INTERNATIONAL JOURNAL OF PHARMACEUTICAL SCIENCES
[ISSN: 0975-4725; CODEN(USA):IJPS00]
Journal Homepage: https://www.ijpsjournal.com

Review Article

Lumpi Skin Disease Virus Is Emerging Challenging For Livestock In Pakistan

Hafsa Arshad , Muhammad Nadeem*, Rizwan Shaukat

Department of Biochemistry, University of Okara, Pakistan

ARTICLE INFO

Received: 02 June 2024
Accepted: 06 June 2024
Published: 12 June 2024
Keywords:
Lumpi, Skin, Disease, Liver, Liverstock, Viruses
DOI:
10.5281/zenodo.11612906

ABSTRACT

Lumpy virus cause the lumpy skin disease in live-stock animals like cattle and buffalo belong to Capri poxvirus genus and Poxviridae family, wide spread in across all country much damage the livestock sector in Pakistan reported in 2022. Cattle were more affected by lumpy virus than buffalo in Pakistan Transmission of lumpy virus mostly carry out via arthropods including as ticks and insects as well as toxic substances during blood feeding, has linear DNA in genome structure of 151 kb showed high stability against intense temperature. Lumpy virus affects badly to domestic animals including cattle and buffalo, mainly symptoms including appearance of lesion on skin, high fever and respiratory and digestive tract mucous membrane. ELISAs, real-time PCR and Indirect fluorescent antibody test effectively use in diagnosis of lumpy virus. This disease leads to huge economic losses such as abortion, reduction of milk and decline in infertility This review is also illustrating variety of vaccine recommended by authority to help full to minimize lumpy skin disease imposed by LSDV. There is no mostly effective treatment and vaccine available, only control and prevention encouragement in block and eradication of LSD.

INTRODUCTION

Lumpy skin disease (LSD), a major threat to stockbreeding, can cause acute or subacute disease in cattle and water buffalo (Givens, 2018; Tuppurainen, Venter, et al., 2017). All ages and breeds of cattle are affected, but especially the young and cattle in the peak of lactation (Tuppurainen et al., 2011). The reason why the World Organization for Animal Health (OIE) has placed this transboundary disease on the notifiable disease list is due to its significant economic losses and the potential for rapid spread (Tuppurainen & Oura, 2012). The recent spread of the disease in disease-free countries indicates the importance of its transmission, as well as control and eradication (Sprygin et al., 2019). Lumpy Skin Disease is a contagious malady, produced by the causative agent Lumpy skin disease virus (LSDV), which

***Corresponding Author:** Muhammad Nadeem
Address: *Department of Biochemistry, University of Okara, Pakistan*
Email : mn.ladheywal@gmail.com
Relevant conflicts of interest/financial disclosures: The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

belongs to Capripoxvirus genus, subfamily Chordopoxvirniae and family Poxviridae. “Neethling virus disease”, “exanthema nodularis bovis”, “Pseudo-urticaria”, and “knopvelsiekte” are some of the names given to this disease yet amongst such forenames, “LSD” is the one most commonly employed (Carn, 1993; Capstick and Coackley et al., 1961). LSD is a vector borne pox viral disease of cattle and buffaloes that is characterized by appearance of skin nodules. The natural hosts for this virus are cattle and Asian water buffaloes (Elhaig et al., 2017). Considering the potential for international spread and economic impacts the World Organization for Animal Health (OIE, 2020) classified the disease as notifiable disease. LSD causes huge economic impact on livestock sector due to decreased milk production, loss of draught power, damage to skin, trade restrictions, loss of body condition, abortions, infertility and the cost of veterinary care. The disease was first identified in Zambia in 1929. The LSD has spread from its origin’s in central Africa to the middle east, Europe and Asia with rapid spread occurring since 2013. India reported first time in the year 2019 in Odisha state. The surrounding countries of India viz., Bangladesh, China, Nepal, Bhutan, Myanmar, Sri Lanka, Pakistan have also reported the LSD outbreaks (Gupta et al., 2020). LSD has spread to a number of countries in Asia, since 2019. Recently, LSD has been detected in India, China, and Iran, all of which share Pakistan's border possibly indicating a transboundary transmission channel from Iran and India, both of which border southern Pakistan The outbreak started in October 2021. In November 2021, Sindh livestock department started investigating an unknown skin disease in cattle which was spreading rapidly in different parts of Sindh province with significant mortality. Cows in Sindh and southern areas of Punjab provinces are still infected with LSD. Insect control, Quarantines, depopulation, cleansing and disinfection of infectious farms/herds are useful ways to reduce disease burden, but vaccination is the most effective prevention and control strategy. The GTPV&SPPV vaccination is effective against the LSD virus due to antigenic similarities (Imran et al., 2022).

