

BOVINE HERPES MAMMILLITIS (*GULWADDEE*) - A LESS KNOWN DISEASE OF COWS AND BUFFALOES IN PAKISTAN

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ABSTRACT

Bovine herpes mammillitis is a disease of cows and buffaloes associated with bovine herpes mammillitis virus 2 and characterized clinically by successive appearance of plaques, vesicles ulcers and scabs on teats and udder. Vesicle formation, a characteristic feature of early stages of the disease in cow is either absent or occurs rarely in dairy buffaloes. The disease is worldwide in distribution but has not been reported from Pakistan. This is despite the fact that one clinical form of the disease (colloquially known as *Gulwaddee* in Punjabi) has been observed by most of the seasoned Pakistani veterinary practitioners. Most cows and buffaloes are affected by latent and sub-clinical form of the disease and only a few infected animals develop its clinical form. Stress of calving, udder edema and hormonal changes close to calving are important predisposing factors. The condition is usually sporadic although outbreaks have also been documented. It is more prevalent in cows and buffaloes in their first lactation. The causative virus multiplies at a temperature lower than the temperature of the rest of the body. Therapeutic interventions that can be used for the treatment of bovine herpes mammillitis include debridement

of the scab covered lesion, topical and parenteral administration of antibiotics, topical administration of lysine/propolis, topical and oral administration of acyclovir (an anti-herpes virus drug), oral administration of zinc sulphate and other immunity enhancing agents and parenteral administration of antihistamine and anti-inflammatory drugs. Therapeutic interventions proposed for future research include investigation of the oral use of zinc sulphate, local infiltration of acyclovir into the lesions and use of infrared lamp to increase the temperature of the lesion. No vaccine is currently available recommended control and prevention measures include biosecurity, prevention and proper treatment of udder edema, insect control and milking time hygiene.

Keywords: *Bubalus bubalis*, buffalo, *Gulwaddee*, herpesvirus 2, sporadic

INTRODUCTION

Bovine herpes mammillitis is a viral disease of cows and buffaloes caused by bovine herpesvirus 2 (BHV2) and characterized clinically in typical cases by appearance of ulcers and

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scabs on teats (Radostits *et al.*, 2007). Various designations used to describe herpes mammillitis in cow and buffalo include ulcerative thelitis (Abd-El-Hady, 2015; Lokanadhamu *et al.*, 2005), necrotic thelitis (Kathirvel and Dharmaceelan, 2016; Mouli, 1992), ulcerative mammillitis (Rao *et al.*, 2003), bovine ulcerative mammillitis (Syed *et al.*, 2009), bovine herpes mammillitis (Bitsch, 2011; Scott, 1989; Sharma *et al.*, 2005; Turner *et al.*, 1976; Weaver *et al.*, 1972; Yager and Scott, 1992), allergicmammitis (Sankaram and Kotayya, 1977), mammillitis (Sundaresh and Sundaresh, 1997) and *Gulwaddee* in Punjabi.

EPIDEMIOLOGY

Bovine herpes mammillitis (BHM) is caused by bovine herpesvirus 2 (BHV2), a virus of the genus *Simplexvirus*, subfamily *Alphaherpesvirinae* and the family *Herpesviridae*. BHV-2 also causes the rare pseudo-lumpy skin disease of cows characterized by appearance of edematous painful skin lesions (nodules) all over the body which later on become necrotic with exudation and are eventually covered by scabs (Bitsch, 2011). Although, BHM has been reported from many countries of the world (Bitsch, 2011), the condition has not as yet grabbed the desired attention of the veterinary practitioners, veterinary academicians and animal health researchers in Pakistan. Consequently, the cognizance of Pakistani animal health professionals is limited to the form of BHM that produces an indolent ulcer at the junction of teat and udder colloquially known as *Gulwaddee* and worst still they do not even know that this rare manifestation of herpetic mammillitis is associated with herpes mammillitis virus.

The condition is usually sporadic although

outbreaks have also been reported (Bitsch, 2011). It is more prevalent in temperate regions of the world. BHM virus is transmitted through milker's hand, milking equipment, biting insects, semen and air (Kemp *et al.*, 2008). Heifers become infected before calving. The stress of calving particularly with udder edema and hormonal changes lead to activation of BHM virus and development of signs (Gibbs, 1984; Kemp *et al.*, 2008; Martin, 1973). Only one teat or at the most two teats are affected in buffalo (Sankaram and Kotayya, 1977). The clinical condition is usually sporadic in occurrence (Sharma *et al.*, 1998) and mostly affects lactating animals particularly the first calvers. Most cases occur within the first two months of lactation (Sharma *et al.*, 1998). Hind teats are more frequently involved than the front teats (Mouli, 1992). Skin lesions of BHM display typical features of inflammation resulting from the activation of complement by the classical pathway as a consequence of production of immune complexes of virus with specific IgM and IgG (Bitsch, 2011). The condition is more prevalent in winter season than in other seasons of the year (Bitsch, 2011; Sharma *et al.*, 2005). Rabbit is a good lab animal for confirmation of BHM. Bacteria-free filtrate of whey obtained from the milk of the affected teat when injected intradermally in rabbits produces mild erythematous lesions 48 h after inoculation that persist for 48 h. Histopathologic examination of the skin of inoculated rabbits may show formation of syncytia (multinucleated cells resulting from fusion of single cells) and intranuclear inclusions in epidermal cells (Lokanadhamu *et al.*, 2005). Mice do not show any such reaction on inoculation (Rao *et al.*, 2003).

