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A Thorough Examination of Lumpy Skin Disorders and their Diagnostic Methods

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Abstract - According to the reviewed literature, LSD has been spreading like wildfire among herds of cattle and Buffalo in several parts of the globe. According to the OIE data, LSD outbreaks were observed in India in late 2019. Recent reports of outbreaks have come from the Beed district and the Marathwada region of Maharashtra (Karyarambh newspaper, 2020). That's why it seemed like a good idea to write a review article on the latest developments in treating this illness. This would be useful not only to Veterinarians in the field for addressing this issue, but also to lab scientists for doing more study on methods of prevention and management.
Keywords- Endemically, Laboratory, Lumpy skin disease.

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I. INTRODUCTION

An emerging disease is defined by the OIE as "an infection that has emerged as a result of the evolution or change of an existing pathogenic agent, the spread of an established infection to a new geographic area or population, or the first diagnosis of a previously unrecognized disease that has a significant impact on animal or public health." When a previously recognized or endemic illness changes its environmental factors, host range, or incidence rates, it is said to be re-emerging.

Recent outbreaks of both new and re-emerging animal illnesses have had devastating effects on both animal and human populations. Lumpy skin disease (LSD) is an extremely contagious viral illness that causes lymphadenopathy and nodules to form anywhere on the body. Cattle and buffalo seem to be the primary hosts for the illness. This illness does not spread to animals. Many different regions of India have reported incidences of this newly developing ailment. This illness has been classified as a "List A" by the OIE (Office International des Epizooties). Recent (sporadic) occurrences of this illness have been recorded from a few locations in India. This evaluation was written for use by practicing veterinarians in light of the seriousness of this new development.

II. WORLD-WIDE EPIDEMIOLOGY

Lumpy skin disease has been classified as a Transboundary Animal Disease (TAD) by the World Organization for Animal Health (OIE, 2016) due to its significant economic impact on production and local communities, as well as the restrictions it imposes on international commerce in affected countries. Lumpy skin disease was first reported in 1929 in Zambia (Gumbe, 2018) and is only seen in Africa. The epidemic subsequently advances northward across Sudan and the remainder of southern Africa. In Africa

and Israel, the disease responsible for the lumpy skin is entirely rampant. Cutaneous lumps were first documented in Ethiopia in 1983 (Gumbe, 2018).

Wainwright et al. (2013) and the OIE World Animal Health Information Database (WAHID) both report LSD outbreaks in Turkey and Iraq at the end of 2013. The European Food Safety Authority Journal (2015) indicates that an LSD outbreak was first detected in Iran in early 2014. High populations of biting flies during the rainy season correlate with an increased incidence of Lumpy Skin Disease (LSD), while the dry season sees a reduction in this frequency (Gumbe, 2018). In May 2014, Azerbaijan acknowledged occurrences of LSD (OIE, 2014). Subsequent reports of outbreaks were documented from Armenia (2015), Kazakhstan (2015), the southern Russian Federation (Dagestan, Chechnya, Krasnodar Kray, and Kalmykia), and Georgia (2016). The OIE's 2019 report on LSD shows that outbreaks occurred in India in late 2019.

III. EPIDEMIOLOGY IN INDIA

An LSD pandemic in the Chhota Nagpur plateau area of India extended to adjacent states including Orissa, Jharkhand, West Bengal, and Chhattisgarh (Pashudhan Praharee, 2019). On December 8, Dr. Chaturvedi informed the OIE of nine instances of LSD in the Orissan districts of Khairbani, Betnoti, and Mayurbhanj (Chaturvedi, 2019). On the 17th of this month, Dr. Chaturvedi documented 20 instances from Patalipura, Betnoti, Mayurbhanj, Orissa. Dr. Hiresh Rajan Bhowmik (2019) reported 66 instances to the OIE from Chittagong, Bangladesh. Following the discovery of 'lumpy skin disease' in cattle in certain regions of Orissa (The Hindu Business Line, New Delhi, 2019), China has issued a warning letter prohibiting imports of cattle and bovine products from India. Recently, the illness has been identified in cattle within the districts of Palakkad, Thrissur, and Malappuram (The Hindu, January 2020). In 2020, the Karyarambh newspaper reported the presence of animals afflicted with the skin disease termed "lumpy skin" in the Beed district of Maharashtra.