EPIDEMIOLOGY

LSDV has started from Zambia in 1929 (Morris 1931) and insects were considered the main vector of disease spread. Later, virus was observed in Zimbabwe, Botswana and South Africa between 1943-1945 (Von Backstrom,1945). In this outbreak approximately eight million cattle were affected and disease continued till 1949 (Thomas and Mare 1945; Diesel, 1949). In 1957and 1972, LSD was reported in Kenyaand Sudan, respectively(Ali and Obeid 1977) while West Africa in 1974, Somalia in 1983 and Senegal, Mauritius and Mozambique in 2001. Now LSD has been widely spread and invaded majority countries especially African countries except Algeria, Libya, Tunisia and Morocco (Tuppurainen and Oura 2012). It has reported in Oman, Kuwait, Egypt, Israel, and Bahrain in 2009, 1991, 2006, 2002-2003, respectively (Kumar 2011; Tageldin 2014; Fayez and Ahmed 2011; Ali and Amina 2013;Shimshony and Economides 2006; Sherrylin et al 2013). This virus has then re-emerged in 2009 from a farm population of 3200 cattle in Oman (Tageldin et al 2014). Now this virus has spread in many countries including Pakistan. In these days, Pakistan is facing dangerous problems of LSDV almost in all districts.

TRANSMISSION

Lumpy skin disease can affect buffalo, cattle, and many wild animals. Small ruminants like goats and sheep were not affected by the virus as reported by El-Nahas et al. (2011) and Lamien et al. (2011) while Baldacchino et al. (2013) observed LSD in sheep and goats. During trade of various products

such as contaminated meat, hides, fresh milk, carcasses, offal, susceptible hosts are come into contact with these poisonous or contaminated products. It has been reported that hematophagous arthropods like insects and ticks feed on the blood of infested animals and transmit toxic materials to other animals during blood feeding (Annandale et al., 2013; Lubinga et al., 2015).

GENOME STRUCTURE

LSD is a viral contagious cattle disease caused by Lumpy skin disease virus (LSDV; Murphy et al., 2008). The virus is a large linear double-stranded DNA genomes of 151 kb and belongs to one of the Capripoxvirus genus, subfamily Chordopoxvirniae, family Poxviridae (Tulman et al., 2001; Bhanuprakash et al., 2006; Moss, 2007; K, 2014). Viruses of the Poxviridae family are very similar in morphological characteristics (Mcfadden, 2005). Since the researchers have not yet resolved the particle pattern diagram of LSDVThe Capripoxvirus genus consists of SPPV, GTPV and LSDV (Tulman et al., 2001; Zare, 2010). These three viruses could cause transboundary disease with serious consequences among the ruminants, causing a major threat to the global animal husbandry (Sprygin et al., 2018a). They all have their own specific natural reservoir. The main hosts of the first two viruses are sheep and goat, while the LSDV mainly affects the cattle and water buffalo (Afonso et al., 2012; Fagbo et al., 2014; Lefkowitz et al., 2018). In recent years, researchers have observed the appearance of LSDV under the electron microscope, which is indeed similar to the appearance of other virus members in the Poxviridae that have been published (Sanz-Bernardo et al., 2020). The length of the virus genome is 151 kb, which consists of a central coding area and a 2.4 kb inverted terminal repeat sequence on both wings. According to the scientific prediction, LSDV has 156 putative genes. It has nine more genes than the other two viruses in the genus. Its morphological characteristics are similar to poxvirus, about 350 nanometers in length and 300 nanometers in width, with envelopes, but no clotting activity. This virus can be proliferated in primary cells, such as lamb and calf kidney or testicular cells, sheep embryonic kidney and lung cells, and chicken embryo fibroblasts. It also can multiply in Madin-Darby bovine kidney cells and baby hamster kidney cells (BHK-21), but the pathological changes are slower.

STABILITY

LSDV is resistant to high temperature and desiccation but viral particles die when exposed to direct Sunlight. It has been reported that virus becomes inactivated at 55 ◦C within 2 hours while takes 30 min at 65◦C temperature or direct contact with lipophilic detergents. Many disinfectants (iodine compounds, formalin, quaternary ammonium compounds, phenol, chloroform, sodium hypochlorite and ether are highly effective against LSDV (Oie, 2013; Mulatu and Feyisa, 2018).

