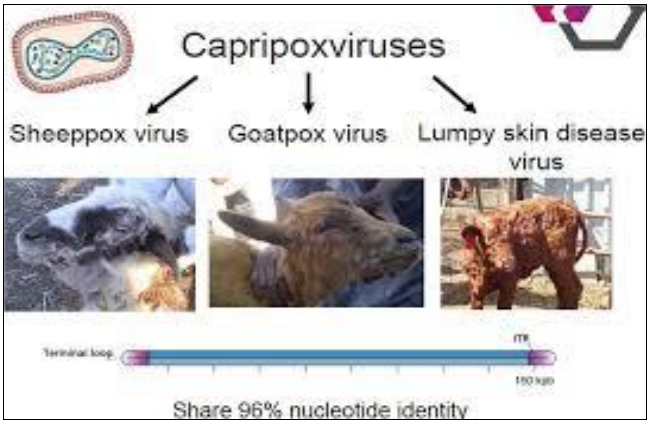

Fig 4

Scientific classification

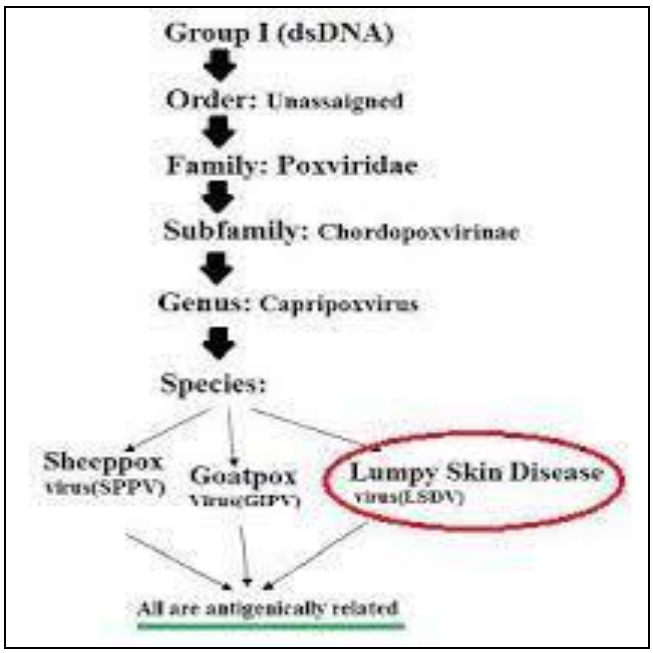

Fig 5

Lympy skin disease preparation

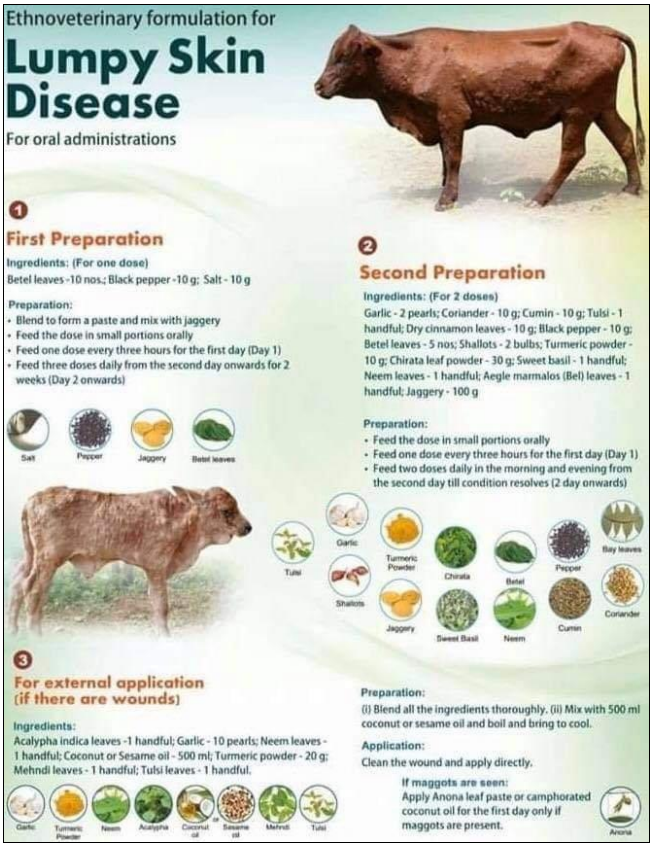

Fig 6

Differentiate diagnosis

When utilizing extremely sensitive fast PCR methods overnight to detect real cases, mild cases and early stages of infection can be challenging to separate from severe cases of LSD. The following conditions can be thought of as alternative diagnoses for LSD

- **Pseudobulky Skin Disease/bovine herpetic mammilitis (bovine herpesvirus 2):** Skin lesions may mirror those of LSDv, but they are less severe and more superficial, and the disease's short-term course.LSDV detection on pcr allows for the exclusion of the disease.

- **Insect bites, urticaria, and photosensitization:** These conditions can cause skin lesions that mirror those of lsdv but are less severe and have a shorter and milder clinical course. the illness resulting from the identification

- **Pseudocowpox (parapoxvirus):** Lesions only appear on teats and udders lsdv via pcr. The PC's identification of LSDV can rule out the disease.

- **Dermatophilosis:** Early tinea lesions with a non-ulcerative surface structure that are more superficial and visibly different.

- **Demodicosis:** Alopecia and skin lesions primarily on the withers, neck, back, and flanks. Using skin scrapings, it is possible to identify mites and rule out the condition.

- **Parapox virus-caused bovine papular stomatitis:** Only the mucous membranes of the mouth experience lesions a pcr test can be used to rule out the disease.

- **Besnoitiosis:** Skin lesions may resemble alopecia with thick, wrinkly skin. These lesions typically affect the scleral conjunctiva. Lsdv can be found utilizing pcr to rule out the disease.

- **Onchocerciasis:** Ventral midline dermal lesions are most likely affected. PCR can be used to rule out disease. Additionally, cow vaccination with live-attenuated Lsdv vaccinemay have negligible adverse effects resembling those of clinical Lsd

Prevention and control

There is currently no cure for LSD that is effective. For the therapy of symptoms, anti-inflammatory drugs and antibiotics are employed. Effective preventative and control measures must be put in place in order to control the disease, including:

