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REVIEW ARTICLE

A review: Lumpy skin disease and its emergence in India

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Abstract

Lumpy skin disease (LSD) is a viral disease caused by lumpy skin disease virus (LSDV), a member of *Capripoxvirus* genus of *Poxviridae* family. It is a transboundary disease of the economic importance affecting cattle and water buffaloes. The disease is transmitted by arthropod vectors and causes high morbidity and low mortality. LSD has recently been reported first time in India with 7.1% morbidity among cattle. Generally, fever, anorexia, and characteristic nodules on the skin mucous membrane of mouth, nostrils, udder, genital, rectum, drop in milk production, abortion, infertility and sometimes death are the clinical manifestations of the disease. The disease is endemic in African and Middle East countries but has started spreading to Asian and other countries. It has been recently reported from China and Bangladesh sharing borders with India. We have summarized occurrence of LSD outbreaks in last 10 years in Asian countries for the first time. In India, currently epidemiological status of the disease is unknown. Vaccination along with strict quarantine measures and vector control could be effective for preventing the spread of the disease. This review aims to summarise the latest developments in the epidemiology with the focus on transboundary spread, aetiology and transmission, clinical presentations, diagnostics and management of the disease.

Keywords Lumpy skin disease · Transboundary spread · Outbreak · India

Introduction

Lumpy skin disease is an infectious viral disease caused by Lumpy skin disease virus (LSDV) of *Capripoxvirus* genus, subfamily *Chordopoxvirniae*, family *Poxviridae*. The disease is known by various names such as "LSD", "Pseudo-urticaria", "Neethling virus disease", "exanthema nodularis bovis", and "knopvelsiekte" (Al-Salihi 2014, Tuppurainen et al. 2017). LSD is a non-zoonotic, vector borne and transboundary disease with limited host range and currently restricted to ruminants viz. cattle and water buffaloes. The arthropod vectors responsible for the disease spread include biting flies, mosquitoes and ticks (Tuppurainen et al. 2011; Lubinga et al. 2013a, b). Natural infection of sheep and goat has not been reported even in close contact with infected cattle and buffaloes but skin lesions have been seen after experimental infection in sheep, goat, giraffe, Giant gazalles, impalas (Davies 1991). LSD is associated with high morbidity but low mortality (Abutarbush et al. 2013). The disease is characterized by fever, lymph node swelling, circumscribed nodules on skin causing severe emaciation, reduction in milk production, infertility. Overall, it affects the economic value of animal as it will affect the meat and milk production, hide quality, draft power of animals and reproductive efficiency (abortion and infertility), (RGBE 2014). It is a notifiable disease having devastating effect on international livestock trade also. The disease is endemic in African countries but recently the disease has been reported from new territories around the world. The first case of LSD was reported from Zambia in 1929 (Morris 1931) and then in southern and northern African countries. Later on, it spread to Israel, Kuwait, Oman and Yemen (Wainwright et al. 2013). According to OIE, at present this disease is prevalent in countries including various African, European and Asian countries (Tuppurainen et al. 2015). The reasons of the disease spread to India are unknown but it may be due to livestock movement across international borders or may be due to vectors movement from the neighbouring countries. In recent years, LSD has been reported from countries neighbouring India like China and Bangladesh. Therefore, understanding the epidemiology of exotic diseases becomes necessary for timely planning the effective disease management. This review summaries the latest updates about the LSD.