CLINICAL SIGNS

In most infected cows (Janett *et al.*, 2000) and buffaloes, the disease is latent and sub-clinical. Only a few infected animals develop clinical signs. The spectrum of the clinical disease varies considerably. Some affected animals may become hard milkers without appearance of physical lesions. Others may show mild teat lesions and a small subset of affected animals develops very severe teat lesions (Abd-El-Hady, 2015; Sandrucci *et al.*, 2014). According to (Sandrucci *et al.*, 2014; Shearer *et al.*, 2008), BHM in cows starts with painful raised edematous swellings of teats followed by appearance of vesicles (0.5 to 5 cm in diameter) which are irregular in shape. Within 24 h, these vesicles rupture leading to formation of ulcers that exude copious serum. Upon drying of this exudate, thick dark reddish-brown scabs appear on the teats. Healing usually occurs in 3 weeks, although ulcerated lesions may persist for months (Sandrucci *et al.*, 2014). Teat and udder lesions may fuse together and may extend to the perineum resulting in vulvovaginitis. Milking causes pain to the affected animal and sometimes the entire teat becomes necrotic. Calves suckling affected cows may develop lesions in mouth (George *et al.*, 2008).

According to (Sharma *et al.*, 1998), in dairy buffaloes, the lesions of BHM mostly occurred at the udder-teat junction of hind teats. The Punjabi term *Gulwaddee* (literally translated as ‘neck cutter’) for BHM derives its colloquial name from the occurrence of a deep non healing ulcerative lesion at the junction of teat with the udder i.e. neck of the teat. Raw ulcer or ulcer covered with thick blue-black scab was the most common presenting lesion. The initial phase of the disease was characterized by the formation of localized plaques (2 to 5 mm) within the thickness of teat wall. These

plaques were surrounded by inflammatory zones and ruptured within 48 h leaving very deep ulcers. Vesicle formation was not observed. As the healing progressed, a very thick blue-black scab developed and covered the healing lesion. Systemic signs of illness (e.g. rise in body temperature, pulse and respiration rates) were not present in the affected buffaloes. Milk of affected teats is usually negative for mastitis (Purohit *et al.*, 2014).

According to (Rao *et al.*, 2003), in buffaloes, the size of the affected teat increases 2 to 3 times its normal size with a glossy appearance. Severe inflammation of teat causes tenderness and loss of flexibility. Out of 101 cases examined, 28% cases developed ulcers on the affected teat whereas in 4% cases, there was local necrosis at the base of the teat. Partial sloughing of the teat was noticed in 12% of the cases. Appearance of the milk did not change as mastitis was absent. Subsequently, there was sloughing of the entire teat leaving only the tissue around the teat canal. When the calves suckled the unaffected teat(s), spontaneous flow of milk from the affected teat was observed through the denuded teat canal. Spontaneous recovery was observed in 9% of animals.

Workers from Indian Punjab (Sharma *et al.*, 2005) have described an unusual form of the disease in a few dairy buffaloes. This form of the disease started with sudden appearance of multiple flattened nodules (1 to 4 cm in diameter) all over the body. In the majority of affected buffaloes, these nodules appeared first on the perineum and later on extended to teats and udder. Some of these nodules became necrotic and sloughed off. Some affected buffaloes also showed systemic signs such as salivation, nasal and lacrimal discharge. BHM affected buffaloes sustained 31.54% reduction in milk yield. In agreement with the findings of Abd-El-Hady (2015), the condition affected buffaloes

only and cattle dwelling in the same areas were spared. These signs are quite similar to those of pseudo-lumpy skin disease of cows which is also caused by the bovine herpesvirus 2 (Bitsch, 2011). Thickening of the wall of the affected teat may result in narrowing of the teat canal and thus pain and difficulty in milking (Abd-El-Hady, 2015).

In dairy buffalo, vesicle formation (a characteristics feature of early stages of BHM in cow) is either absent (Sharma *et al.*, 2005; Sharma *et al.*, 1998) or occurs rarely (Lokanadhamu *et al.*, 2005).

