



A review article on lumpy skin disease

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Abstract:-

Summary Contagious nodular skin disease caused by contagious nodular skin disease virus is one of the major health problems affecting livestock farming in most African countries. Skin lesions are the main sources of infection; although the virus is evacuated through various body secretions and excretions, including semen. Therefore, susceptible hosts become infected with the virus primarily through mechanical means from blood-sucking arthropods, including flies, mosquitoes, and ticks. Transstadial and transovarian persistence is also possible in various tick species. After infection, the characteristic lesions of contagious nodular skin disease may burst 7 to 14 days after infection in experimental conditions, while in natural cases it takes 2 to 5 weeks. Multiple skin nodules (severe forms), sometimes involving the mucous membranes of the respiratory tract, urogenital system and other internal organs. As a result, there will be reduced milk production, miscarriages, temporary or permanent sterility, skin damage and death, further contributing to catastrophic economic losses in livestock producing countries. Therefore, large-scale vaccination combined with other appropriate control measures is the most effective way to limit the spread and economic impact of contagious nodular skin disease. This review aims to provide the latest information on the biology of lumpy skin disease virus, the mechanism of spread, and the clinical and pathological features of lumpy skin disease

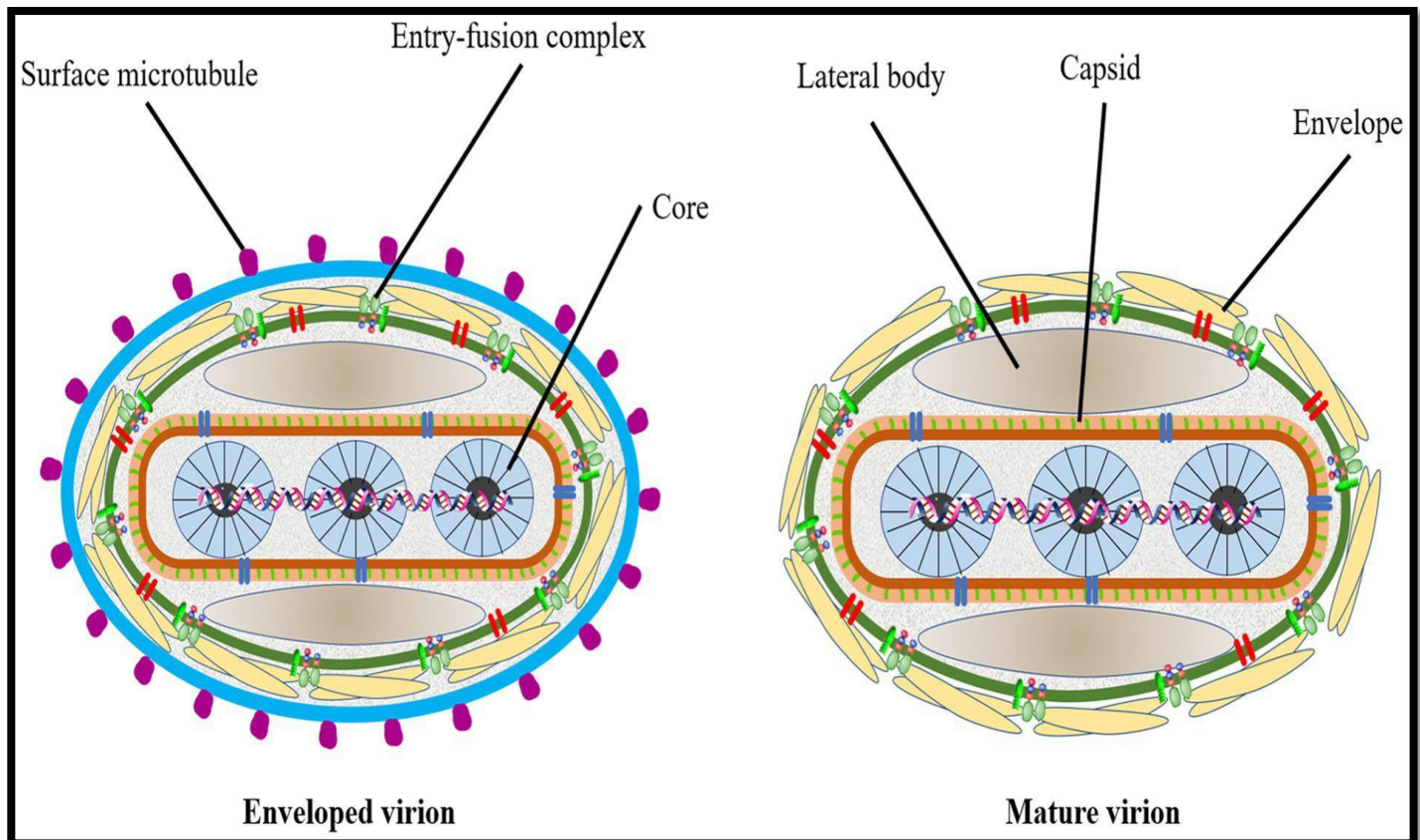
Keywords: cattle, ELISA, lumpy skin disease, nepal, : Lumpy skin disease, cow, knopvelsiekte, Middle East. | Propolis, Alginate, Nanoparticles, Lumpy Skin Disease infection, Cattle.