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Lumpy skin disease

Aetiology

Lumpy skin disease virus (LSDV) causing Lumpy skin disease belongs to *Poxviridae* family that contains group of viruses causing diseases in most of the domestic animals except dog. The family contains two subfamilies: *Chordopoxvirinae*, infecting vertebrate host and *Entomopoxvirinae* infecting invertebrate hosts (Quinn et al. 2016). The *Chordopoxvirinae* subfamily comprises 10 genera including *Capripoxvirus* genus. This genus contains viruses of three species, sheeppox virus (SPPV), goatpox virus (GTPV) and lumpy skin disease virus (LSDV) infecting sheep, goat and cattle, respectively (King et al. 2012). LSDV is a brick shaped enveloped virus, 320 × 260 nm size, with double stranded DNA have complex symmetry and replicates in cytoplasm of the host cell. The LSDV genome is 151 kbp large, consists of a central coding region surrounded by identical 2.4 kbp-inverted terminal repeats and contains 156 putative genes. LSDV contains 30 structural and non-structural genes homologous to sheeppox and goatpox virus sharing 97% nucleotide identity (Tulman et al. 2001, 2002). Gene loss limits the host range of poxviruses in subsequent evolution and same pattern has been observed within *Capripoxviruses* when comparing SPPV, GTPV and LSDV. The terminal regions of LSDV virus encodes nine genes including *IL-1* receptor, vaccinia virus *F11L, N2L, K7 L* genes, myxoma virus *M003.2* and *M004.1*, LSDV unique gene LSDV132 with likely 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 only (Tulman et al. 2002; Biswas et al. 2019). In comparison with other *Chordopoxviruses*, 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 share average of 65% collinearity and amino acid identity with gene of other poxvirus in particular with suipox virus, leporipoxvirus, and yatapox 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 (*IL-10*), IL-1 binding proteins, G protein-coupled CC chemokine receptor (*GPCR*), and epidermal growth factor-like protein which are found in other poxvirus genera (Tulman et al. 2001).

Stability of virus

The virus is stable in ambient conditions for long period. It can persist in desiccated skin crusts for 35 days, in necrotic nodules for 33 days and in air-dried hides for at least 18 days. Sunlight and lipid detergents can destroy virus quickly but virus can persist for many months in dark environment like animal sheds and feed stores. Virus gets inactivated at 55ºC temperature for 2 h and 65ºC for 30 min. It is susceptible to highly alkaline or acid pH but can sustain pH 6.6–8.6 for 5 days at 37°C without significant reduction in titres. The virus is susceptible to ether (20%), chloroform, formalin (1%), phenol (2% for 15 min), sodium hypochlorite (2–3%), iodine compounds (1:33 dilution) and quaternary ammonium compounds (0.5%) (OIE 2013). LSDV is very stable and can be recovered even after 10 years from the skin nodules kept at -80ºC and after 6 months from the infected tissue culture fluid kept at 4ºC (Mulatu and Feyisa 2018).

Transmission

Mechanical transmission by vectors is the prime route of spread of disease. In most of the endemic countries like sub Saharan Africa, Egypt and Ethopia, the disease incidences significantly increase with the onset of seasonal rains and summer season, coinciding with the peak activity of the vectors (Mulatu and Feyisa 2018). Incidences decrease significantly with the onset of winters and reappears with arrival of spring and summer. It was observed that despite restricted animal movements from Egypt, infection spreads to Israel, 80 to 200 km away through air movement of bitting insects (AU-IBAR 2013). The reduction in cases during dry condition with no insects or low insects density has confirmed the role of insect vectors in disease spreading rather than by direct or indirect contact (Nawathe et al. 1982; Kondela et al. 1984), those are considered inefficient routes (Gumbe 2018; Carn and Kitching 1995). The tick *Amblyomma* spp., *Rhipicephalus decoloratus, Rhipicephalus appendiculatus and Amblyomma hebraeum* have been reported as a mechanical vectors and reserviors of virus (Ali and Obeid 1977; Lubinga et al. 2013a b; Lubinga 2014; Tuppurainen et al. 2013a, b). The bitting flies (*Stomoxy calictrans and Biomyia fasciata*) and mosquitoes (*e.g. Culex mirificens* and *Aedes natrionus*), are also involved in mechanical transmission of disease. The evidence of direct transmission of LSDV is scarce but the experimental studies and field observations of Weiss 1968 concludes the low rate of transmission by direct route. Whereas, there were studies concluding that direct contact of animals has no role in transmission of virus (Carn and Kitching 1995; Magori-Cohen et al. 2012). Virus is secreted in milk, nasal secretions, saliva, blood and lachrymal secretions forming indirect source of infection for animals sharing feeding and watering troughs (Ali et al. 2012). LSD virus transmission through intrauterine route has been documented in literature (Rouby and Aboulsoud 2016). The infection has been assumed to be transmitted from infected mother to calf *via* milk secretions and skin abrasions (Tuppurainen et al. 2017). The virus persists in the semen for up to 42 days