PATHOGENESIS

There have been few studies conducted on the pathogenesis of LSD in cattle (El-Kenawy and El-Tholoth 2010). In the generalized form there is viremia and fever, followed by localization in the skin and development of inflammatory nodules. Following Subcutaneous or intradermal inoculation of cattle with LSDV, localized swelling at the site of inoculation developed 4 to 7 DPI which is varying in size from 1 to 3 cm and covering up to approximately 25% of the skin surface. Enlargement of the regional lymph nodes and generalized eruption of skin nodules usually follows 7 to 19 DPI. Viremia and Low levels of viral shedding in oral and nasal secretions was detectable between 6 and 15, and 12 and 18 DPI, respectively following febrile reaction. LSDV is also demonstrated in saliva, semen and skin nodules for at least 11, 42 and 39 days after the development of fever, respectively. Viral

replication in microphages, fibroblasts, pericytes, endothelial cells and probably other cells in blood vessel and lymph vessel walls causes vasculitis and lymphagitis in some vessels in affected areas, while thrombosis and infarction may result in severe cases (Coetzer and Tuppurainen et al.,2004). In natural infection, very young calves, lactating cows, and malnourished animals seem to develop more severe disease that may be due to an impaired humoral immunity. Antibodies was detectable 21 DPI using serum neutralization tests (Babiuk et al., 2008). Immunity after recovery from natural infection is life-long; calves of immune cows acquire maternal antibody and are resistant to clinical disease for about six months (Al-Salihi, 2014; Tuppuraine et al., 2005). Eventually, affected animals clear the infection and there is no known carrier state for LSDV (Tuppuraine et al., 2017).

CLINICAL SIGNS

The distinguishing character of the disease is the appearance of skin nodules in about 50% of susceptible animals, generally, of size less than 1cm to 8cm which are round in shape and emerge above the skin surface along with fever up to 40 to 41oC (50% of animals) and nasal discharge (in about 81.3% cases), swollen lymph nodes in 100% of cases, abortion (0.4%) in pregnant animals, decreased in milk production (on average 72.5% in buffalo and on an average 54.16% in cattle), lameness (6% of cases) (Awadalla and Hassan, 2011; Gibbs, 2021; KC et al., 2020; Mathan, 2011; OIE, 2017; Spickler, 2008). In the histological examination of infected animals, the epidermis has degenerative and necrotic parts of the keratocyte, intracytoplasmic inclusion bodies, and vesicles, the dermis has edema, hemorrhage, and the influx of lymphocyte, disrupted wall of a muscular blood vessel (Sanz-Bernardo et al., 2020).

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. Epithelial "cells clavelauses" cells, which are also reported in sheep pox, are invading the lesion. The agar gel precipitation test is not specific for LSD since other capripoxviruses and parapoxviruses share the LSDV antigen. In novel habitats, virus isolation can be employed for confirmation diagnosis. For viral isolation, pre-pubertal lambs and bovine testes are the most sensitive primary and secondary cultures. The most effective and quick test for disease diagnosis is molecular diagnostics using PCR. For quick diagnosis, conventional and real-time PCR have been developed (Bowden et al., 2008; Heine et al., 1999; Mangana-Vougiouka et al., 1999; Orlova et al., 2006; Tuppurainen et al., 2005; Zheng et al., 2007)10. It has been developed to distinguish LSDV from other Capripoxviruses using real-time PCR (Lamien et al., 2011). Antibody ELISAs have been developed with limited success. Indirect fluorescent antibody test is labor intensive and requires longer time duration thereby making it less cost effective than ELISA (Babiuk et al., 2008). Government of Pakistan's Institutes are fully equipped to test and report any outbreak in Pakistan. Some of these Labs might include 'National Agriculture Research Council' (NARC)

Islamabad, Veterinary Research Institute (VRI), Lahore. VRI is a Punjab government institute that is dealing with animal infectious diseases and developing vaccines for local infectious agents like, New castle disease vaccine, Hemorrhagic Septicemia disease vaccine, Foot and mouth disease vaccine and Enterotoxemia vaccine. Cholistan University of Veterinary and Animal Sciences, (CUVAS) Bahawalpur Pakistan, is fully equipped for dealing animal's infectious agents (Khan et al., 2021).

