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Review Article

Transboundary Spread of Lumpy Skin Disease with Emphasis on Diagnosis and Control

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Abstract

Lumpy skin disease is a disease which had its origin from Africa and spread almost to three by fourth of the globe. Though it may not be zoonotic and do not cause high mortality rate in animals, it is a disease of great importance as it cause high economic loss. The disease is caused by a strain of pox virus and cannot be effectively controlled by quarantine or other safety measures alone as the spread of the disease depends on the population of blood sucking parasites. The population of blood sucking parasites depends on the temperature and humidity and studies have shown that rising global warming and rise of temperature has increased the rate of progression of the disease. Vaccination alone as a control method lack efficiency hence other measures like vector control, isolation of affected animals, quarantine of newly purchased animals and restriction of movement of animals are implemented simultaneously along with vaccination strategies.

Keywords: Cattle, Control, Diagnosis, Lumpy Skin Disease, Virus

Introduction

Lumpy skin disease is a notifiable disease affecting mainly cattle and water buffalo causing great economic loss due to significant reduction in production and trade. Lumpy skin disease is caused by lumpy skin disease virus classified in genus Capri poxvirus of subfamily Chordopox viridae and family Pox viridae (Buller *et al.*, 2005). The virus has characters similar to vaccinia variola group and was used as vector virus in many cases. Exotic breeds of cattle are more susceptible to this while indigenous cattle show certain level of immunity (Vohra *et al.*, 2020). Disease occurs as acute, sub-acute or inapparent form and the characteristic features of the disease are fever and circumscribed skin nodules which usually undergo necrosis, lesions are also seen in mucosa of digestive and respiratory tract and skeletal muscles (Tuppurainen *et al.*, 2011). Though the disease originated from Africa, in a time period of a century it has spread to Middle East, Europe and Asia and covered almost three by fourth of the continents of the world.

Global Spatial and Temporal Distribution of LSD

The disease was first identified in Northern Rhodesia and the progression of the disease in different countries in different years are summarized in the table.

Table 1: Emergence of disease in different countries in different years

Year	Countries first reported	Continent	Reference
1929	Zambia (Northern Rhodesia)	Africa	(Morris, 1931)
1943-45	Botswana, Zimbabwe, Republic of South Africa	Africa	(Thomas and Mare, 1945; Von Backstrom, 1945; Diesel, 1949).
1957	Kenya	Africa	(MacOwan,1959)
1972	Sudan	Africa	(Ali and Obeid, 1977)
1974	West Africa		(Davies *et al.*, 1981)
1983	Somalia, Ethiopia	Africa	(Davies, 1991 a and b) Mebratu *et al*., 1984
1984	Oman (Middle East)		Tupprurainen and Oura, *2012*
1986	Kuwait		House *et al.*, 1990
1988	Egypt		El-Kholy *et al*., 2008
1989	Israel		Yeruham *et al.*, 1994
1993	Lebanon	Asia	Tupprurainen and Oura,2012
1995	Yemen	Asia	Tupprurainen *and* Oura,2012
2000	UAE	Asia	Tupprurainen and Oura, 2012
2003	Baharain	Asia	Tupprurainen and Oura, 2012
2012- 2013	Syria, Lebanon, Israel	Asia	Ben-Gera *et al.*, 2015
2013	Turkey	**Asia and Europe**	Şevik and Dogan, 2017
2014	Iran, Iraq	**Asia**	Al – Salihi, 2014
2015	Greece	**Europe**	
2016	Bulgaria, Serbia, Kosovo, Albania, Montenegro	**Europe**	Casal *et al.* ,2018
2018	Russia, Georgia	**Asia**	EFSA., 2019
2019	China, Bangladesh	**Asia**	Lu *et al*., 2020; Rahman ,2020
2019	India (Odisha)	**Asia**	Sudhakar *et al*., 2020

Agro climatic variations, abundance of vector population, sharing of watering points and grazing areas, increased trade of animals, under reporting etc increased risk of transmission.