post-infection (Irons et al. 2005) and it has been established by experimental infection (Annandale et al. 2013). Iatrogenic route can be another route of spread of virus when single needle used for mass vaccination that can acquire the virus from the skin scabs or crusts (Mulatu and Feyisa 2018). The summary of transmission of virus is shown in Fig. 1. Therefore, it suggests that quarantine could not be the only method to prevent the spread of LSD as movement of vector can blow out the disease (EFSA 2015).

Host range

Cattle (*Bos indicus* and *Bos taurus*) and buffalo (*Bubalus bubalis*) are susceptible hosts. *Bos taurus* is more susceptible than indigenous cattle breeds. Animals of all ages are susceptible but calves are more susceptible and develop lesions within 24 to 48 h (Al-Salihi 2014). Wild animals under natural conditions, are resistant to infection but experimental infection produced clinical lesions in Giraffe (*Giraffe camelopardalis*) and impala (*Aepyceros melampus*), Arabian oryx (*Oryx leucoryx*), springbok (*Antidorcas marsupialis*), and oryx (*Oryx gazelle*) and Thomson's gazelle (Davies 1991; Padilla et al. 2005). Normally the role of wildlife in the transmission and maintenance of LSDV has been found almost negligible. Humans are also resistant to the virus (OIE 2013).

Transboundary spread

The disease made its first emergence in Zambia in 1929 and which later on spread to whole African continent except Libya, Algeria, Morocco and Tunisia (Tuppurainen and Oura 2012). With increasing demand of food, Middle east countries have increased the transportation of animals from neighbouring countries. LSD infection in Egypt in 1988 was due to the movement of infected cattle from affected African countries. In 2006 again, the disease re-emerged due to un restricted movement of cattle from African horn countries (Ali et al. 1990; Fayez and Ahmed 2011). The first outbreak in Israel in 1989 was thought to be due to the movement of infected *Stomoxy calcitrans* from Egypt (Yeruham et al. 1995). The data of reported outbreaks as documented in OIE disease outbreak report during the period of 2010–2019 has been shown in Fig. 2. From 2012 to 2013 the disease appeared for the first time in Syria, Jordan, and Lebanon. The outbreak in Jordan appeared near the border of Israel and Syria