VACCINATION

A homologous live-attenuated LSD vaccine called Lumpi-ProVacInd was created by the ICARNational Research Center on Equines (ICAR-NRCE), Hisar (Haryana), and the ICAR-Indian Veterinary Research Institute (IVRI), Izzatnagar, Uttar Pradesh. There is a live attenuated vaccination for LSD. Businesses created vaccinations based on several LSD virus strains. It is either based on the SIS Neethling type or the Neethling strain used in products like the Lumpy Skin Disease Vaccine for Cattle (Onderstepoort Biological Products; OBP, South Africa) and Bovivax (MCI Sante Animale, Morocco) (Lumpyvax, MSD Animal Health-Intervet, South Africa). Since the virus that causes sheep pox and goat pox is closely related to LSD, the vaccination for those diseases can be used to treat LSD. According to OIE, many viral strains are employed as vaccine strains homologous virus causing lumpy skin disease. Three years of protection are provided by the South African Neethling strain, which is passed through 60 times in lamb kidney cells and 20 times on the chorioallantoic membrane of embryonated chicken eggs. Kenyan sheep pox virus passed 18 times in lamb testis (LT) cells or foetal calf muscle cells, Yugoslavian RM 65 sheep pox strain, and Romanian sheep pox strain are among the sheep pox strains utilized as vaccinations against LSD. Local responses are brought on by the strains of the heterologous vaccination. As these vaccinations may act as a source of infection for a vulnerable population of sheep and goats, they are not recommended in locations where sheeppox and goat pox are prevalent. Live attenuated Gorgan goatpox strain offers effective side-effect-free protection for cattle. 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. It is advisable to immunize fresh animals before bringing them to the impacted farm. At the age of 3 to 4 months, calves that have been nursed by moms who have received vaccinations or are infected naturally should be inoculated. Each year, pregnant cows and breeding bulls might receive vaccinations (Brenner et al., 2009; Capstick and Coackley, 1962; Capstick and Coackley, 1961; Tuppurainen et al., 2015)

ECONOMIC LOSSES

Lumpy skin disease has led to serious economic losses in affected countries. The disease causes aconsiderable reduction in milk yield (from 10% to 85%) due to high fever and secondary mastitis. Other consequences of the disease include damaged hides, decline of the growth rate in beef cattle, temporary or permanent infertility, abortion, treatment and vaccination costs and death of infected animals (Alemayehu et al., 2013; Babiuk, Bowden, Boyle, et al., 2008; Sajid et al., 2012; Sevik & Dogan, 2017). The total cost of the LSD outbreaks in 393 surveyed herds was 822 940.7 GBP in Turkey (Sevik & Dogan, 2017). In Ethiopia, the estimated financial loss was 6.43 USD and 58 USD per head for local zebu and Holstein Friesian, respectively (Gari et al., 2010). Total production losses resulting from the disease have been estimated at 45%–65% in industrial cattle farming (Tuppurainen & Oura, 2012). The causative agent, capripoxvirus, can induce sheeppox and goatpox as well, and these diseases have economic significance, given that they

present a major hindrance to international trade and may be abused as an economic bioterrorism agent. LSD is very dangerous and emerging disease, spreading very quickly in various countries like Pakistan. LSD has led to serious economic losses in many developed and developing countries. During high fever and mastitis disease caused abortion, 10-85% milk reduction, decline growth Lumpy Skin Disease: An insights in Pakistan development, damaged hides, decline infertility, death of infected hosts, emaciation, high cost of vaccination, reduced quality and quantity of skin and hides, which are not bearable to the small and large herdsmen (Alemayehu et al., 2013; Sajid et al., 2012) in affected countries (Turkey, Ethiopia, Pakistan etc.).

IMPACT

Emergence of LSD in the country like Pakistan having an agriculture- based economy and 2nd largest livestock population in world would have devastating consequences. Livestock population of Pakistan comprises a mighty population of 49.6 M cattle and 41.2 M buffaloes with having a 3.1 M and 1.2 M annual increment in numbers respectively. Livestock is the largest sub-sector of the country's agricultural production contributing Rs.1466 billion as value addition which is 2.5% more than aforementioned years. Livestock sector in Pakistan is contributing 60.6% in value addition in agriculture sector and have an 11.7% share in GDP with 3.1% share in total exports of the country as cradle of foreign exchange. About 8 M families are directly committed with livestock and earning 35–40% of their livelihood from this sector. The economic implications after the emergence of this devastating disease (LSD) on a country with already fragile and dwindling economy can be of grave nature and long lasting due to impositions of anticipated embargoes on livestock trade and due to severe downfall in the rural economy of eight million families (Khan et al., 2021).