DIAGNOSIS

Diagnosis of BHM can be based on the composite of clinical signs, epidemiologic features, electron microscopy and serologic tests like agar gel precipitation test and neutralization test in rabbit (Janett *et al.*, 2000; Lokanadhamu *et al.*, 2005; Rao *et al.*, 2003). Histopathologic examination of biopsy samples taken from the lesions may reveal intranuclear inclusion bodies. In fact, the occurrence of multiple syncytia in the stratum spinosum layer containing basophilic or eosinophilic intranuclear inclusions is the characteristic diagnostic histologic feature of naturally occurring BHM-2 infection in buffaloes (Sharma *et al.*, 1998), cows (Rweyemamu *et al.*, 1969) and experimentally inoculated rabbits (Rao *et al.*, 2003). The condition may resemble pseudocowpox but in pseudocowpox, inclusion bodies are seen inside the cytoplasm and not in the nucleus (George *et al.*, 2008). Moreover, lesions of pseudocowpox are ring shaped (Sandrucci *et al.*, 2014). Other conditions that require consideration in differential diagnosis include stephanofilariasis (a parasitic disease of connective and subcutaneous

tissue caused by several nematode species of the genus *Stephanofilaria* e.g. *Stephanofilaria assamensis* that causes dermatitis in buffalo, goat and cattle called humpsore and *Stephanofilaria zeheeri* which is found in the inner side of the ear pinna of buffalo and causes earsore), nonspecific skin infections, photosensitivity, allergic reactions, chemical irritation and trauma (Kathirvel and Dharmaceelan, 2016), feedstuff exanthemata (Heidrich and Renk, 1967).

TREATMENT

Bovine herpes mammillitis, in particular its ulcer at the junction of teat and udder (*Gulwaddee*) does not respond to routine treatment (antibiotic injections, antibiotic creams/ointments, antiseptics etc. (Lokanadhamu *et al.*, 2005). Many cases of *Gulwaddee* in buffaloes are refractory to routine treatment for upto 3 months (Personal observation of Dr. Ghulam Muhammad). For improving the cure rate, the following treatment measures should be adopted.

Since BHM is infectious (Bitsch, 2011), the affected animal(s) should be isolated immediately and milked after healthy animals have been milked. Milking equipment used on affected animals should be sterilized by boiling or by disinfectants (Kemp *et al.*, 2008). All other biosecurity measures should also be adopted. In cases with severe thelitis (i.e. inflammation of teat), sterile teat siphon or Larson's teat tube (Abd-El-Hady, 2015) may be used for milking of the affected teat(s). Post-milking antiseptic teat dipping using iodophore teat dip (e.g. Lanodip 4:1, Kilco Chemical Ltd., UK and Masodine 1:3, Evans Vanodine International, UK) should be practiced on all animals in the herd to prevent spread of the

disease and to help a fast healing of the lesions of affected animals. In addition, insect control measures should be immediately instituted.

Chronic, large sized indolent ulcer with scabs when present at the junction of teat and udder (*Gulwaddee*) should first be properly debrided using a sterile scalpel blade. If the lesions are small, they can be cauterized by using silver nitrate stick held in hand with thick rubber gloves. For proper removal of the scab, the use of water should be avoided when cauterization is attempted with silver nitrate stick. Some practitioner use lump of copper sulphate for debridement. Debridement with sterile scalpel blade or chemical cauterization with silver nitrate stick or copper sulphate lump should be continued until a light oozing of blood starts. Since the virus of bovine herpesmammillitis is sensitive to chloroform (Sharma and Singh, 2006), after debridement, the affected area may be rinsed with chloroform followed by local application of an antibiotic cream (e.g. Bepanthen Plus cream, Bayer Health Care, Pakistan) or an insect repellent wound spray (e.g. Pinkspray, Komi Pharma, South Korea). Debridement or cauterization will have to be repeated 2 to 3 times at weekly intervals or as the situation warrants.

Syed *et al.* (2009) successfully treated a BHM affected cow with a 5 day treatment consisting of ceftriaxone injection (3 grams daily intramuscular), injection chlorpheniramine maleate (10 ml intramuscular) and injection meloxicam (10 ml intramuscular). In addition, the ulcerated area was treated with povidone-iodine ointment twice daily.

Use of drugs for correction of immunosuppression: Since the cows and buffaloes that suffer from BHM are in a state of immunosuppression (Bitsch, 2011), immunity enhancing agents such as levamisole (e.g. Nilverm;

about 80 ml per cow or buffalo per day for 4 days PO), Inj. vitamin E-selenium (e.g. Inj. Selevit Fatro Pharma), and Zinc Sulphate (e.g. zinc sulphate marketed by Syngenta Company which can be obtained from stores which sell crop fertilizers) 2 to 3 grams per cow or buffalo per day for 10 days can be used for improving the immunity against the herpes virus.