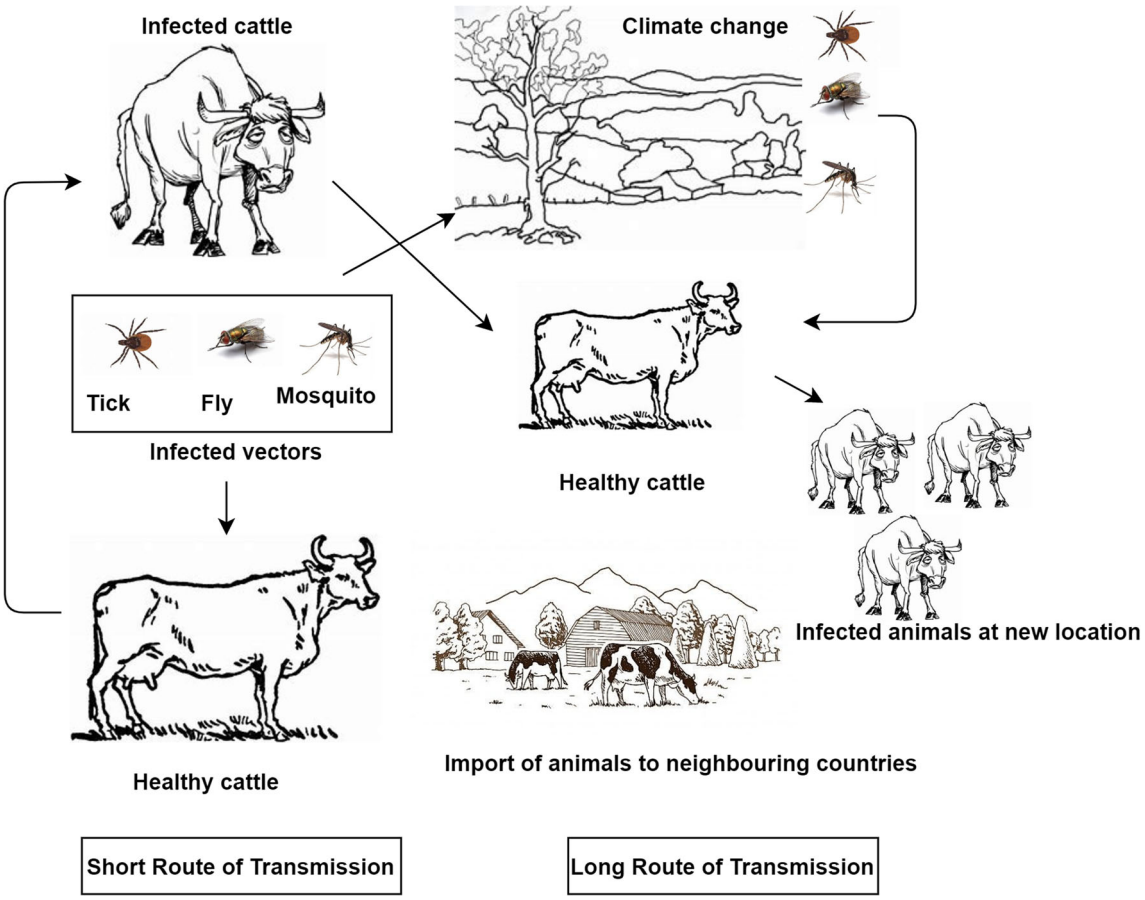

Fig. 1 Summary of transmission of LSD virus

indicating the transboundary spread of disease (Abutarbush et al. 2013). The disease further spread to other nearby countries like Turkey and Iraq in 2013 and Iran in 2014. Later, LSD was also reported from Cyprus, Azerbaijan and Turkey (Data not available in OIE) (BY-ND 2016)). From the OIE report, LSD has been re-emerged in Israel after 6 years in 2019 due to decrease in vaccination of animals, which was earlier mandatory for the animals (European Food Safety Authority et al. 2020). Figure 2 shows the temporal distribution of number of outbreaks in Asian Countries from 2010 to 2019 (Data source: OIE Disease Information). For the first time, LSD outbreaks were reported from India, China and Bangladesh sharing boundaries with each other. In the month of August, 2019, China reported the first incidence of disease with 65 animals affected in the village Illi Kazakh Autonomous Prefecture, near the border of Kazakhstan. The last outbreak reported in Kazakhstan was in 2016. Similar disease outbreak was reported in Bangladesh in the months of July and September, 2019, where 66 animals were affected out of 360 exposed animals. In 2019, diseases have been re-emerged in south province of Turkey and east province of Russia (European Food Safety Authority et al. 2020). In India, first outbreak of the disease was reported in Odisha state in the month of August (2019), in monsoon season with high humidity and vector density. The first incident started on 12 August 2019, in Khairbani, Betnoti, Mayurbhanj districts of Orissa, where in a farm of 135 animals and 9 cases were reported. Then after few days, second outbreak was reported from the same region at new place Patalipura, where in a farm of 441 susceptible animals, 20 LSD cases were observed. With no time third case outbreak was reported on 20 August 2019 in Rajendrapur, Bhandaripokhari, Bhadrak, Odisha, with in a farm of 356 animals and 50 cases (https://www.oie.int/). In first published report of LSD in India, it was found that out of 2539 animals, 182 were positive with no mortality but 7.1% morbidity. On the basis of phylogenetic analysis, the strain present in India was genetically close to South African NI2490/KSGP-like strains rather than European strains (Sudhakar et al. 2020).

