IJARSCT
ISSN (Online) 2581-9429
International Journal of Advanced Research in Science, Communication and Technology (IJARSCT)
Volume 3, Issue 2, March 2023
Impact Factor: 7.301

Lumpy Virus and Skin Infection: A Review

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Abstract: *Lumpy skin disease (LSD) responsible for losses in the lives of animals. It is caused due to the virus called Lumpy skin disease virus (LSDV). It belongs to the family Poxviridae and genus Capripoxvirus. It includes sheep pox virusas well as goat pox virus. This disease mainly affect cattle and water buffaloes. The transmission of virus is through the arthropod vectors. it is manifested by characteristics like skin nodules, pyrexia, nasal discharge, swelling of superficial lymph node, lachrymation. The disease is first observed in African and Middle East countries but has started spreading from China and Bangladesh sharing borders with India. LSD causes in female abortions and in male sterility. It can also cause the milk and beef production. This review aims to summaries the latest developments in the epidemiology with the focus on spread, aetiology and transmission, clinical presentations, diagnostics and management of the disease.*

Keywords: Lump skin disease, animal, India, Virus

I. INTRODUCTION

Production of livestock is one of the principle of achieving living standards to develop the world. The livestock holds about 40%value of agriculture globally, itsupports the live hoods and provide food security to a billion of people [1]. The disease is native to African countries but recently the disease has been reported from all over around the world. The first case of LSD was reported from Zambiain 1929 [2] and then in southern and northern African countries. Later on, it spread to Israel, Kuwait, Oman and Yemen [3]. According to OIE, at present thisdisease is prevalent in countries including various African, European countries [4]. This disease can be diagnosed by transmission electron microscopic (TEM), immune-peroxidase (IMP) staining, antigen trapping enzyme-linked immunosorbent assay (ELISA) anda polymerase chain reaction [PCR] test. Thereis no proper treatment for LSD. whereas, relative treatment should be given to animals which are infected to alleviate analytical indication and to control all secondary issues. Immunization is one of the effective method for susceptible animals to control the disease in South Africa, and the effective vaccines are madefrom the Neethling strain virus [5].

AETIOLOGY

Lumpy skin disease virus (LSDV) is responsible for causing Lumpy skin Poxviridae family that holds group of viruses causing diseases in most of the domestic animals but dog is exceptional here. These contain two subfamilies:Chordopoxvirinae, it contaminate vertebrate host and Entomopoxvirinae infecting invertebrate hosts [6]. The subfamily Chordopoxvirinae, it comprises 10 genera and it includes Capri poxvirus genus. Three species of genus contains viruses of three species they are sheep pox virus (SPPV), goat poxvirus (GTPV)and lumpy skin disease virus (LSDV) infecting sheep, goat and cattle, respectively [7]. LSDV is a brick shaped enveloped virus andit has 320 × 260 nm size, with double stranded DNA have complex symmetry and replicates in cytoplasm of the host cell (Fig1). The LSDV genome is 151 kbp large, it containa central coding area surrounded with similar 2.4kbp- inverted terminal repeats and has156 putative genes. LSDV contains 30 structural and non-structural genesas same assheep pox and goat pox virus sharing 97% nucleotide identity [8]. Loss of gene limits the host range of poxviruses insubsequent evolution and identical pattern has been seen within Capri poxviruses after comparing SPPV, GTPV and LSDV. The terminal areas of LSDV virus encodes nine genes including IL-1 receptor, vaccinia virus F11L, N2L, K7L genes, myxoma virus M003.2 and M004.1, LSDV unique gene LSDV132 with likewise virulence and host range functions disrupted by accumulated mutations both in SPPV and GTPV. However, this disruption does not affect the sequence length of genome of three viruses but absence of these genes in SPPV and GTPV suggests the role in host restriction to bovines

