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The Pharma Innovation



ISSN (E): 2277- 7695 ISSN (P): 2349-8242 NAAS Rating: 5.03 TPI 2019: 8(7): 374-377 © 2019 TPI www.thepharmajournal.com Received: 13-05-2019 Accepted: 15-06-2019

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Contagious ecthyma (Orf): A disease of goats and sheep

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Abstract

Contagious ecthyma is an infectious dermatitis of sheep and goats that affects primarily the lips of young animals. The disease is usually more severe in goats than in sheep; it is marked by an increase in incidence and severity if not controlled among small ruminant herds. Primary orf lesions are the most severe with a clinical progression of erythematous macule, papule, vesicle, pustule and scab formation. Though the disease is self-limiting, secondary bacterial infections lead to complications that may lead to mortality. People are occasionally affected through direct contact. This reviews of present disease help in the effective and systemic management of the disease leading the reduction of the economic losses to a great extent.

Keywords: Contagious ecthyma, goat, sheep, Orf, parapox virus, scab

1. Introduction

Contagious Ecthyma also known as contagious pustular dermatitis, infectious labial dermatitis, sore mouth or scabby mouth and Orf (de Wet and Murie, 2011)^[5]. It is a highly contagious, zoonotic skin disease of small ruminants. The disease is caused by Orf virus, the type species of Parapox virus of subfamily Chordopoxvirinae and family *Poxviridae* (Haig and Mercer, 1998) ^[13]. The disease is characterized by scabby sores around lips, muzzle and mouth. Goats are more severely affected then sheep. The disease has a worldwide occurrence and can occur at any time of the year but is more frequently reported during spring and summer mainly among lambs and kids (Robinson and Balassu, 1981; Gokce et al., 2005)^[26, 8]. It is a non systematic eruptive skin disease having worldwide in distribution (de la Concha-Bermejillo, 1995; Mondal et al., 2006) ^[4, 19]. Morbidity and mortality has been reported to range from 20% to 100% and 1% to 93% respectively, and the rate of mortality depends on conditions like stress, immune system problems, etc. Financial losses are related to decline in production, devaluation of meat, leather and wool in national and international markets, as well as costs related to being zoonotic. Prolongation of the infection and an increase in severity are nearly associated with secondary bacterial infections (Gukce and Woldehivet, 1999; Ndikuwera et al., 1992)^[9, 22]. Some infected animals become carriers and shed the virus for a long period. The mortality occurs, especially in young sucking lambs, due to dehydration and starvation, as the pain and distortion of the lips and mouth preclude the lamb from sucking. The lambs affected by the mouth form or with strawberry foot rot show considerably reduced growth performance. The disease is also known as sore mouth, contagious pustular dermatitis or scabby mouth (Thomas et al., 2003) ^[30]. The disease is manifested by proliferative lesions on the mouth and muzzle that usually resolved in 1-2 months (McKeever et al., 1988)^[15]. Primary Orf lesions are the most severe with a clinical progression of erythematous macule, papule, vesicle, pustule and scab formation in 4-6 weeks and young animals are at high risk. Severe facial and oral lesions in lambs may interfere with suckling, lesions on the udder may result in the abandonment of offspring and foot lesions can cause transient lameness. Reinfection lesions progress through the same clinical stages but are generally smaller, not proliferative and resolve more rapidly usually within 2-3 weeks. Orf is not normally fatal but is a debilitating disease that can be fatal if lambs and kids are prevented from suckling or succumb to secondary bacterial or fungal infections (Haig and McInnes, 2002)^[12]. The morbidity of the disease may reach up to 100% and mortality due to secondary bacterial infections may reach to 15% (Gumbrell and McGregor, 1997) ^[10]. Some infected animals become carriers and shed the virus for a long period. The mortality occurs especially in young sucking lambs, due to dehydration and

starvation, as the pain and distortion of the lips and mouth preclude the lamb from sucking. The lambs affected by the mouth form or with strawberry foot rot show considerably reduced growth performance. Zoonoses occur most frequently during lambing, shearing, docking, drenching or slaughtering of affected animals. Most infections in humans are localized and heal spontaneously. However, large poorly healing lesions are usually seen in immunosuppressive individuals. As per World Organization for Animal Health, Orf is a notifiable disease and also a zoonotic disease transmitted from animals to humans.