Predisposing Factors

- Extensive LSD outbreaks are often initiated by circumstances including an increasing population of naïve animals, a surplus of active blood-feeding vectors, and unregulated animal migrations.
- The introduction of one or more new animals into an established herd, or their placement in close proximity to it, is the most prevalent event.
- During the acute phase of the illness, milk from infected animals may be separable; however, there is no evidence that the virus persists in the meat of these animals (Alaa et al., 2008).

Etiology

The lumpy skin disease virus (LSDV) belongs to the Poxviridae family and the Capripox genus. The initial strain of this virus was designated Neethling poxvirus. Antigenic resemblance among the LSD virus, Sheep pox virus, and Goat pox virus has been identified (Vegad and Katiyar, 2008).

Virus Susceptibility

Sodium hypochlorite (2-4%), phenol (2%), and iodine compounds have all been shown to be effective against the influenza virus. Within the pH range of 6.3-6.8, LSDV is exceptionally stable and lives well in severely cold and dry settings. It has been discovered that it is sensitive to changes in pH. The virus may be killed by heating it to 55 degrees Celsius for 2 hours, or 65 degrees Celsius for 30 minutes (OIE).

Host

The virus has very strict host requirements. The LSD virus is said to exclusively infect cattle and buffalo. Breeds with thinner skin, such the Holstein Friesian (HF) and Jersey, are higher at risk of LSDV infection (Gumbe, 2018). Furthermore, no wildlife reservoir of LSDV has been discovered despite large serological studies of wild ruminant species in Africa (World Organization for Animal Health, 2017). The LSDV virus is not zoonotic.

Morbidity and Mortality

Mortality rates are typically around 10% but morbidity rates might range from 2-45%. Death rates have been reported at about 2% on average, however this may fluctuate with epidemics (Radostits et al., 2006). The virus has a 2- to 4-week incubation period.

The host's susceptibility to this illness is influenced by factors such as their immune system, age, and breed (FAO Manual, 2017).

Transmission of the Disease

Insects are the primary vector of transmission. Haematophagous flies, such as Tabanus spp., Stomoxys spp., Culicoides spp., mosquitoes, and various kinds of ticks are likely to be implicated in LSD. Transmission occurs mechanically, making interrupted-feeding flies like Tabanus and Stomoxys the ideal vectors (Abdulqa et al., 2016). The primary vectors, however, remain a mystery (Hunter & Wallace, 2011). Different ecosystems and locales will have different primary vectors (FAO Manual, 2017). Water, saliva, milk, sperm, and even touch with lesions on sick animals may all serve as vectors for the spread of disease. Because the virus may live for a long time in the semen of infected bulls, it can spread to females via natural mating or artificial insemination (AI). Cows with the virus are also known to give birth to calves with skin problems. Infected milk or skin sores on the teats may spread the virus to nursing calves. Injecting pre-eruptive nodule blood or emulsified nodule tissue has been shown to spread the illness in experiments (Sastry, 2001).

Pathogenesis

After 4-7 days, regional edema and inflammation occur at the site of LSDV inoculation in cattle that was given subcutaneously or intradermally. Within 7–19 days following inoculation, a broad eruption of skin nodules and lymph node enlargement follow the development of localized edema. Vasculitis and lymphangitis result from viral replication in pericytes, endothelial cells, and other cells lining blood vessels and lymph vessels. Infarction, which may cause organ edema and necrosis in extreme situations. Most cattle have permanent immunity after recovering from a normal illness. Calves born to immunized cows develop a natural immunity to the illness about six months later (Coetzer, 2014).

Molecular Pathogenesis

LSD's molecular etiology is poorly understood.