Lumpy Skin Disease in India

The first outbreak of the disease occurred in India during the month of August 2019 in Odisha state. In this outbreak,

2 districts were affected and almost 182 cattle were infected. The first two outbreaks were reported in Mayurbhanj district and the third from Bhadrak district of Odisha. There was an apparent morbidity rate of 7.1percent and no mortality (Sudhakar *et al.,* 2020). Samples were tested for LSD by PCR and confirmed by sequencing. 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). Subsequently the disease was reported from other states like Karnataka, West Bengal, Chhattisgarh, Jharkhand, Assam, Maharashtra, Madhya Pradesh, Kerala, Tamil Nadu, Telangana and Andhra Pradesh. In January 2020, outbreak of lumpy skin disease was noted in Palakkad, Thrissur and Malappuram districts of Kerala, nevertheless the disease was controlled by proper preventive measures. Hyderabad, Telangana had similar LSD outbreaks recently in February and March 2020 in which all the infected animals succumbed to death (Vohra and Wattamwar, 2020).

Transmission

It mainly occurs due to mechanical arthropod vectors which are blood feeders (Weiss, 1968; Chihota *et al.,* 2001). The outbreaks of LSD are usually associated with wet and warm weather conditions which are breeding time of vector arthropod and it was not possible to control the disease by quarantine measures (Thomas and Mare, 1945; Weiss, 1968). It was proved that female *Aedes aegypti* mosquitoes can transmit the disease 2-6 days after feeding from infected to susceptible cattle in laboratory conditions. Similarly, both *Culex* and *Anopheles* mosquitoes along with *Culicoides* are mechanical transmitters (Chihota *et al.,* 2001, Sprygin *et al.,* 2019). In ticks, both transtadial as well as transovarian transmission of disease was reported (Tuppurainen *et al*., 2011). There was no association between cattle movement and disease prevalence (Gari *et al.,* 2010).

The disease can be transmitted through seminal fluid, milk, direct skin to skin contact, but it is more efficiently transmitted with the help of insect vectors. In experimental studies it was concluded that direct or indirect contact between infected animals and susceptible animals is inefficient for transmission of disease. (Weiss, 1968; Carn and Kitching, 1995). Transmission through seminal fluid is not experimentally proven in laboratory conditions but LSD virus is isolated in semen of experimentally infected bulls for 22 days post infection. However, by both virus isolation and PCR virus persistence was detected in epididymis and testes (Irons *et al.,* 2005). Intrauterine route of transmission of virus has also been proven where the new born calf delivered from a LSD infected cow has shown multiple skin lesions on birth and died 36 hours later. Serological study of the particular calf showed pre-colostral LSD antibodies, and the virus was identified by molecular methods like PCR (Rouby and Aboulsoud , 2016)

Clinical Manifestation and Economic Impact

The characteristic clinical signs are described by many authors. (Thomas and Mare, 1945 Weiss, 1968; Tuppurainen 2011). The incubation period in natural outbreak is estimated to be 1-4 weeks. Affected animals exhibited lacrimation, pyrexia (40-41^{o}C) which lasted for 3-4 days and enlargement of subscapular as well as pre-crural lymph nodes (Tuppurainen and Oura, 2012). The skin nodules started appearing within 48 hours of febrile condition. There were ulcerative lesions in conjunctival, oral and nasal mucous membranes. The lesions in mouth lead to excessive salivation and in nasal cavity may lead to labored breathing. Lesions were also detected in pharynx, larynx, trachea, lungs and throughout alimentary tract. Sometimes animals exhibited oedematous swelling of brisket and one or more limbs and lameness (Body *et al.,* 2011). The morbidity rate was high with negligible mortality and greatly depends on the immunity of the host and vector concentration in the area. The disease might be transmitted through production and marketing chains, thus becoming a transboundary disease (Rossiter and Hammadi, 2009). It is economically important in cattle industry due to symptoms like milk drop, abortion, permanent or temporary sterility which cause major depression in profits of the industry (Babiuk *et al.,* 2008). It highly affects cows at peak lactation more as it causes sharp drop in milk yield due to fever caused by the virus as well as secondary bacterial mastitis. The main cause of economic loss is due to lowering of milk yield and still birth of calves as it decreases reproduction (Ayelet *et al.,* 2013). There is also high chance the recovered animals might have pneumonia (Body *et al*., 2008) due to secondary bacterial infection. Emaciation and temporary or permanent infertility is noticed in cows and bulls and due to a longer convalescence period of several months there is marked decrease in production (Weiss, 1968). The scar caused by nodular lesions lead to low value of the hide (Green, 1959). Even restrictions in animal transportation within the country lead to nation wise financial loss (Tuppurainen and Oura 2012). Around 45-65 percent financial loss were noticed due to direct or indirect production loss in intensive cattle farms but the small-scale producers are the ones who are highly affected (Tupparainen and Oura ,2012).