PREVENTION AND CONTROL STRATEGIES

To this date, LSD does not have a viable treatment plan consequently; disease therapy is usually based only on the use of antibiotics and anti-inflammatory drugs. The only feasible solution to effectively control of this disease is to develop a preventive plan. To minimize the possible transboundary spread of disease, animals from endemic regions should be restricted especially across the borders. If a case is suspected with such lesions inside the country, quarantine policy should be adopted for a thorough evaluation (Molla et al., 2017). It cannot be said that outbreak of disease or disease emergence can be controlled by only control of vector. But it's a part in controlling the disease. By controlling the vector, we can reduce spread of disease at some level. It is true that no measurable values for spread of virus through vectors is established that vector involvement in the disease spreading is a major threat in disease spreading. Disease vectors can move long distances with the winds storms and may transmit the disease. So, Vector control strategies such as vector traps insecticide usage should be adopted in areas with high vector population to control the disease spread (Davies ,1991).

CONFLICT OF INTEREST

The authors declare no competing interests.

REFERENCES

1. Abutarbush, S. M. (2017). Lumpy skin disease (Knopvelsiekte, PseudoUrticaria, Neethling Virus Disease, Exanthema Nodularis Bovis). In J. Bayry (Ed.), Emerging and re-emerging infectious diseases of livestock (pp. 309–326). Springer International Publishing.
2. Afonso, P. P., Silva, P. M., Schnellrath, L. C., Jesus, D. M., Hu, J., Yang, Y., et al. (2012).

Biological characterization and next-generation genome sequencing of the unclassified Cotia virus SPAn232 (Poxviridae). J. Virol. 86, 5039–5054.
3. Alemayehu, G., Zewde, G., & Admassu, B. (2013). Risk assessments of lumpy skin diseases in Borena bull market chain and its implication for livelihoods and international trade. Tropical Animal Health and Production, 45, 1153–1159.
4. Ali BH and Obeid HM. (1977). Investigation of the first outbreak of Lumpy skin disease in the Sudan. Brit. Vet. J., 1333:184-189.
5. Al-Salihi, K. (2014). Lumpy skin disease: Review of literature. Mirror of research in veterinary sciences and animals, 3(3), 6-23.
6. Annandale, C.H., Holm, D.E., Ebersohn, K. & Venter, E.H. 2013. Seminal transmission of lumpy skin disease virus in heifers. Transbound. Emerg. Dis., 61(5): 443–448.
7. Awadalla, S.F., and Hassan, O.A., 2011. Incidence of lumpy skin disease among Egyptian cattle in Giza Governorate, Egypt. Vet. World, 4(4): 162–167.
8. Babiuk, S., Bowden, T. R., Boyle, D. B., Wallace, D. B., & Kitching, R. P. (2008). Capripoxviruses: an emerging worldwide threat to sheep, goats and cattle. Transboundary and emerging diseases, 55(7), 263-272.
9. Baldacchino, F., Muenworn, V., Desquesnes, M., Desoli, F., Charoenviriyaphap, T., & Duvallet, G. (2013). Transmission of pathogens by Stomoxys flies (Diptera, Muscidae): A review. Parasite,20, 26.
10. Bhanuprakash, V., Indrani, B. K., Hosamani, M., and Singh, R. K. (2006). The current status of sheep pox disease. Comp. Immunol. Microbiol. Infect. Dis. 29, 27–60.
11. Bowden TR, Babiuk SL, Parkyn GR, Copps JS, Boyle DB. Capripoxvirus tissue tropism and shedding: A
12. Brenner J, Bellaiche M, Gross E, Elad D, Oved Z, Haimovitz M, Wasserman A, Friedgut O, Stram Y, Bumbarov V, Yadin H. Appearance of skin lesions in cattle populations vaccinated against lumpy skin disease: statutory challenge. Vaccine. 2009 Mar 4;27(10):1500-3.
13. Capstick PB, c W. Lumpy Skin Disease—The Determination of the Immune State of Cattle by an Intradermal Test. Research in Veterinary Science. 1962 Jul 1;3(3):287-91.
14. Capstick, P. B., & Coackley, W. (1961). Protection of Cattle Against Lumpy Skin Disease: I.—Trials with a Vaccine Against Neethling Type Infection. Research in Veterinary Science, 2(4), 362-368.
15. Carn VM, Kitching RP, Hammond JM, Chand P. Use of a recombinant antigen in an indirect ELISA for detecting bovine antibody to capripoxvirus. Journal of virological methods. 1994 Oct 1;49(3):285-94.
16. Carn, V. M. (1993). Control of capripoxvirus infections. Vaccine, 11(13), 1275-1279.
17. Coetzer, J. A. W., & Tuppurainen, E. (2004). Lumpy skin disease. Infectious diseases of livestock, 2, 1268-1276.
18. Davies FG, Krauss H, Lund J, Taylor M. The laboratory diagnosis of lumpy skin disease. Research in Veterinary Science. 1971 Mar 1;12(2):123-8.
19. Davies, F. G. (1991). Lumpy skin disease of cattle: a growing problem in Africa and the Near East. World Animal Review, 68(3), 37-42.
20. Diesel AM. (1949). The Epizootiology of Lumpy Skin Disease in South Africa. In Proceedings of the 14th International Veterinary Congress, London, U.K., pp.492-500.
21. Elhaig, M.M., Selim, A. and Mahmoud, M., 2017. Lumpy skin disease in cattle: Frequency of occurrence in a dairy farm and a