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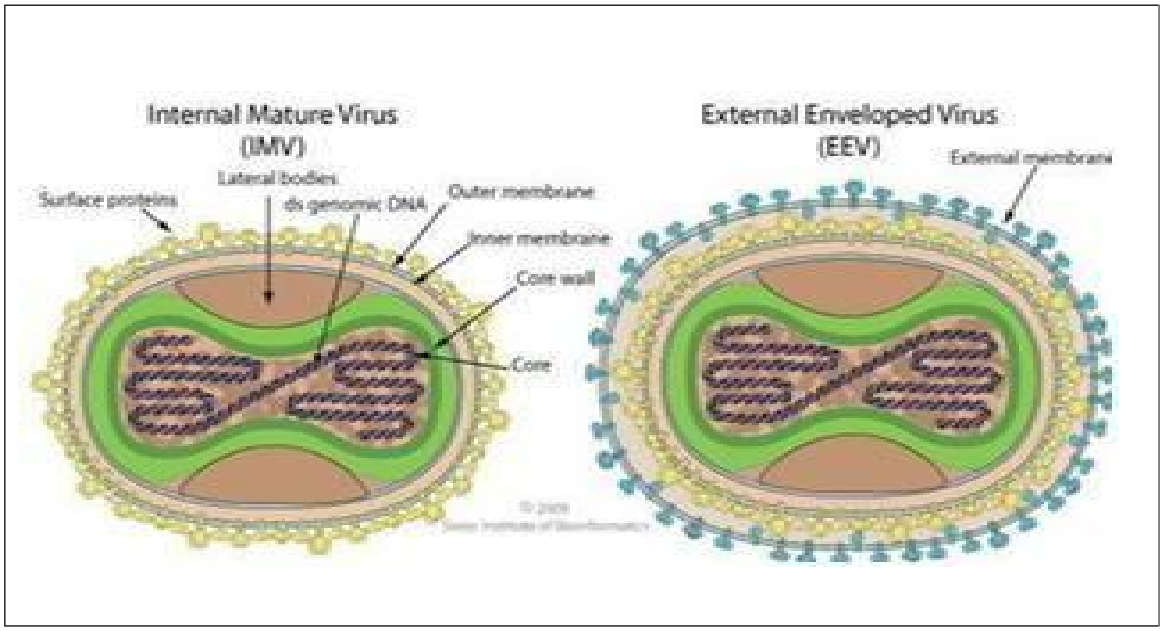
only [9][10]. In comparison with other Chordo pox viruses, LSD virus has146 conserved genes encoding proteins involved in DNA replication, transcription, mRNA synthesis, nucleotide metabolism, structure formation and stability, virulence and host range. The central region genes shareaverage of 65% collinearity and amino acid identity with gene of other poxvirusin particular with sui pox virus, leporipoxvirus, and yata pox virus. The terminal region, there is difference in the genes involved in viral virulence and host range with either absence or disruption sharing lower percentage of amino acid identity with an average of 43% only. LSDV contains homologues genes such as interleukin-10 (IL10), IL-1 binding proteins, G protein-coupled CC chemokine receptor (GPCR), and epidermal growth factor-like



proteinwhich are found in other poxvirus genera [11].

Fig.1. Morphology of LSDV

EPIDEMIOLOGY

Lumpy skin disease has a geographical distribution [12] as the disease was first observed in 1929 in Zambia earlier, it was considered to be the result either of poisoning or a hypersensitivity due to insect bites as at that time was it the year when populations of insects biting were highest between 1943 and 1945, cases appeared in Botswana, Zimbabwe [13]. LSD was limited to countries in sub-Saharan Africa from 1929 to 1986 (Fig. 2) and it's native in most Africancountries including Madagascar. This Followed by reports of the virus in the Middle East Since 2012 LSD has been expanded onan unusually wide range throughout Middle Eastern countries as eventually yearswas reported from Oman, Yemen, Israel, Kuwait, Bahrain, Egypt, Iran, Saudi Arabia Lebanon, Jordan, and in United Arab Emerate[14]. The data examinationfrom the national disease outbreak report database during the period 2000-2009 showed that major epidemic outbreaks of LSD occurred in 2000/2001 in the northern parts of the country in Amhara and West Oromia regions. Then it lengthen to the central and the southern parts of the country, in 2003 and 2004 covering bulky parts of Oromia and Southern Nation, Nationalities and Peoples (SNNP) domains. In 2006, 2007 other large outbreak come out in Tigray, Amharaand Benishangul areas in the northern and north western parts of the country[15].Therefore from 2007 up to 2009 the outbreak number progressively increased inOromia Region situated in the central part of the country. According to 2010 annual report of Ministry of Agriculture, Animal and plant health regulatory directorate in the department of epidemiology prevalence of the disease indifferent regional state of the country shows us; 1.63%, 0.49%, 5.2%, 2.69%, 0.37%, 0.7%, and 3.8% in Addis Abeba, Amhara, Gambela, Oromia, SNNP, Somali and Tigray regions respectively. The 2011 annual report shows prevalence of; 0.36%, 1.13%, 0.22%,0.65%, 0.24% and 0.30% in Amhara, Gambela, Oromia, SNNP, Tigray areas respectively[16].