Clinical Signs and Symptoms

Anorexia, despair, and fatigue accompany the high fever that may reach 42 degrees Celsius. The other symptoms (Fig. 1) include snoring, oculo-nasal discharge, and ptyalism. Lameness caused by leg nodules. The secondary bacteria that cause mastitis may enter broken skin nodules. When nodules form on the trachea, throat, or bronchi, it might be difficult to breathe. There is a possibility of blindness and corneal opacity. Animals in pregnancy may have miscarriages.

Gross lesions

Dermatological lesions manifest as circular, confined lesions with diameters between 0.5 cm and 5.0 cm and, in some cases, a ring of hemorrhages (Fig. 2). Lymphadenopathy is the medical term for swollen lymph nodes. Ulcers may develop everywhere, including the respiratory tract. Tissues in the windpipe, throat, and lungs often develop nodules. Nodules have a wide range of outcomes; they may go away fast or stay as a hard lump for a year or more (Sastry, 2001). The condition is made worse by a secondary bacterial infection.

Morbid Specimen Collection

Blood/serum of infected animal (for serological test); Pathological evidence (in the form of lesions); Damage to the skin or dermis—used to diagnose HP and to isolate viruses. Semen from an infected bull and milk from a cow or buffalo with udder lesions may be used to diagnose the disease, as can samples of the diseased animal's lungs and trachea.

Microscopic Lesions

The epidermis, the subcutaneous tissue, and even the surrounding muscles may be affected by the nodules. There are granulomatous lesions in the upper respiratory and digestive tracts, as well as on other mucous membranes and in certain organs (especially the lungs). Epidermis thickening (acanthosis), parakeratosis (in which the stratum corneum becomes thicker and contains pyknotic nuclei), and hyperkeratosis are all seen. Tageldin et al. (2014) describe eosinophilic intracytoplasmic inclusion bodies as being present in a variety of cell types, including keratinocytes, fibroblasts, macrophages, and more (Fig. 3).

Diagnosis

Typical clinical manifestations and lesions are used in field diagnosis. Keratinocytes, fibroblasts, macrophages, and so on all include eosinophilic intracytoplasmic inclusion bodies (Brenner et al., 2006) when examined under the microscope for inclusion bodies.

Laboratory Diagnosis

The enzyme-linked immunosorbent assay (ELISA) is used to detect and quantify antibodies in blood. The FAT test is used to identify the presence of a specific antigen. The most efficient and reliable approach for LSDV detection is polymerase chain reaction (PCR). The LSDV may be detected in skin nodules, blood, or nasal and saliva samples. Neutralization assays for viruses look for antibodies that can stop the virus from reproducing.

Pathological Diagnosis

Histopathology involves the regular collection and processing of tissues (such as lungs, trachea, and skin lesions) in 10% buffered formal saline. Eosinophilic intracytoplasmic inclusion bodies, necrotic epidermis, and squamous epithelial cell ballooning were also seen (Brenner et al., 2006). Primary diagnosis may be made using electron microscopy. Capripox virus, as seen via electron microscopy.

Figure 1: Note copious thick yellow occulo-nasal discharge and thick copious oral discharge (ptyalism) from LSD affected cattle.

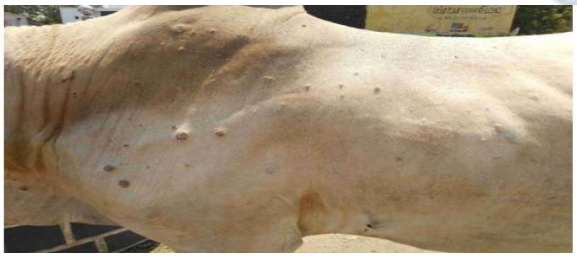

Figure 2: Note discriminate nodules of varied size and shape throughout the body of LSD affected cattle.