Clinical signs and lesions

The incubation period of disease in natural condition is between 2 and 5 weeks but in experimental condition, the duration ranges from 7 to 14 days. The LSD takes three forms: acute, subacute and chronic form. The illness begins with biphasic fever. The clinical manifestations in mild form of infection appears as one or two lumps of nodules within 2 to 3 days of onset of fever, emaciation, ocular discharge, agalactia. Later on, nodular lesions, which are painful and hyperemic may be observed on the animal body especially in the skin of the muzzle, nares, back, legs, scrotum, perineum, eyelids, lower ear, nasal and oral mucosa, and tail (Salib and Osman 2011). In severe condition, more than hundred nodules developed on skin all over the body and this stage persist for 7 to 12 days. The nodules are firm and slightly raised from surrounding skin, separated by narrow

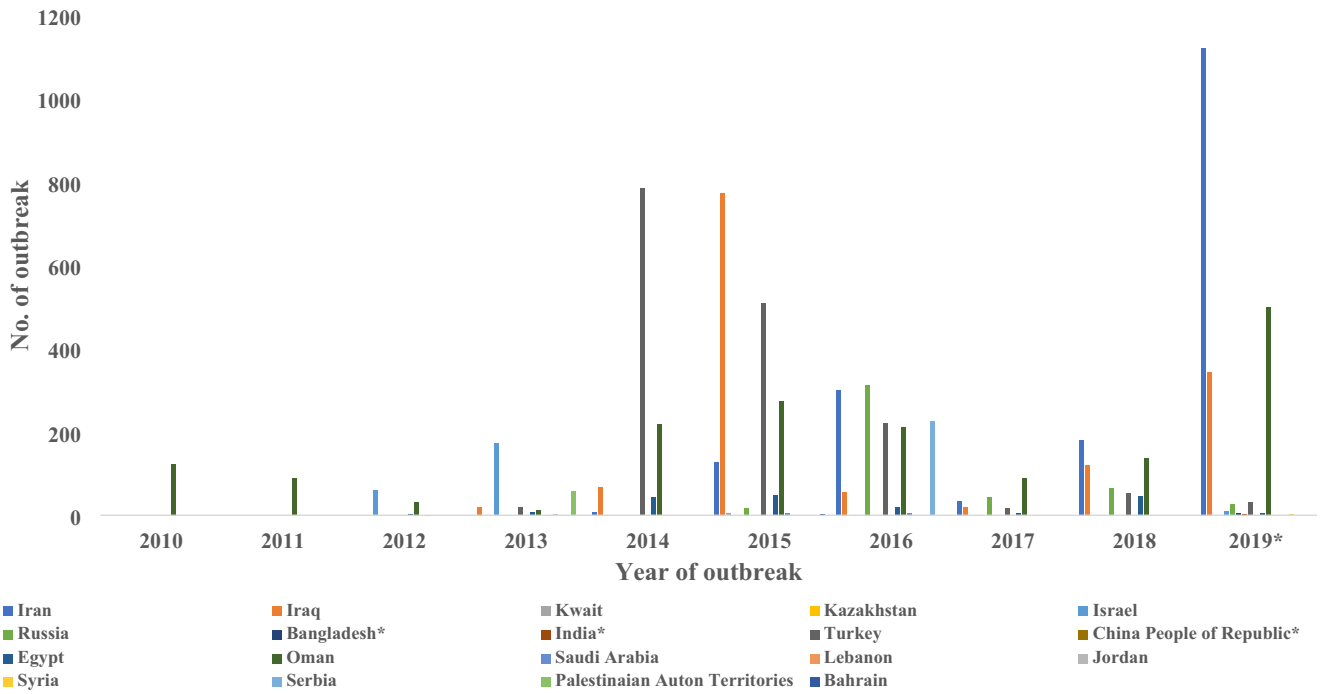

Fig. 2 Temporal distribution of LSD virus in Asian countries from 2010 to 2019 (Data source: OIE disease record) * mark shows the countries where disease appeared recently in 2019