Introduction :

Infectious nodular skin disease (LSD), a major threat to livestock, can cause acute or subacute illness in cattle and water buffalo (Givens, 2018; Tuppurainen, Venter, et al., 2017). All age groups and cattle breeds are affected, but especially young and lactating cattle (Tuppurainen et al., 2011). The reason why the World Organization for Animal Health (OIE) included this transboundary disease on the list of notifiable diseases is because of its significant economic impact. Losses and potential for rapid multiplication (Tuppurainen & Oura, 2012). The recent spread of the disease in disease-free countries demonstrates the importance of its transmission, control and eradication (Sprygin et al., 2019). Infectious nodular skin disease (LSDV) is a double-stranded DNA containing approximately 150 kilobase pairs (kbp) with relatively large sizes (230-260 nm) encapsulated in a lipid envelope and belongs to the genus Capripoxvirus, which is genetically related to sheep smallpox (SPPV) and goatpox virus (GTPV) (Bhanuprakash et al., 2006; Buller et al., 2005; Givens, 2018). This virus is the most economically important virus in the Poxviridae family affecting domestic ruminants. The capsid or nucleocapsid of the virus is brick-shaped or oval and contains the genome and lateral bodies. Reaction and mutual protection between members. Although capripoxviruses are generally considered to be host specific, SPPV and GTPV strains can naturally or experimentally infect each other and cause disease in both host types. In contrast, LSDV can experimentally infect sheep and goats, but no natural infection of goats with LSDV has been reported.

Figure1:- Morphological structure LSDV(2)**2. CLINICOPATHOLOGY:-**

Medical features of the condition include fever, loss of appetite, runny nose, drooling and lacrimation, enlarged lymph nodes, greatly reduced milk production, low body weight, and occasionally death (Abutarbush et al., 2013; Annandale et al., 2014; Babiuk et al., 2008; Tasioudi et al., 2016). The disease is also characterized by firm, scarcely raised, circumscribed skin nodules (Figure 1) that are 2–7 cm in diameter and commonly appear on the neck, feet,