preliminary assessment of its possible impact on Egyptian buffaloes. Onderstepoort Journal of Veterinary Research, 84(1), pp.1-6.

22. El-Kenawy, A. A., & El-Tholoth, M. S. (2010). Sequence analysis of attachment gene of lumpy skin disease and sheep poxviruses. Virologica Sinica, 25(6), 409-416.3. Coetzer JAW, Tuppurainen E (2004) Lumpy skin disease. In: Infectious diseases of livestock. Oxford University Press, Southern Africa 2: 1268-1276
23. El-Nahas, E. M., El-Habbaa, A. S., El-bagoury, G. F., & Radwan, M. E. I. (2011). Isolation and identification of lumpy skin disease virus from naturally infected buffaloes at Kaluobia. Egypt. Global Veterinaria, 7, 234–237.
24. Fagbo, S., Coetzer, J. A., and Venter, E. H. (2014). Seroprevalence of Rift Valley fever and lumpy skin disease in African buffalo (Syncerus caffer) in the Kruger National Park and Hluhluwe-iMfolozi park, South Africa. J. S. Afr. Vet. Assoc. 85, e1–e7.
25. Fayez Awadalla Salib and Ahmed Hassan Osman. (2011). Incidence of lumpy skin disease among Egyptian cattle in Giza Governorate, Egypt. Veterinary World.4 (4):162- 167.
26. Gari, G., Waret-Szkuta, A., Grosbois, V., Jacquiet, P., & Roger, F. (2010). Risk factors associated with observed clinical lumpy skin disease in Ethiopia. Epidemiology and Infection, 138, 1657–1666.
27. Gibbs, P., 2021. Lumpy skin disease in Cattle. MSD Veterinary Manual.
28. Givens, M. D. (2018). Review: Risks of disease transmission through semen in cattle. Animal, 12(S1), s165–s171
29. Gupta, T., Patial, V., Bali, D., Angaria, S., Sharma, M., & Chahota, R. (2020). A review: Lumpy skin disease and its emergence in India. Veterinary research communications, 44(3), 111-118.
30. Heine HG, Stevens MP, Foord AJ, Boyle DB. A capripoxvirus detection PCR and antibody ELISA based on the major antigen P32, the homolog of the vaccinia virus H3L gene. Journal of immunological methods. 1999 Jul 30;227(1-2):187-96.
31. Imran, M., Hashmi, A. H., Khalique, F., & Iqbal, M. Z. (2022). Lumpy Skin Disease Emerging Problem in Pakistan.
32. KC, G., Karki, S., Koirala, P., Upadhyaya, D., Regmi, B., and Pande, K., 2020. First report of Lumpy skin disease outbreak in cattle and buffaloes of Gandaki Province, Nepal. Authorea, pp. 1-8.
33. Khan, Y. R., Ali, A., Hussain, K., Ijaz, M., Rabbani, A. H., Khan, R. L., ... & Sajid, H. A. (2021). A review: surveillance of lumpy skin disease (LSD) a growing problem in Asia. Microbial Pathogenesis, 158, 105050.
34. Kumar S M. (2011). An Outbreak of Lumpy Skin Disease in a Holstein Dairy Herd in Oman: A Clinical Report. Asian Journal of Animal and Veterinary Advances, 6, 851–859.
35. Lamien CE, Lelenta M, Goger W, Silber R, Tuppurainen E, Matijevic M, Luckins AG, Diallo A. Real time PCR method for simultaneous detection, quantitation and differentiation of capripoxviruses. Journal of virological methods. 2011 Jan 1;171(1):134-40.
36. Lamien, C. E., Le Goff, C., Silber, R., Wallace, D. B., Gulyaz, V., Tuppurainen, E., Madani, H., Caufour, P., Adam, T., El Harrak, M., Luckins, A. G., Albina, E., & Diallo, A. (2011). Use of the Capripoxvirus homologue of Vaccinia virus 30 kDa RNA polymerase subunit (RPO30) gene as a novel diagnostic and genotyping target: Development of a classical PCR method to differentiate Goat

poxvirus from Sheep poxvirus. Veterinary Microbiology, 149, 30–39.