Diagnosis

The first and foremost method is by characteristic clinical signs, but has to differentiate from diseases like herpes virus infection. Usually, clinical diagnosis is confirmed by PCR or real- time PCR to be more specific (Tupperainen *et al*., 2005). Other gold standard methods like virus isolation was used for diagnosis of the disease by inoculation of eight-day-old embryonated egg using chorioallantoic membrane (CAM) route primarily. This diagnostic method shows specific cytopathic effect common for all capripox viruses (House *et al.*, 1990). Virus isolation can also be done by cell culture using fetal lamb testes cells (LT), primary goat kidney (GK) cell and fetal bovine lung (FBL) cells (House *et al.*, 1990). Other test for capri pox virus like electron microscopy and serum or virus neutralization of antigen and antibody are still used for detection (Tupperainen and Oura, 2012). Histological studies using haematoxylin and eosin staining was done on skin biopsies and tissues obtained from necropsy which are 4-5µm in thickness examined under light microscopy show intraepithelial microvesicles and acantholysis. (House *et al.*, 1990)

Immunity against capripox virus is predominantly cell-mediated although humoral immunity also plays a role (Kitching *et al.*, 1987). The OIE recommended serological tests used for LSD diagnosis are essentially IFAT (indirect fluorescent antibody test), ELISA and VNT (Virus neutralization test) (OIE, 2004). The disadvantage to serological tests is that the tests cannot differentiate between infected and vaccinated animals or antibodies resulting from LSDV infection from those of other *Poxviruses (Awad et al., 2010; Gari et al., 2012).* There were attempts to develop Enzyme – linked immune sorbent assay [ELISA] but due to difficulty in detection of capripox antibody it was unsuccessful. This might be due to difficulty in producing inactivated whole virus in sufficient volume and due to instability of recombinant antigen (Bowden *et al.*, 2009). In most cases virus neutralization test (VNT) is considered as reference test which has a strong specificity but less sensitivity for capripox virus (OIE, 2004; Bhanuprakash *et al.*, 2006). Gari *et al*. (2008) evaluated IFAT for diagnosis of LSD using Bayesian method and found that it has high accuracy and can be used for serosurveillance analysis of LSD in target population. As the most common and confirmatory diagnostic method for the disease PCR can be performed using scab tissue and vesicular fluid (Sudhakar *et al.*, 2020). RT-PCR assay was found to be simple, sensitive, rapid, and reliable for the detection of LSDV in blood and skin nodule biopsies of suspected cattle. More numbers of LSDV-positive samples were detected by RT-PCR, followed by conventional PCR and then FAT. The indirect ELISA detected more antibody-positive samples than the IFAT from cattle serum samples (Zeedan *et al.*, 2019). Alexander *et al.* (2019) developed a real-time PCR assay based on the unique site in LSD044 for the universal detection of DNA from field, vaccine, and recombinant strains of LSDV.