tail, and back soon after the onset of fever (Beard, 2016; Sevik & Dogan, 2017). Necrotic and ulcerative nodules increase the possibility of myiasis (Beard, 2016). Edema of the legs and lameness are noted in some cases (Tuppurainen & Oura, 2012). LSDV can cause abortion (Radostitis et al., 2006), mastitis and orchitis (Awadin et al., 2011). However, nodules were no longer detected in aborted fetuses (Sevik & Dogan, 2017). Nodules in the lungs and gastrointestinal tract were regularly found during autopsy, edema and pulmonary congestion (Zeynalova et al., 2016). Tissues such as the snout, nasal cavity, larynx, trachea, labia minora, dental pads, gums, abomasum, udder, teats, uterus, vagina, and testicles are likely to be seen. It has been mentioned as keratitis, dysentery, lameness, pneumonia, mastitis and myiasis (Al-Salihi & Hassan, 2015; Tuppurainen et al., 2017). Histopathological examination of nodules in the pores and skin could also detect pathognomonic eosinophilic intracytoplasmic inclusions in our body within keratinocytes, macrophages, endothelial cells and pericytes and are related to ballooning degeneration of stickleback cells. Infiltration of the superficial skin tissues of the affected regions is monitored using inflammatory cells including macrophages, lymphocytes and eosinophils. In addition, in some cases significant vasculitis and severe coagulative necrosis can be detected in the subcutaneous muscle tissue (Constable et al., 2017; Sevik et al., 2016). Pseudo-bulky skin disease, urticaria, streptotrichosis (infection with *Dermatophilus congolensis*), ringworm, *Hypoderma bovis* infection, photosensitization, bovine papular stomatitis, foot-and-mouth disease, bovine viral diarrhea and malignant catarrhal fever are considered the differential prognosis of LSD (Abutarbush, 2017). (1)



Figure2 :- Cow infected with LSD reveals multiple skin nodules (from Iraq recent outbreak) (3)

3. PATHOGENESIS:-

After LSDV contamination, viral replication, viraemia, fever, cutaneous localization of the virus and improvement of the nodules occur

(Constable et al., 2017). Experimentally, the following activities were reported after intradermal inoculation of the virus:

- 4 to 7 days post-contamination (DPI): localized swellings as 1 to 3 cm nodules or plaques on the inoculation site
 - 6 to 18 DPI: Viremia and virus loss through oral and nasal secretion
 - 7 to 19 DPI: local adenopathies and improvement of generalized skin nodules
 - 42 days after fever: presence of viruses in semen (Coetzer, 2004)
- . The intracellular replication of the virus in fibroblasts, macrophages, pericytes and endothelial cells ends in vasculitis and lymphangitis in the affected tissues (Coetzer, 2004). It appears that younger calves, lactating cows

and underweight animals are more susceptible to herbal infections, probably due to impaired humoral immunity (Babiuk, Bowden, Boyle, et al., 2008).

Animals that have recovered from plant contamination by the virus have shown lifelong immunity. Calves from their inflamed mothers are medically safe for approximately 6 months due to preserved maternal antibodies (Tuppurainen et al., 2005). Affected animals clear the contamination and no supplier kingdom has yet considered LSDV (Tuppurainen, Alexandrov, et al.(3)

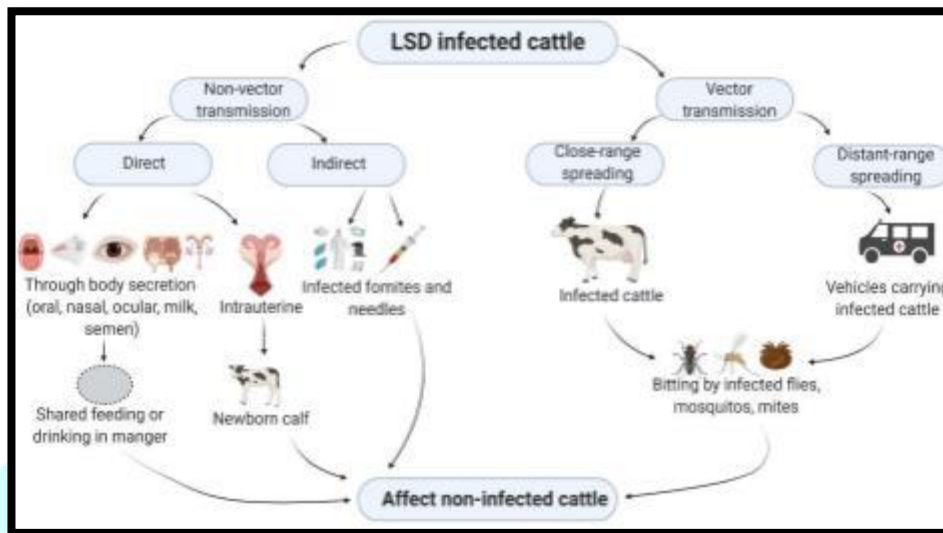


Figure3 :-- Epitome of possible modes of transmission of LSDV. LSD infected cattle may affect non-infected cattle through vector or non-vector