(Lokanadhamu *et al.*, 2005) compared 3 treatment protocols for the treatment of herpes mammillitis in 24 buffaloes. Protocol A consisted of Inj. enrofloxacin (15 ml of 10% solution), Inj. diclofenic sodium (20 ml), Inj. Avil (pheniramine maleate; 10 ml), Tab. Acivir (acyclovir; 800 mg; 3 tablets daily), topical application of tablet lysine and topical application of ointment Acivir (5%). Protocol B was the same as protocol A except that oral administration of tab. Acivir and topical application of lysine was omitted. Protocol C was the conventional treatment consisting of Inj. enrofloxacin, Inj. diclofenic sodium and Inj. Avil. The cure rate with protocol A was 62.5% in 12 days whereas the cure rate with protocol B was 37.5% within 24 days. The conventional treatment (protocol C) had the cure rate of only 12.5% after 35 days treatment. Acyclovir is an antiviral drug specifically effective against herpes infections. Lysine has been shown to reduce the severity of herpes infections in herpes simplex infections of human (Griffith *et al.*, 1987). Other workers (Abd-El-Hady, 2015; Bentz *et al.*, 2006; Lokanadhamu *et al.*, 2005; Purohit *et al.*, 2014) have also suggested the combined use of antibiotics, antihistamines and anti-inflammatory drug to hasten the recovery and to prevent secondary bacterial complications like mastitis.

In horses, a high variability in serum acyclovir-time profiles, low C_{max} (i.e. peak serum concentration of the drug) as well as a poor

bioavailability after intragastric administration of 10 or 20 mg/kg acyclovir do not support a therapeutic benefit for the oral dosing of horses with this antiherpetic drug). However, there is no (Bentz *et al.*, 2006) data on the pharmacokinetics of acyclovir in cows and buffaloes. Ten ml vials each containing 500 mg acyclovir sodium are marketed in Pakistan by Abbott Pharma. Local infiltration of this drug in cases of *Gulwaddee* may potentially be effective in this form of BHM. Nonetheless, local infiltration of this antiherpesvirus drug has not as yet been evaluated in the treatment of *Gulwaddee*.

According to (Letchworth and Carmichael, 1984; Kathirvel and Dharmaceelan, 2016), the assembly and maturation of BHM virus is inhibited at higher body temperature. This is probably because higher body temperature stimulates local production of highly active form of interferon which inhibits the replication of BHM virus. Temperature of skin of teats and udder is lower than that of body and the interferon produced locally in the BHM virus affected area of teats and udder is not only low in quantity but also of low specific activity. That is why BHM virus mostly produces lesions in the cool skin areas (i.e. skin of teats and udder where the temperature is lower than the rest of the body due to low blood circulation). Temperature of the BHM lesions on the teats and udder can be raised theoretically by using infrared lamp (a fairly cheap lamp used in human medicine). However, this lamp has not as yet been tried in the treatment of *Gulwaddee* and other clinical forms of BMH.

Abd-El-Hady (2015) suggested that an early treatment of ulcerative thelitis with local application of 10% zinc oxide ointment along with anti-inflammatory enzyme and antibiotic can prevent ulceration, necrosis and sloughing of teats. He further documented that application of propolis (a resinous mixture that honey bees collect from

tree buds, sap flows, or other botanical sources and used as a sealant for unwanted open spaces in the hive) on affected teat at the ulcerative stage speeds up the healing and restoration of milk yield within two weeks. A small percentage of BHM affected animals may develop ischemic necrosis of the teat base (Sharma and Singh, 2006). In view of an impending danger of teat sloughing, these cases require heroic treatment efforts including debridement local and oral administration of acyclovir, topical application of lysine, recouplement of immunity by zinc sulphate and other immune enhancers, local antiseptic and parenteral antibiotic.

CONTROL AND PREVENTION

Do not purchase BHM affected cows and buffaloes. Check the udders and teats of animals at the time of their purchase. Healing of BHM may leave depigmented skin area on the teat (Gibbs *et al.*, 1970; Yager and Scott, 1992) which may be used as an indication of an exposure of the cows and buffaloes to BHM virus. It is recommended that such animals should never be purchased. Currently, no commercial vaccine is available for immunization against BHM.

Since, edema of udder is a very important predisposing factor to BHM (Bitsch, 2011), all measures should be taken to prevent the occurrence of parturient udder edema and if it still occurs, appropriate therapy should be instituted in a timely manner.

Insect should be controlled as they have documented to transmit BHM virus (Bitsch, 2011).

Milking time hygiene and post milking antiseptic teat dipping are obviously important particularly in the face of an outbreak of bovine

herpes mammillitis.

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