a. **Limit movement:** In order to stop the spread of transboundary disease, it is imperative that LSD-infected animals not be moved at all. To stop the rapid spread of disease inside a nation, animals having these lesions should be isolated for inspection.

b. **Limit vector movement:** Disease transmission may be facilitated by vector movement brought on by strong winds. Using vector control strategies, such as the use of vector traps and pesticides, the disease can also be avoided.

c. **Immunization:** There is a live attenuated vaccination for LSD. depending on various strains Companies created vaccinations against the LSD virus. It is either based on the SIS Neethling type (Lumpyvax, MSD Animal Health-Intervet, South Africa) or the Neethling strain, such as the Lumpy Skin Disease Vaccine for Cattle (Onderstepoort Biological Products; OBP, South Africa) or Bovivax (MCI Sante Animale, Morocco). Since the sheeppox and goatpox viruses and LSD are closely related, the sheeppox and goatpox vaccines can be utilized to treat LSD (Tuppurainen *et al*. 2015). According to OIE, many viral strains are employed as vaccine strains. The South African homologous Neethling strain of the Lumpy Skin Disease virus protects for three years after passing through lamb kidney cells 60 times in lamb kidney cells and 20 times on the chorioallantoic membrane of embryonated chicken eggs. Kenyan sheeppox virus passaged 18 times in the sheeppox vaccinations used against LSD Romanian sheep pox strain, Yugoslavian RM 65 sheep pox strain, lamb testis (LT) cells, or fetal calf muscle cells. The strains of the heterologous immunization cause local reactions. These vaccines are not recommended in locations where sheeppox and goatpox are prevalent because they may act as sources of infection for sheep and goat populations that are vulnerable. According to Garietal. (2015), Brenner *et al*. (2009), Capstick and Coakley 1961–1962, and Carn *et al*. (1994), live at tenuated Gorgan goatpox strain offers effective protection in cattle with almost no side effects. Since the LSD virus is stable and lasts a long time in the environment, long-term immunization with 100% coverage should be made mandatory for disease control and prevention. new animals to the afflicted farm before doing so, They need to get vaccinated. Calves need to be vaccinated immunized mothers or mothers who are naturally infected when they are 3 to 4 months old. Annual vaccinations are possible for breeding bulls and pregnant cows (Tupprainen *et al*. 2015).