Transmission:-

The mechanism of transmission of LSDV is useful for assessing the epidemiology of the virus and thus contributes to the strategy of progressive control and eradication of the disease [4,5]. An excerpt of the possible types of LSDV transmission is shown in Figure 2. Figure 2. Epitome of possible routes of transmission of LSDV. LSD-infected cattle can affect uninfected cattle by vectorial or non-vectorial transmission. Non-Vector Transmission Although ineffective, non-vector transmission of LSD occurs when clinically affected animals come into contact with contaminated materials without the need for biological or mechanical vectors. Infectious LSDV is shed via saliva, nasal and ocular secretions and contaminates community foods. And areas of drinking and disease spread [6,7,8]. Transmission through contaminated needles during vaccination, spread through infected sperm during sexual intercourse, ingestion of milk and intrauterine transmission can also serve as sources of infection [6,9,10]. Vector transmission The role of arthropod vectors in transmission of this virus has been confirmed experimentally [11,12] Various blood-sucking hard ticks, eg *Rhipicephalus appendiculatus* (brown ear tick), *Rhipicephalus decoloratus* (blue tick). And *Amblyomma hebraeum*, the mosquito *Aedes aegypti* and the flies *Stomoxys calcitrans*, *Haematobia irritans* and *Musca domestica* have been implicated in the spread of LSDV in sub-Saharan Africa [10].] and is transmitted transovarially in cold temperatures [13,14]. The virus can spread over short distances of a few kilometers [15] and even longer distances due to the unrestricted movement of animals across international borders .

The causative organism:-The causative agent The genus *Capripoxvirus* from the relatives *Poxviridae* is the causative agent of Lumpy Skin Disease. These 3 viruses are different, they cannot be distinguished with ordinary serological tests (Figure 1). LSDV is endangered from 55°C/2 hours and 65°C/30 minutes. It can be extracted and stored from pores and skin nodules-80°C for 10 years. Inflamed tissue subculture fluid can be stored at 4°C for 6 months. The virus runs the risk of having an exceptionally alkaline or acidic pH. P> Maintained at pH 6.6-8.6 at 37°C for five days. LSDV is vulnerable to ether (20%), chloroform, formalin (1%) and some detergents, sodium laurilsulfate. You are also at risk from phenol (2%/15 minutes), sodium hypochlorite (2-3%), iodine compounds

(1:33 dilution), Virkon® (2%) and quaternary ammonium compounds (0.5 percent). LSDV is remarkably stable, surviving for long periods at room temperature, especially in dry crusts. LSDV can be very resistant to inactivation. Survived up to 33 days or longer in necrotic skin nodules, up to 35 days in dry scab, and at least 18 days in air-dried skins. It may remain in the area for a long period of time. Meanwhile, the virus is vulnerable to exposure to daylight and detergents containing grease solvents, while it can survive for many months in dark environments, including infected animal pens. A collection of LSDV is identified (Tulman et al. 2001). The LSDV genome (151 kbp) contains a relevant coding region bounded by 2.4 kbp inverted terminal repeats and includes 156 putative genes. However, cordopoxviruses of various known genera have 146 conserved genes encoding proteins related to transcription and mRNA biogenesis, nucleotide metabolism, DNA replication, protein processing, and virion shape and assembly, and viral virulence and host range. The LSDV genes share a high degree of collinearity and amino acid identity (65% shared) of their genomic region with genes from other known mammalian poxviruses, notably suipoxvirus, yatapoxvirus, and leporipoxvirus. Collinearity is disrupted and poxvirus homologues are absent or have a lower percentage of amino acid identity (average 43%) in the terminal regions. IL-10, IL-1 binding proteins, G protein-coupled CC chemokine receptor and epidermal growth factor-like protein found in other poxvirus genera. Unique complement of genes responsible for viral host diversity and virulence. The complete genomic sequences of several capripoxviruses have been published, including LSDV (Tulman et al. 2001), varicella virus and goatpox virus (Tulman et al. 2002). (16)

Lumpy virus skin disease preparation:-



1

First Preparation

Ingredients: (For one dose)