haemorrhagic ring. The nodules involve dermis, epidermis, adjacent subcutis and musculature. The lesions then progress towards papules, vesicles, pustule with exudation and then slowly to scab formation. Healing of the lesions is very slow. With time lesions develop on mucous membranes of nostrils, respiratory tract, mouth and vulva. After 2-3 weeks, the cutaneous lesions become harder and necrotic causing discomfort to animals and they become reluctant to move. The sloughing of the lesions may create hole form "sitfast", the characteristic lesion, which subsequently cause invasion by screwworm fly and bacterial invasion that can further lead to septicaemia (Abutarbush et al. 2013; Constable et al. 2017). The generalized lymph node swelling also observed in infected animals. In histopathology, lesion of lumpy skin disease show ballooning degeneration of epithelial cells, presence of eosinophilic intracytoplasmic inclusions bodies (Tuppurainen and Oura 2012). The sequela of LSD is pneumonia due to the inhalation of necrotic material by the animal itself. Abortion occurs in acute phase of infection. The infertility is another sequela of the disease in both male and female. Female remains in anoestrus for long time. Infected bulls with lesions on genital region also remain infertile for months. Recovery is very slow due to secondary bacterial infection, pneumonia, mastitis and fly strike in necrotic lesions leaving deep holes in the body (Al-Salihi 2014).

Economic importance

The world organization for animal health (OIE) categorises the LSD as notifiable disease due to its economic impact. LSD has been considered as agro-terrorism agent due to its ability to spread from Africa to other parts of world (Abutarbush 2017). The economic implications of the disease are high due to morbidity rather than mortality, as the mortality rate is usually low. The significant losses are due to severe emaciation, hide damage, infertility in males and females, mastitis, drop in milk production and abortions (Tuppurainen and Oura 2012). Due to reduction in quality of the animal, the effect can be seen in overall trade of the live animals and animal products. This may cause huge financial losses to meat industry, milk industry, leather industry and other industries associated with livestock and its by-products. Not only industries, poor farmers holding the livestock have to suffer the crisis due to the disease. Total losses on the account of milk, meat, beef, power of draft, treatment and vaccination, in Ethiopia, were estimated to be 6.43) USD per head in local zebu and 58 USD per head for Holstein Friesian (Gari et al. 2010). In an outbreak in Jordan, supportive antibiotic treatment cost was estimated to be 27.9 British pounds per head (Abutarbush et al. 2013).

Diagnosis

The diagnosis of exotic diseases is little challenging due to lack of familiarity and logistics. In case of LSD, clinical signs can be confused with other diseases like foot and mouth disease (FMD), insect bite, demodicosis and hypersensitivity. Tentative diagnosis can be made on the basis of skin nodules observed on face, eyelid, neck, muzzle, nostrils, udder, limbs. Skin biopsy sample can be collected for further confirmation of disease. Samples should be transported in transport medium with 20 to 50% glycerol in phosphate buffer saline. Skin samples can be checked by electron microscopy to identify virus (Davies et al. 1971). Samples of skin also show characteristic histopathological changes, which include vasculitis and perivascular infiltration with white cells causing a thrombosis of the vessel in the dermis and subcutis. Cells infiltrating the lesion are epithelial cells, known as "celles clavelauses", which are also described in sheep pox. Agar gel precipitation test is not specific for LSD as the antigen of LSDV is shared with other capripoxvirus and parapox viurs.