Control

Lumpy skin disease does not have any particular treatment or drug of choice and as its self - limiting as most other diseases of pox viridae family, the best way of control of the disease and its succeeding effects is by vaccination with live attenuated vaccines (Babiuk, 2018). Live attenuated vaccine of goat pox, sheep pox, or LSD virus can be used as preventive measure as there is cross protection due to antigen homology, yet it does not provide complete protection (Kitching, 1983). Since there is antigen homology between the vaccine, the strain RM65 of live attenuated seep pox vaccine was used for vaccination in the outbreak in 1989 and in 2006-2007 (Yeruham *et al.*, 1994). Later the inefficiency of the vaccine was reported and in 2013 Israel decided to vaccinate all the cattle in Israel with RM65 live attenuated sheep pox vaccine at 10 times the dose rate along with attenuated strain of Neethling lumpy skin disease vaccine and observed that the Neethling vaccine is significantly more effective than x10RM65 in preventing LSD morbidity, though it might cause a low incidence of Neethling associated disease. (Ben-Gera *et al.*, 2015). Various strains of goat pox, sheep pox and LSDV are developed as in LSDV neethling strain, Kenyan sheep and goat pox virus (KSGPV) O-240 and O-180 strains, Yugoslavian RM65 sheep pox (SPP), Romanian SPP and Gorgan goat pox (GTP) strains (Kitching *et al.*, 1987, Lefevre *et al.*, 2010) Due to its lack of complete safety, LSD vaccine are not advised in disease free countries. A study proved that clinical cases of LSD was five times greater in unvaccinated compared to vaccinated herd (Brenner et al., 2009). It is very difficult to differentiate vaccinated animals from infected with the test used presently hence the control is very limited (Tupperainen *et al.*, 2012). Quarantine methods do not make much difference as transmission due to contact is very low and is mainly due to mechanical vectors (Weiss 1968).

Even then vaccination of all susceptible animals is considered as the main method of control, other methods of control such as stamping out, animal movement restriction and vector control are considered. Vaccination of susceptible animals and stamping out of animals in non -vaccinated infected herds is the best fool proof method for

control available (Milovanović *et al.*, 2019). As of today, there is no universal vaccine available. The main reason for this is that though capripox vaccine can be used in a particular host it is not essentially necessary that it shall induce immunity in a different host and this may be due to vaccine being not fully attenuated, overly attenuated and /or not immunogenic (Babiuk *et al*., 2018). The Government of India recommends ring vaccination in village up to 5 km around the affected village. Cattle and buffaloes should be vaccinated with goat pox vaccine at age of 4 months and above through subcutaneous route with $10^3 TCID_{50}$ of GTPV vaccine (Uttarkashi strain). Affected animals should not be vaccinated. (Advisory on LSD, DADF, Govt of India).

Ectoparasiticides can be applied topically or sprayed twice a week on susceptible neighboring farms from the infected herd. Restricted animal movement for 30 days should be advised to the infected farms from the identification of last infected animal (Zeynalova *et al.*, 2016). Biosecurity measures advised y Govt of India includes isolation of affected animals, clinical surveillance against LSD in affected districts, disinfection of premises at regular intervals, application f ectoparasiticide on animals, reporting of disease and restriction of movement of animals and people to and from the affected area.

Conclusion

Lumpy skin disease may be considered not highly dangerous due to low mortality but it is economically highly degrading disease and whose spread should be arrested. The disease started in Africa in 1929 and within a century it has infected almost the entire world. From the progression of the disease, we know for fact that the disease is transboundary. Due to the climate having a direct impact on occurrence of the disease the environmental factors such as global warming yet again affects the disease spread negatively due to increased population of mechanical vectors in the hot and humid climate. In addition to that vaccination may not give complete protection, even the vaccinated animal may become carriers for disease which may spread by blood sucking parasite. The observations underline the need for improved vaccines, fast serological assays which are sensitive and specific to the disease are to be developed and should be introduced in non- endemic countries. Fast recognition and control of disease is essential for arresting the spread of the disease and its eradication.

Conflict of Interests

There is no conflict of interest.

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