Betel leaves - 10 nos.; Black pepper - 10 g; Salt - 10 g

Preparation:

- Blend to form a paste and mix with jaggery
- Feed the dose in small portions orally
- Feed one dose every three hours for the first day (Day 1)
- Feed three doses daily from the second day onwards for 2 weeks (Day 2 onwards)



Salt



Pepper



Jaggery



Betel leaves



2

Second Preparation

Ingredients: (For 2 doses)

Garlic - 2 pearls; Coriander - 10 g; Cumin - 10 g; Tulsi - 1 handful; Dry cinnamon leaves - 10 g; Black pepper - 10 g; Betel leaves - 5 nos; Shallots - 2 bulbs; Turmeric powder - 10 g; Chirata leaf powder - 30 g; Sweet basil - 1 handful; Neem leaves - 1 handful; Aegle marmalos (Bel) leaves - 1 handful; Jaggery - 100 g

Preparation:

- Feed the dose in small portions orally
- Feed one dose every three hours for the first day (Day 1)
- Feed two doses daily in the morning and evening from the second day till condition resolves (2 day onwards)



Tulsi



Garlic



Turmeric Powder



Chirata



Betel



Pepper



Bay leaves



Shallots



Jaggery



Sweet Basil



Neem



Cumin



Coriander

3

For external application (if there are wounds)

Ingredients:

Acalypha indica leaves - 1 handful; Garlic - 10 pearls; Neem leaves - 1 handful; Coconut or Sesame oil - 500 ml; Turmeric powder - 20 g; Mehndi leaves - 1 handful; Tulsi leaves - 1 handful.



Garlic



Turmeric Powder



Neem



Acalypha



Coconut oil or Sesame oil



Mehndi



Tulsi

Preparation:

(i) Blend all the ingredients thoroughly. (ii) Mix with 500 ml coconut or sesame oil and boil and bring to cool.

Application:

Clean the wound and apply directly.

If maggots are seen:

Apply Anona leaf paste or camphorated coconut oil for the first day only if maggots are present.