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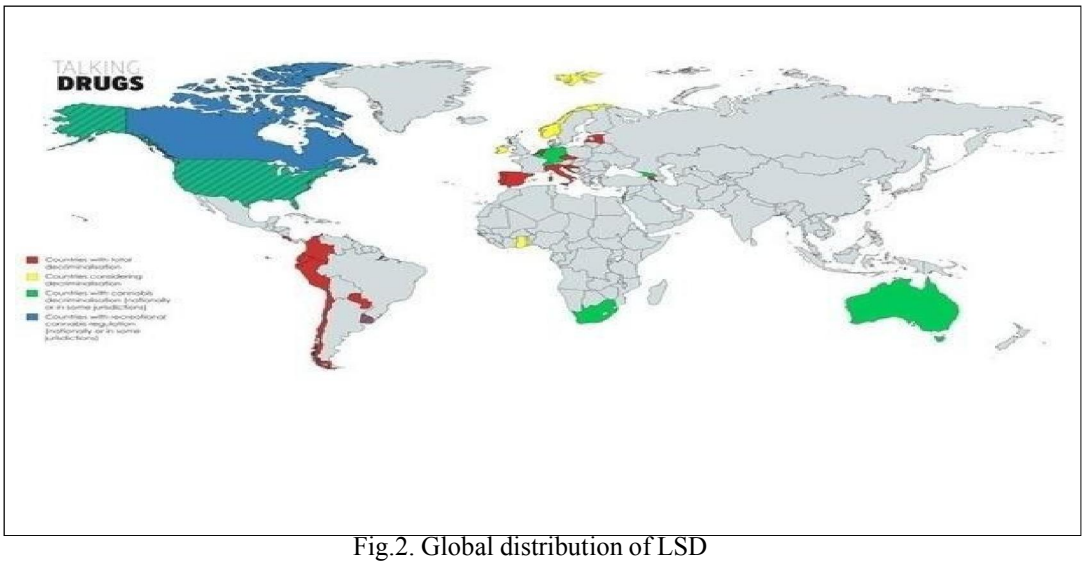

Fig.2. Global distribution of LSD

II. TRANSMISSION OF LSDV

Evaluate (Fig.3) the epidemiology of the virus the mechanism of LSDV transmission is useful. This mechanism helps for a give a strategy and extinction of disease [17,18]. An epitome for possible modes of transmission of LSDV in given fig.3

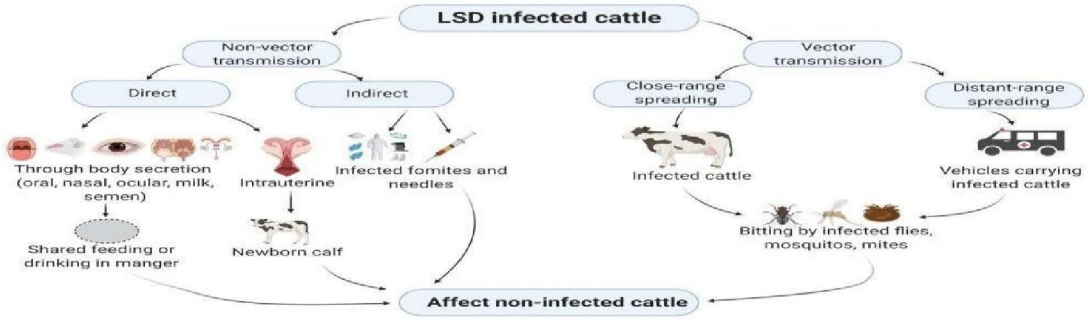

Fig.3. Epitome of possible modes of transmission of LSDV

PATHOGENESIS

There have been some (Fig.4) observations held on pathogenesis of LSD in cattles [19]. The basic symptom of LSDare fever, viremia followed by appearing inflammatory nodules and localisation in skin [20]. LSDV is also detected in saliva, semen and in nodules of skin at least for 11, 42 and 39 days after development of fever respectively [21,22]. LSD skin nodules may give serum atthe begining but develop a characteristic inverted greyish pink conical zone of necrosis. Adjoining tissue gives congestion, hemorrhages and oedema. Thenecrotic cores become isolate from the adjacent skin and are mention to as 'sit- fasts'. Multiple virus encoded factors are produced during infection, which influence pathogenesis and disease [23]. The routes which are used in experimental infections are intravenous, intradermal and subcutaneous. Severe generalized infection are being developed by intravenous route, while the intraepidermal inoculation develops only 40% to 50% of animals may developed localized lesions or no apparent disease at all. A localized swelling at the site of nodes, develop after subcutaneous or intradermal inoculation of cattle with LSDV[24].