37. Lefkowitz, E. J., Dempsey, D. M., Hendrickson, R. C., Orton, R. J., Siddell, S. G., and Smith, D. B. (2018). Virus taxonomy: the database of the international committee on taxonomy of viruses (ICTV). Nucleic Acids Res. 46, D708–D717.
38. Lubinga, J. C., Clift, S. J., Tuppurainen, E. S. M., Stoltsz, W. H., Babiuk, S., Coetzer, J. A. W., & Venter, E. H. (2014). Demonstration of lumpy skin disease virus infection in Amblyomma hebraeum and Rhipicephalus appendiculatus ticks using immunohistochemistry. Ticks and Tickborne Diseases, 5, 113–120.
39. Mangana-Vougiouka O, Markoulatos P, Koptopoulos G, Nomikou K, Bakandritsos N, Papadopoulos O. Sheep poxvirus identification by PCR in cell cultures. Journal of Virological Methods. 1999 Jan 1;77(1):75-9.
40. Mathan, K.S., 2011. An outbreak of lumpy skin disease in a holstein dairy herd in Oman: A clinical report. Asian J. Anim. Vet. Adv., 6(8): 851–859.
41. Mcfadden, G. (2005). Poxvirus tropism. Nat. Rev. Microbiol. 3:201.
42. Molla, W., de Jong, M. C., Gari, G., & Frankena, K. (2017). Economic impact of lumpy skin disease and cost effectiveness of vaccination for the control of outbreaks in Ethiopia. Preventive veterinary medicine, 147, 100-107.
43. Morris JPA. (1931). Pseudo-urticaria. Northern Rhodesia Department of Animal Health, Annual Report 1930: 12.
44. Murphy, F. A., Fauquet, C. M., Bishop, D., Ghabrial, S. A., and Summers, M. D. (2008). Virus taxonomy: classification and nomenclature of viruses. Encyclopedia Virol. 140, 9–23.
45. OIE, 2017. Lumpy skin disease technical diseases card. OIE.
46. OIE, Office International des Epizooties. (2017). Manual of diagnostic tests and vaccines for terrestrial animals 2017. Chapter 2.4.13 Lumpy skin disease. Retrieved from http://www.oie.int/fileadmin/Home/ eng/Health
47. Orlova, E. S., Shcherbakov, A. V., Diev, V. I., & Zakharov, V. M. (2006). Differentiation of capripoxvirus species and strains by polymerase chain reaction. Molecular Biology, 40(1), 139-145.
48. Bowden, T. R., Babiuk, S. L., Parkyn, G. R., Copps, J. S., & Boyle, D. B. (2008). Capripoxvirus tissue tropism and shedding: A quantitative study in experimentally infected sheep and goats. Virology, 371(2), 380-393.
49. Tuppurainen, E. S. M., Venter, E. H., Shisler, J. L., Gari, G., Mekonnen, G. A., Juleff, N., ... & Babiuk, L. A. (2017). Capripoxvirus diseases: current status and opportunities for control. Transboundary and emerging diseases, 64(3), 729-745.
50. Sajid, A., Chaudhary, Z., Sadique, U., Maqbol, A., Anjum, A., Qureshi, M., Hassan, Z. U., Idress, M., & Shahid, M. (2012). Prevalence of goat poxdisease in Punjab province of Pakistan. Journal of Animal and Plant Sciences, 22, 28–32.
51. Sanz-Bernardo, B., Haga, I. R., Wijesiriwardana, N., Hawes, P. C., Simpson, J., Morrison, L. R., ... & Beard, P. M. (2020). Lumpy skin disease is characterized by severe multifocal dermatitis with necrotizing fibrinoid vasculitis following experimental infection. Veterinary pathology, 57(3), 388-396.
52. Sanz-Bernardo, B., Suckoo, R., Haga, I. R., Wijesiriwardana, N., Harvey, A., Basu, S., et al. (2022). The acquisition and retention of lumpy skin disease virus by blood-feeding

insects is influenced by the source of virus, the insect body part, and the time since feeding. J. Virol. 96:e0075122.