Anona

Scientific classification:-

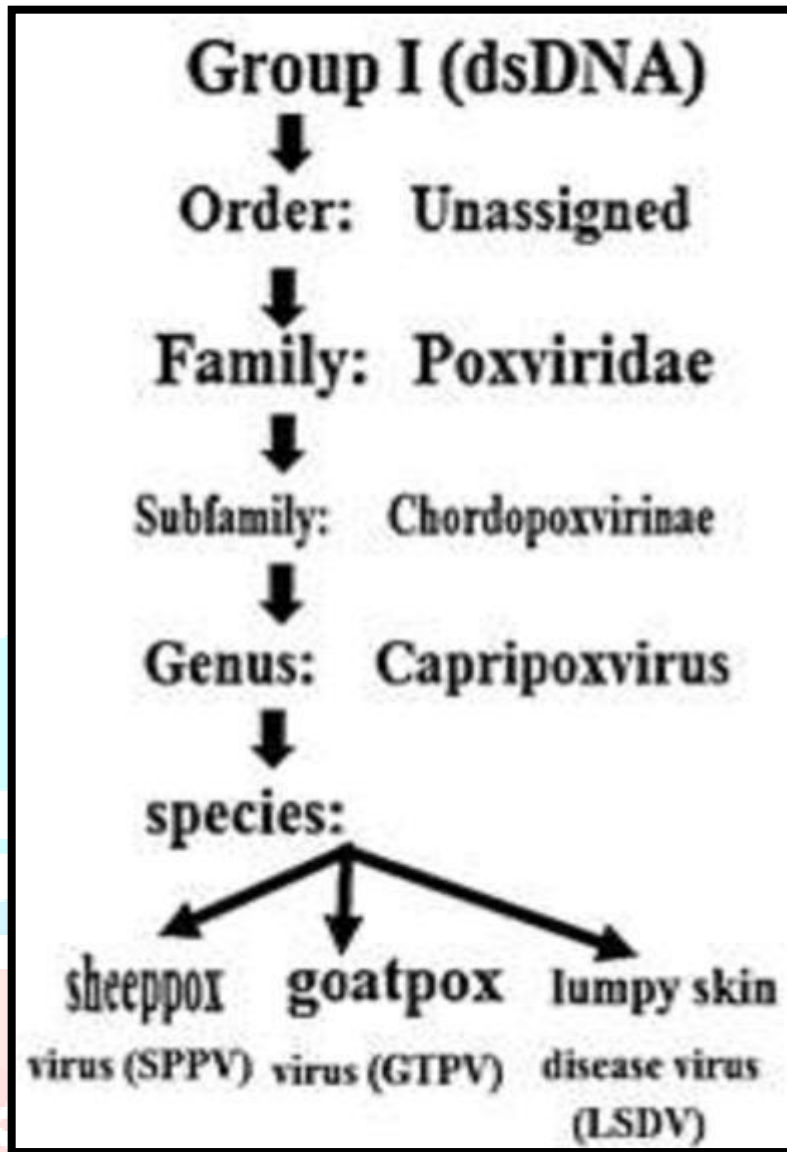


figure:- classification of lumpy skin disease virus(17)

Differentiate diagnosis:-.

Severe cases of LSD are very distinctive and easy to recognize, but early stages of infection and mild cases can be difficult to distinguish when using highly sensitive rapid PCR methods overnight to distinguish true cases. The following disorders can be considered as differential diagnoses for LSD:

- Pseudobulky Skin Disease/bovine herpetic mammilitis (bovine herpesvirus 2) (Fig. 19): Skin lesions may resemble those of LSDv but are more superficial and the course of the disease is shorter and less severe .the disease can be ruled out by pcr detection of lsdv
- insect bites, urticaria and photosensitization: skin lesions can resemble those of lsdv but are more superficial and the course of the disease is shorter and less severe (fig. 20) the Disease caused by detection of lsdv by pcr.
- Pseudocowpox (parapoxvirus) (Fig.21): Lesions only occur on teats and udders. The disease can be excluded by the detection of LSDV by PC.

- Dermatophilosis (Fig.22): Early tinea lesions, more superficial, clearly different, non-ulcerative surface structure of the tinea lesion.
- Demodicosis (Fig.23): Skin lesions predominantly on the withers, neck, back and flanks, often with alopecia. The disease can be ruled out by detecting mites using skin scrapings
- Bovine papular stomatitis (parapox virus) (Fig. 24): Lesions only occur on the mucous membranes of the mouth. The disease can be ruled out by a pcr test
- Besnoitiosis (Fig. 25): Lesions usually occur on the scleral conjunctiva, and skin lesions may represent alopecia with thick, wrinkled skin. The disease can be ruled out by detecting Lsdv using pcr
- . • Onchocerciasis (Fig. 26): Dermal lesions most likely in the ventral midline.Disease can be ruled out by PCR. In addition, live-attenuated Lsdv vaccines in cattle may produce mild side effects similar to clinical Lsd (see pages 37-40 for currently available vaccines).(18)

Prevention:



The control and prevention of contagious nodular skin disease is based on four tactics: movement control (quarantine), vaccination, killing campaigns, and management strategies. Specific national control plans vary from



country to country, so advice should be sought from relevant authorities and veterinarians. Vaccination is the most effective means of control and homologous live vaccines containing a Neethling-like strain of LSD are recommended. (19,20,21,22,23,24)

Treatment:

There is no treatment for the virus, so prevention through vaccination is the most effective way to combat it. Secondary skin infections can be treated with nonsteroidal anti-inflammatory drugs (NSAIDs) and also with antibiotics (topical +/- injections.) if appropriate

- Antiviral treatment with methylene blue
- Use of non-steroidal anti-inflammatory drugs to treat the inflammatory condition • Use of paracetamol for high fever
- Administration of antibiotics to fight secondary infections
- Vaccination

Conclusion:

In conclusion, PCR is the fastest and most accurate method of confirming LSD infection when laboratory facilities are available; However, histopathology and IHC can also be used in routine pathology laboratories to detect LSDV antigen in nodular skin tissues to confirm LSD infection, which was comparable to virus identification findings and results of PCR.

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