2. Etiology

Contagious ecthyma is caused by infection of orf virus, a member of the genus Parapoxvirus in the subfamily Chordopoxvirinae and family Poxviridae. Other members of the same genus are bovine papular stomatitis (BPS) virus, Parapoxvirus of red deer in New Zealand (PVNZ), pseudo acowpox virus (PCPV) as well as three tentative species of the genus, auzdyk disease virus, chamois contagious ecthyma and seal pox virus (Buchen Osmond, 2003)^[1]. Virions are cocoon shaped with about 260nmin length and 160nm in width and covered with long thread like surface tubules resembling a ball of yarn. The viral genome, one of the smallest in Poxviridae family, is composed of 140 kbp linear ds DNA with closed hairpin loop ends and genes located on both strands with a bidirectional orientation. The terminal 3 kbp DNA at each end forms an inverted terminal repeat (ITR) (Robinson et al., 1982; McInnes et al., 2001)^[14]. Conserved genes are found in the central region whereas variable genes are at the terminal ends (Gassmann et al., 1985; Fraser et al., 1990; Mercer et al., 2002) ^[7, 6, 18]. In common with other poxviruses, Orf viruses replicate in the cytoplasm of host cells and encode its own machinery for DNA transcription and replication. The central region contains genes that are essential for DNA replication and the production of virus particles in the cytoplasm of infected cells, whereas terminal nonessential virulence genes involved in viral pathogenesis (Moss, 1996; Mercer and Haig, 1999)^[20, 17].

3. Pathogenesis

Skin is the main site of predilection and essential for establishment and development of lesions. The incubation period in natural cases of CPD is 2-3 weeks (Zamri-Saad *et al.*, 1992)^[33]. Initially virus replicates in the epidermal cell layers derived from the walls of wool follicle. At the time of grazing, the dried stemmy and spiny feed may abrade the tissues of lips, nostrils, mouth as well as fore stomach. Through such

abrasions virus penetrates the skin of mucosa and leads to formation of acanthosis, ballooning degeneration of spinose cells, hyperplasia of basal cells and edematous and granulomatous inflammation of dermal cells. The virus produces the characteristic lesions in a sequence of papules, vesicles, pustules, scabs and resolution. The pustules develop within a few days. The rupture of pustules results into ulcer formation followed by thick overlaying crust or scab, which is shed within 3-4 weeks leaving no scar. Though the pathogenesis of orf is simple, it becomes complex from secondary bacterial infection. In malignant form of disease, cauliflower like growth on oral mucosa and necrotic or deep ulcerative lesions in the buccal cavity, pharynx, larynx, oesophagus or throughout the alimentary tract were noticed by Thorp (1942)^[31]. After clinical recovery, immunity of affected animals last for eight months to one year. However, the immune response to virus infection is predominantly accompanied by humoral immunity, cell-mediated immune mechanism plays an important role in the process of recovery (McKeever et al., 1987)^[16].

4. Clinical History and Diagnosis

The period of incubation the disease varies from 4 to 8 days with an slight rise in temperature intially, development of papules and pustules often at oral commissures, skin of lips and nose followed by thick, tenacious scabs covering a raised area of ulceration, granulation and inflammation. Similar skin lesions of few milli meters to several centimeters are also seen in the lips of the kids particularly below two months of age and disseminated to the skin of face, ears, feet, flanks, and scrotum (Fig.1 and 2) (Reid, 2000; de la Concha-Bermejillo et al., 2003; Guo et al., 2003)^[25, 3, 11]. In the mouth of mountain-goats, dry, brown, proliferative lesions are prominent on the markedly edematous hyperemic lips, but severity diminishes toward the commissures. Lesions of the oral cavity especially on the gum become moist, reddish-brown and in certain sites, intensely hyperemic (Samuel et al., 1975) [28]. The affected kids and lambs unable to graze and also unable to suckling from doe (Chan et al., 2007)^[2]. The affected lesion and scabs are fragile and bleed easily and lead to anorexia. Some lesions may moist and dirty lead to foul smelling. In some cases, it may leads to mastitis due to secondary bacterial infection of other bacteria like Mannheimia haemolytica and Gram-negative Streptococci. The venereal form of Orf leads to appearance of papule and vesicles on the scrotum of rams and ulcers on the skin of the vulva of ewes (de la Concha-Bermejillo et al., 2003)^[3].