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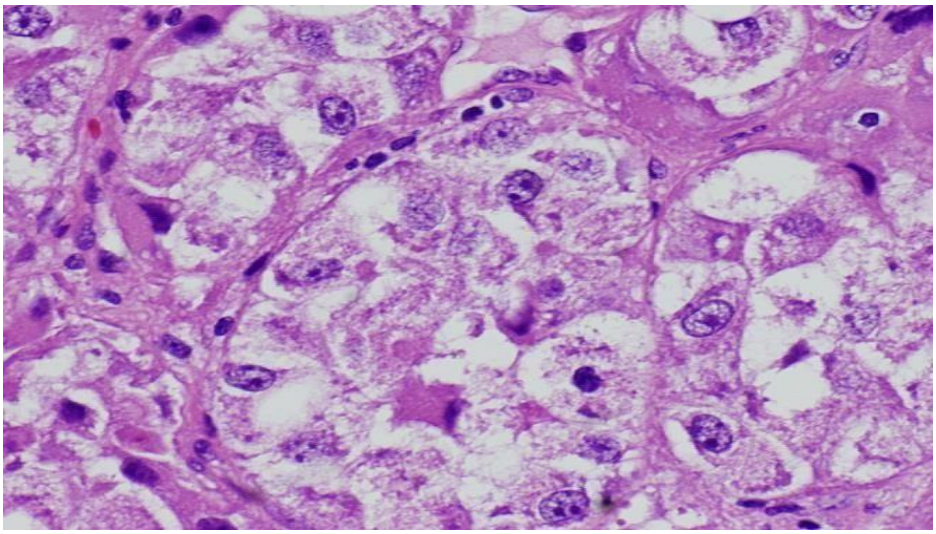

Fig.4.Pathogenesis of LSDV

DIAGNOSIS

Field diagnosis (Fig.5) of LSD is often depend on characteristic clinical signs ofthe disease. However, mild and subclinical forms require rapid and reliable laboratory testing to confirm diagnosis [25]. Molecular diagnosis with thehelp ofPCR is most easy, applicable and fast test for the diagnosis of this disease. Convinient and real-time PCR have been developed for time saving diagnosis ofLSD [26]. Differentiation of LSDV from other Capri poxvirus by real- time PCRhas been developed [27]. Rapid diagnostic verification of the indefinite field diagnosis is basic for the successful control and elimination of LSD in native andparticularly in non-native countries. There are some diseaseshaving same sign asLSD. LSD can be confused with the following diseases:

Pseudo-lumpy-skin disease

Bovine virus diarrhoea/mucosal diseaseDemodicosis (Demodex)