53. Sevik, M., & Dogan, M. (2017). Epidemiological and molecular studies on lumpy skin disease outbreaks in Turkey during 2014–2015. Transboundary and Emerging Diseases, 64(4), 1268–1279.
54. Sherrylin Wainwright, Ahmed El Idrissi, Raffaele Mattioli, Markos Tibbo, Felix Njeumi, Eran Raizman. (2013). Emergence of lumpy skin disease in the Eastern Mediterranean Basin countries. empres watch. Volume 29 NOVEMBER 2013. © FAO 2013.
55. Shimshony A, Economides P. (2006). Disease prevention and preparedness for animal health emergencies in the Middle East. Revue Scientifique et Technique - Office International des Épizooties, 25(1):253-269.
56. Spickler, A.R., 2008. Lumpy skin disease.
57. Sprygin, A., Artyuchova, E., Babin, Y., Prutnikov, P., Kostrova, E., Byadovskaya, O., et al. (2018a). Epidemiological characterization of lumpy skin disease outbreaks in Russia in 2016. Transbound. Emerg. Dis. 65, 1514–1521.
58. Tageldin Mohamed Hassan & Wallace David Brian & Gerdes Gertruida Hermanna & Putterill John Fraser & Greyling Roelf Rudolph & Phosiwa Maanda Noaxe & Al Busaidy Rashied Mohammed & Al Ismaaily Sultan Issa. (2014). Lumpy skin disease of cattle: an emerging problem in the Sultanate of Oman. Trop Anim Health Prod (2014) 46:241–246.
59. Thomas A D and Mare C V E (1945). Knopvelsiekte. J. S. Afr. Vet. Med. Assoc., 16: 36-43.
60. Tulman, E. R., Afonso, C. L., Lu, Z., Zsak, L., Kutish, G. F., and Rock, D. L. (2001). Genome of lumpy skin disease virus. J. Virol. 75, 7122–7130.
61. Tuppuraine ES, Alexandrov T, Beltran-Alcrudo D (2017) Lumpy skin disease field manual - A manual for veterinarians. FAO Animal Production and Health Manual 20: 1-60.
62. Tuppuraine ES, Coetzer JA, Venter EH (2005) He detection of lumpy skin disease virus in samples of experimentally infected cattle using di؛erent diagnostic techniques. Onderstepoort J Vet Res 72: 153-164.
63. Tuppurainen ES, Venter EH, Coetzer JA, Bell-Sakyi L. Lumpy skin disease: attempted propagation in tick cell lines and presence of viral DNA in field ticks collected from naturally-infected cattle. Ticks and Tick-borne Diseases. 2015 Mar 1;6(2):134-40.
64. Tuppurainen ES, Venter EH, Coetzer JA. The detection of lumpy skin disease virus in samples of experimentally infected cattle using different diagnostic techniques. Onderstepoort Journal of Veterinary Research. 2005 Jun 1;72(2):153-64.
65. Tuppurainen, E. S. M., & Oura, C. A. L. (2012). Review: Lumpy skin disease: An emerging threat to Europe, the Middle East and Asia. Transboundary and Emerging Diseases, 59, 40–48. https://doi.org/10.1111/j.1865-1682.2011.01242.x
66. Tuppurainen, E. S., Venter, E. H., & Coetzer, J. A. W. (2005). The detection of lumpy skin disease virus in samples of experimentally infected cattle using different diagnostic techniques. Onderstepoort Journal of Veterinary Research, 72(2), 153-164.
67. Von Backstrom U (1945). Ngamiland cattle disease. Preliminary report on a new disease, the aetiological agent probably being of an infectious nature. J. S. Afr. Vet. Med. Assoc., 16: 29-35.

68. Zare, P. (2010). Concise Review of Veterinary Microbiology (P.J. Quinn, B.K. Markey).
69. Zheng M, Liu Q, Jin NY, Guo JG, Huang X, Li HM, Zhu W, Xiong Y., A duplex PCR assay for simultaneous detection and differentiation of Capripoxvirus and Orf virus. Mol Cell Probes 2007. 21:276

HOW TO CITE: Hafsa Arshad , Muhammad Nadeem, Rizwan Shaukat, Lumpi Skin Disease Virus Is Emerging Challenging For Livestock In Pakistan, Int. J. of Pharm. Sci., 2024, Vol 2, Issue 6, 670-680. https://doi.org/10.5281/zenodo.11612906