Treatment

The virus cannot be treated, thus the best defense is immunization, which can help avoid infection. Nonsteroidal anti-inflammatory medications (NSAIDs) and antibiotics (topical +/- injectable) can both be used to treat secondary skin infections. if applicable,

- Methylene blue antiviral therapy
- Antibiotics are given to treat secondary infections, non-steroidal anti-inflammatory medicines are used to treat the inflammatory disease, paracetamol is used to treat high fevers, and vaccinations are given.

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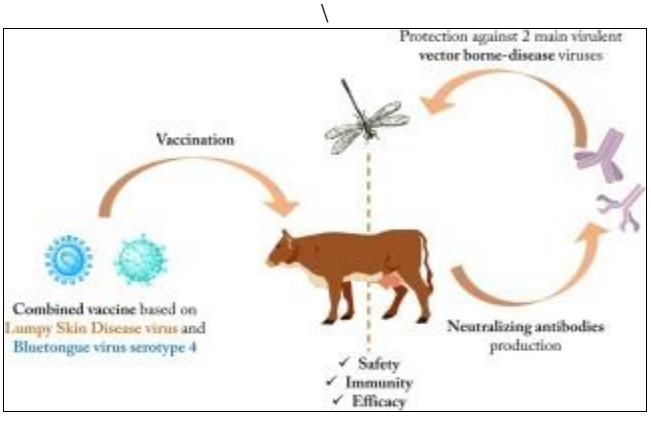

Fig 7

Conclusion

Important livestock like cattle and buffaloes have a significant role in the global economy. Cattle and buffalo are susceptible to the dangerous disease lumpy skin disease. Prior to recently, the disease was confined to a small number of countries in Asia and Africa; however, given that the economies of the majority of these nations are based on agricultural, the recent expansion of the disease to these nations raises concerns for the cattle rearing industry. The expansion of this disease to more extensive geographic areas of the Indian subcontinent will undoubtedly have a negative impact on all sectors of the economy, but the rural economy in particular. LSD can also cause a drop in the export of commodities relating to livestock and cattle. Along with epidemiological random screening, it is necessary to analyze the factors that led to the introduction of LSD in India across various regions to determine the true disease prevalence. The only way to avoid the disease is through vaccination, in addition to efficient quarantine measures and vector control techniques