Bovine malignant catarrhal fever (Snotsiekte)Rinderpest

Besnoitiosis Oncocercariasis Insect bite allergies

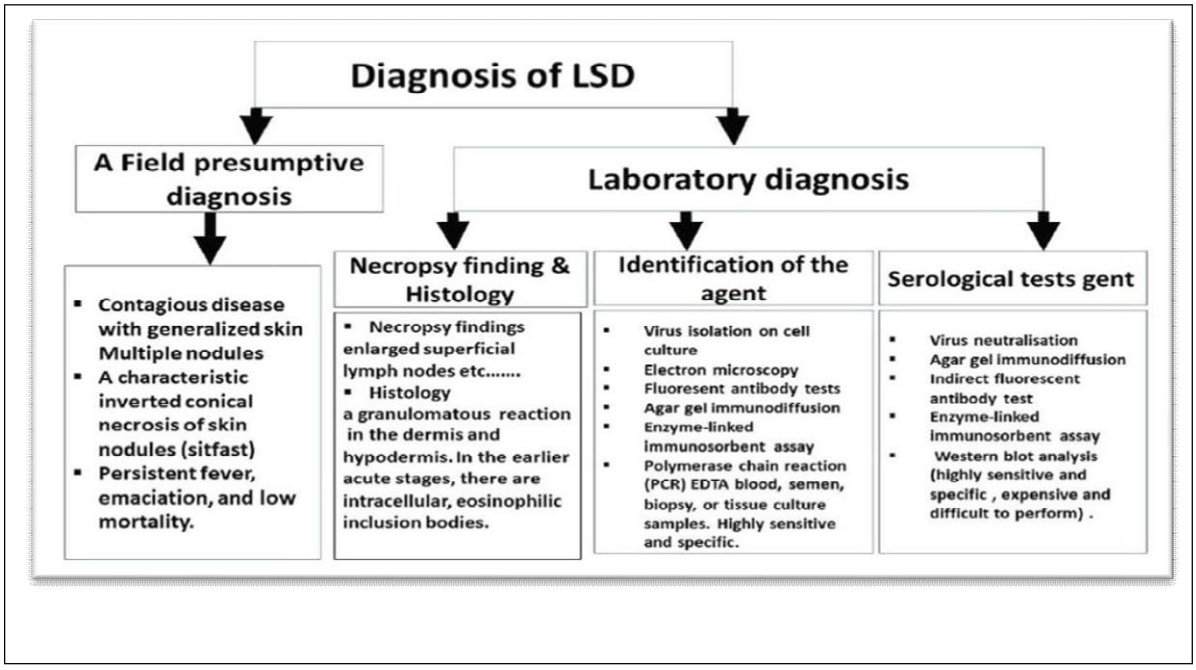

Fig.5.The diagnostic procedures of the LSD

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III. CLINICAL SIGN AND LESSIONS

The incubation (Fig 6) period of the disease in natural conditions ranges from 2 to 5 weeks, while in experimental conditions it ranges from 7 to 14 days. There are three forms of LSD: acute, subacute and chronic. The disease beginswith a biphasic fever. The clinical manifestations of a mild infection are fever, weakness, eye discharge, and massesof one or two nodules within a few days ofthe onset of anthrax. Subsequently, painful, hyperemic nodular lesions were observed on the body of the animal, especially on the muzzle, nostrils, back, legs,scrotum, perineum, eyelids, lower ears, mucous membranes of the nose and mouth, and the skin of the tail [28].

Fig.6. LSD in cattle

ECONOMIC IMPORTANCE

The World Organization for Animal Health (OIE) has classified LSD as a diseaseof note due to its economic impact.It has been considered an agent of agroterrorism due to its ability to spread from Africa to other parts of the world [29]. Animal degradation affects the entire trade in live animals and animal products. This can lead to huge economiclosses in the meat, dairy, leather and other industries related to livestock and their by-products. in danger. Total losses of milk, meat, beef, drafts, treatments and vaccines in Ethiopia have beenestimated at $6.43 per person for native Zebus and $58 per person for Holstein Friesians (30).

TREATMENT

Lumpy skin disease is caused due to virus and hence has no identified cure. However, to treat secondary bacterial infections or to deal with fever 10 or inflammation or to improve animal's apetite antibiotvs, anti-inflammatory drugsor shots of vitamins are used.

Vaccination in Endemic Areas:

Immunity gained from natural infection of the disease might be lifelong and vaccination can be used successfully. Vaccination of cattle every year can controlLSD. It is possible to protect cattle against LSD using strains of Capri poxvirus derived from either of the sheep or goats as used in Egypt by Romanian sheep pox strain [31].

Vaccination in New Areas:

Risks of introducing of the disease in to the new areas are by infected animals and contaminated materials [32]. Ring vaccination of cattle, quarantination of animals and movement of animals should be restricted. These two practices of vaccination were followed by Egypt and Israel and it was effective [33].

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The following vaccines have been used in protection of the animals:

Homologous live attenuated virus vaccine (Neethling strain: immunity conferredlasts up to 3 years).

Heterologous live attenuated virus vaccine (Sheep or goat pox vaccine, but may cause local, sometimes severe reactions) (Fig 7). This vaccine is not advised in countries free from sheep and goat pox because the live vaccines could otherwiseprovide a source of infection for the susceptible sheep and goat population.

Fig.7. Vaccine for LSD

IV. CONCLUSION

Lumpy dermatosis (LSD) is a contagious, eruptive and sometimes fatal bovine disease, a systemic skin disease caused by Tamil virus, a relative of Nicelingpoxvirus in thegenus Capripoxvirus of the family Poxviridae. Cattle andbuffaloes are important livestock that make a significant contribution to the globaleconomy. Rough skin disease is a serious disease of cattle and buffaloes. LSD was first reported in Zambia, found in most other African countries, is now endemic in most African countries and has recently spread from Africa to the Middle East, Asia and Europe. Due to the economic importance of the disease, the spread of the disease to the wider geographical area of the Indian subcontinentwill certainly hamper rural economies in particular. LSD may also lead to a decline in livestock and livestock exports. The reason for the arrival of LSD in India needs to be studied along with random epidemiological screenings in different regions to get a true prevalence of the disease. Apart from these effective quarantine and vector control methods, vaccination is the only way to prevent disease.

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