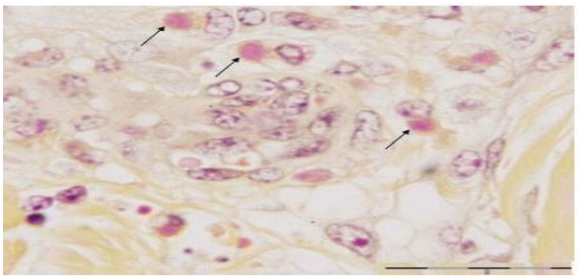

Figure 3: Mononuclear cells displaying intracytoplasmic inclusions (arrows).

Differential Diagnosis

The illness has to be distinguished from these other diseases. When compared to Lumpy Skin illness, the symptoms of Pseudo-Lumpy Skin Disease (BHV-2) (Allerton strain) are milder and the illness course is shorter. PCR can tell the difference between true lumpy skin illness and the more common pseudo lumpy skin disease. Pseudo cowpox (Para poxvirus): lesions occur mostly on teat and udder, and the illness may be distinguished from Lumpy skin disease by polymerase chain reaction (PCR).

Teat, udder, and snout lesions are common places for Vaccinia and Cowpox (Ortho poxviruses) to manifest themselves. Cowpox, in contrast to Lumpy skin disease, is a zoonotic illness that can be distinguished from Lumpy skin disease by polymerase chain reaction. Dermatophilosis is characterized by the development of superficial lesions (which are often wet and have a crusty, keratinized appearance).

Infection with Hypoderma bovis causes the skin of an animal's back to swell or disintegrate, and the larvae that live there to become visible. If the spinal cord is affected, lower body and leg paralysis might develop. LSDV detection by PCR allows for the exclusion of lumpy skin condition.

Dermal lesions in photosensitization are similar to those in lumpy skin disease, however the lesions in photosensitization are more superficial and may be distinguished using polymerase chain reaction (PCR). Urticaria, caused by insect or tick bites, causes scattered wheals to grow across the skin. Urticaria, in contrast to Lumpy skin disease, causes swelling of the face, limbs, and ventral side of the belly, and may be diagnosed by polymerase chain reaction (PCR). Subcutaneous TB causes the development of a single or numerous firm, painless nodules anywhere on the animal's body. LSDV detection by PCR allows for the

distinction between lumpy skin condition and cutaneous TB.

The lesions of onchocercosis, known as intradermal nodules, are most often seen along the dorsal midline. Demidocosis is caused by parasites that dwell in hair follicles and are linked to the skin's oil glands, resulting in the development of papules and nodules all over the animal's body. LSDV detection by PCR allows for the exclusion of lumpy skin condition.

Prophylaxis

Currently, there are no killed or inactivated vaccinations available for LSD. Cattle in Africa are vaccinated against LSDV using an attenuated Neethling strain vaccine (Coetzer, 2004). The vaccination developed to prevent disease in sheep and goats may also be administered to cattle (Ganguly, 2016; Capstick and Coackley, 1961). following vaccination, antibodies begin to show up 10 days later, with peak levels occurring 30 days following immunization.

IV. TREATMENT

Animals who have been afflicted may get symptomatic care. Antibiotic treatment for 7-10 days to prevent a subsequent infection. It may also be prudent to consider dosing the patient with anti-inflammatory and anti-histamine medications. Paracetamol may be used in the event of fever. It is suggested that an antibiotic ointment that also repels flies be applied to the damaged skin. Multivitamin injections or pills are recommended. Infected animals should be fed a diet of liquid food, soft feed and fodder, and succulent pasture.

V. CONTROL

Physical, biological, cultural, and chemical methods are all part of an integrated approach to vector control. Practices including adjusting grazing times to minimize direct sunlight and use biocontrol products to combat pests like flies and ticks are advocated. Herbal pesticides may be used as repellants, while chemical insecticide sprays can be used as a last resort. Immunization in high-risk zones. Infected or newly arriving animals are put in quarantine. Animal housing and other areas are disinfected with 2% phenol, 2-3% sodium hypochlorite, etc. During an epidemic, it is best not to trade or relocate animals that have been afflicted.

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