Reference

1. Veterinary Medicine and Science Wiley-Blackwell Lumpy skin disease, an emerging transboundary viral disease: A revi Fatemeh Namazi and Azizollah Khodakaram
2. frontiers in microbiology, Understanding the research advances on lumpy skin disease: A comprehensive literature review of experimental evidence, Zhengji Liang1, Kaishen Yao1, Shasha Wang1, Juanbin Yin1, Xiaoqin Ma1, Xiangping Yin1*, Xiangwei Wang1* and Yuefeng Sun1*
3. Lumpy skin disease, an emerging transboundary viral disease: A reviewFatemeh Namazi and Azizollah Khodakaram Tafti.Vetarnay medicine and science
4. R:-Calistri P, De Clercq K, Gubbins S, Klement E, Stegeman A, Cortiñas Abrahantes J, *et al*. Lumpy skin disease epidemiological report IV: data collection and analysis. EFSA Journal,2020:18(2):6010.
5. Zeynalova S, Asadov K, Guliyev F, Vatani M, Aliyev V. Epizootology and molecular diagnosis of lumpy skin disease among livestock in Azerbaijan. Frontiers in Microbiology,2016:7:1022.
6. Ali A, Gumbe F. Review on lumpy skin disease and its economic impacts in Ethiopia,2018:7:39-46.
7. OIE. Lumpy skin Disease (LSD). Situation in Bhutan, 2021.https://rr-asia.oie.int/wp content/uploads/2021/01/1-presentation-on-lsd-outbreaks-in-bhutan-2020.pdf.
8. Gubbins S. Using the basic reproduction number to assess the risk of transmission of lumpy skin disease virus by biting insects. Transboundary and Emerging Diseases,2019:66:1873-1883.
9. Tuppurainen E, Alexandrov T, Beltrán-Alcrudo D. Lumpy skin disease field manual – A manual for veterinarians. FAO Animal Production and Health Manual,2017:20:1-60.
10. Annandale CH, Holm DE, Ebersohn K, Venter EH. Seminal transmission of lumpy skin disease virus in heifers. Transboundary and Emerging Diseases,2014:61:443-448.
11. Kasem S, Saleh M, Qasim I, Hashim O, Alkarar A, Abu-Obeida A, *et al*. Outbreak investigation and molecular diagnosis of Lumpy skin disease among livestock in Saudi Arabia 2016. Transboundary and Emerging Diseases,2018:65:e494-500.
12. Sprygin A, Pestova Y, Wallace DB, Tuppurainen E, Kononov AV. Transmission of lumpy skin disease virus: A short review. Virus Research,2019:269:197637.
13. Ali H, Ali AA, Atta MS, Cepica A. Common, Emerging, Vector-Borne and Infrequent Abortogenic Virus Infections of Cattle. Transboundary and Emerging Diseases,2012:59:11-25.
14. Rouby S, Aboulsoud E. Evidence of intrauterine transmission of lumpy skin disease virus. Veterinary Journal,2016:209:193-195.
15. Lubinga JC, Tuppurainen ESM, Stoltsz WH, Ebersohn K, Coetzer JAW, Venter EH. Detection of lumpy skin disease virus in saliva of ticks fed on lumpy skin disease virus-infected cattle. Experimental and Applied Acarology,2013:61:129-138.
16. Mirror of Research in Veterinary Sciences and Animals (MRVSA) Review ArticleLumpy Skin disease: Review of literatureK. A. Al-Salihi 1BSC, MSC, Ph.D in Veterinary Medicine and Pathology / Faculty of Veterinary Medicine / The University of Nottingham / UK. Email address: kama_akool18@yahoo.co.uk
17. https://www.researchgate.net/figure/Classification-of-Lumpy-skin-disease-virus_fig1_272795279
18. LUMPY SKIN DISEASEA field manual for veterinarians Authors Eeva Tuppurainen Independent consultant Tsviatko Alexandrov Bulgarian Food Safety Authority (BFSA)Daniel Beltrán-AlcrudoFAO
19. https://www.nadis.org.uk/disease-a-z/cattle/lumpy-skin-disease/
20. https://www.apnikheti.com/en/pn/%E0%A8%AE%E0%A8%BE%E0%A8%B9%E0%A8%B0%E0%A8%BE%E0%A8%82-%E0%A8%95%E0%A8%BF%E0%A8%8A-%E0%A8%90%E0%A8%82%E0%A8%A1-%E0%A8%8F-%E0%A8%B5%E0%A9%87%E0%A8%B0%E0%A8%B5%E0%A8%BE/kisan-veer-es-kit-di-varto-karn-lumpy-skin-disease-to-bachav-layi/pb
21. https://goelvetpharma.com/product/homeonest-marigold-lsd-25-kit-for-lumpy-skin-disease/ https://www.ingenetix.com/en/product/veterinary/Lumpy+Skin+Disease+Virus/
22. https://www.capecross.co.za/shop/cattle-sheep/lumpyvax-vaccine-for-cattle-100ml-100-dose/