Fig.1 Fig.2 Fig 1 & 2: Show Orf lesions around mouth, nose and lips in goat

Diagnosis of Orf is based on the history and clinical signs of disease. Other laboratory techniques like Electron microscopy(EM), serological techniques like agar gel precipitation test(AGPT), agglutination test, complement fixation test (CFT), enzyme linked immune-sorbent assays (ELISAs), serum neutralization test (SNT), histopathology of affected tissues, nucleic acid based assays including polymerase chain reaction (PCR) and restricted fragment length polymorphism (RFLP) analysis.

5. Treatment and Discussion

During the outbreak, the isolation of affected animals and the quarantine of new animals in the herd are maintained on compulsory basis. Sick animals should be separated, fed and treated after feeding the herd. Consumption of milk from does that present lesions on the teats and udder should be avoided. Proper preventive measures for the cure of disease should be maintained. Proper disinfection of the houses and premises of the animal herd and incineration of all infected materials extracted from sick animals are required to reduce the risk of new infection. Vaccination with live virus vaccine should not be recommended on a farm having no previous history of Orf outbreak as the live virus may contaminate environment.

Although it is a self-limiting disease hence the symptomatic treatment can be given with antiseptic dressing is helpful. As to prevent the secondary bacterial infection a As secondary bacterial contamination in Orf virus infection is not uncommon, therefore topical and systemic antibiotics must be used in treatment schedule. Occasionally levamisole as an immunostimulant is indicated in Orf virus infection (Wilson *et al.*, 2002) ^[32]. Debilitated sheep need to be treated with 10% glucose saline intravenously (Rao and Bandyopadhyay, 2000) ^[24]. Lesions should be washed with 1:100–1:10,000 KMnO4 lotion and application of 1:10 boric acid (Rao *et al.*, 1994) ^[23], mild antiseptic (Singari *et al.*, 1990) ^[29] or antibiotic ointment (Nandi *et al.*, 1999) ^[21] topically with parenteral antibiotic injection is recommended to prevent secondary bacterial complications.

6. Conclusion

Contagious ecthyma, caused by parapox virus, is one of the most common skin diseases of sheep, goats and other domesticated as well as wild ruminants. It is responsible for producing skin lesions at the area of mouth, lips and nose for the incubation period of approximately, one week. At present, the CE and its allied impediments have the status of disease of worldwide occurrence which can arise in rural as well as urban areas due to less awareness, casual negligence and through religious or cultural practices involving animal handling and slaughtering. Since Orf is a zoonotic disease, so its handling and management should be done very vigilanty. Though the disease is self-limiting, secondary bacterial infections lead to complications that may lead to mortality. The most effective means of controlling losses is vaccination. Vaccination of susceptible animals with a potent and efficacious vaccine surrounding the infected flock(s) should be considered (ring vaccination). However, feasibility of eliminating infected and exposed flocks by slaughter, properly disposing of animals and contaminated material and cleaning and disinfecting contaminated premises and equipment should also be considered. Depopulation of infected and exposed flocks should be implemented if limited spread has occurred. If the disease has spread extensively, massive vaccination and control of animal movements from the area represent a viable

and ideal strategy to control and then eradicate Orf (Nandi *et al.*, 1999) ^[21]. Since the CE is a viral disease, there is no definitive treatment for infection in humans or animals. Antibiotics, anti-inflammatory drugs, antiviral drugs and surgical resection usually have only limited success.

7. Acknowledgements

Authors are thankful to the Dean, Post Graduate Institute of Veterinary Education and Research (RAJUVAS), Jaipur; Head/ In-charge of Department of Veterinary Clinical Complex and Department of Veterinary Gynaecology and Obstetrics, PGIVER, Jaipur for motivating to write the review. No funds were utilized from any grant.

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