Virus isolation can be used for the confirmatory diagnosis in new niches. The bovine testes and pre-pubertal lamb, primary and secondary culture is most sensitive for isolation of virus.

Molecular diagnosis with PCR is most efficient and rapid test for the diagnosis of disease. Conventional and real-time PCR have been developed for rapid diagnosis (Heine et al. 1999; Mangana-Vougiouka et al. 1999; Orlova et al. 2006; Tuppurainen et al. 2005; Zheng et al. 2007; Bowden et al. 2008). Differentiation of LSDV from other *Capripoxvirus* by real-time PCR has been developed (Lamien et al. 2011).

Prevention and control

Till date no effective treatment against LSD has been developed. Anti-inflammatory and antibiotics are used for symptomatic treatment. To control the disease, effective control and preventive measures need to be implemented, which include:

a) *Restrict movement*: Movement of infected animals with LSD should be strictly prohibited to prevent the spread of transboundary disease. Within countries, if animal with such lesions are observed, they should be quarantined for inspection to prevent the rapid spread of disease. b) *Restrict vector movements*: Vectors movement due to prevailing winds may cause disease transmission. Vector control methods like use of vector traps, use of insecticides can also be used for preventing the disease. c) *Vaccination*: A live attenuated vaccine is available for LSD. Based on different strains of LSD virus, companies prepared vaccines. It is either based on Neethling strain like Lumpy Skin Disease Vaccine for Cattle

(Onderstepoort Biological Products; OBP, South Africa) or Bovivax (MCI Sante Animale, Morocco), or based on SIS Neethling type (Lumpyvax, MSD Animal Health-Intervet, South Africa). As LSD is closely related to sheeppox and goatpox virus, vaccine against sheeppox and goatpox can be used for LSD (Tuppurainen et al. 2015). Different strains of virus used as vaccine strain as per OIE. Homologous Lumpy skin disease virus Neethling strain from South Africa, passaged 60 times in lamb kidney cells and 20 times on the chorioallantoic membrane of embryonated chicken eggs provides immunity for 3 years. Sheeppox vaccines used against LSD includes Kenyan sheeppox virus passaged 18 times in lamb testis (LT) cells or fetal calf muscle cells, Yugoslavian RM 65 sheep pox strain, Romanian sheep pox strain. The heterologous vaccine strains cause some local reactions. These vaccines are not advised in sheeppox and goatpox affected areas as such vaccines may serve as source of infection for susceptible population of sheep and goat. Live attenuated Gorgan goatpox strain provide good protection in cattle with practically no side effect (Gari et al. 2015; Brenner et al. 2009; Capstick and Coakley 1961 1962; Carn et al. 1994). For effective control and prevention of disease, long term vaccination with 100% coverage should be made mandatory as LSD virus being stable survives in environment for long time. Before introducing new animals to the affected farm, they should be immunized. Calves should be immunized at the age of 3 to 4 months raised from mothers, who are vaccinated or naturally infected. Pregnant cows, breeding bulls can be vaccinated annually (Tupprainen et al. 2015).

Conclusion

Cattle and buffaloes are important livestock contributing substantially to the world economy. Lumpy skin disease is a serious disease of cattle and buffalo. Earlier the disease was restricted to African countries and few other countries but the recent spread of disease to India and other Asian countries, previously disease-free region, is a matter of concern for the livestock rearing sector as most of these countries have agriculture-based economies. As this disease in economically important, spread of this disease to larger geographical regions of Indian subcontinent will surely hamper the rural economy in particular. LSD can also lead to reduction in export of livestock and livestock products. The reasons behind the entry of LSD in India need to be investigated along with epidemiological random screening in different regions to access the actual disease prevalence. Besides, effective quarantine methods, vector control methods, vaccination is the only method to prevent the disease.

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Compliance with ethical standards

Conflict of interest The authors declare that they have no competing interest.

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Consent to participate Not applicable.

Consent for publication Not applicable.

Code availability Not applicable.

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