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Review Article

Udder and Teat Skin Lesions in Bovines

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Abstract

Udder and teat lesions are early indicators for increased risk of mastitis. It causes financial losses by decrease milk production, loss of quarter, reduced cost of animal and aesthetic value. The susceptibility to udder diseases is high among pure exotic breeds like HF, Jersey etc. followed by crossbreds. The resistance of local breeds of cattle and buffaloes to the infection is basically attributed to their lower milk production and a better immunity levels. The teat sphincter and teat canal are important primary barriers against pathogen invasion into the udder. Teat lesions, generally do not cause mastitis directly, but do interfere with the milking process and may cause secondary problems. These lesions are readily colonized by bacteria and thus serve as an important reservoir of infection. Economically, losses may be severe as animals usually resist milking and must be culled. In future mastitis research, understanding the way in which microorganisms penetrate the teat canal will become increasingly important. This review comprehends the clinical understanding of udder and teat lesions in dairy animals.

Keywords: Buffalo, Cow, Skin Lesions, Teat, Udder

Introduction

Bovine mastitis is most prevalent disease in dairy animals affecting production efficiency, and one of the major problems affecting world's dairy industry (Miller and Dorn, 1990; Miller *et al.,* 1993; Bennett *et al.,* 1999). In India, the value of output from dairy sector was more than Rs. 549 billions and accounts for 67.69 per cent of the value of the livestock sector output in the year 2015-16 (Shree and Prabhu, 2019). At present, milk is the largest agricultural produce in terms of value, it is valued at around Rs. 6.50 lakh crores, which is more than the total value of paddy and wheat put together. Healthy udder and teat skin are desirable for welfare, comfort and health of animals, for aesthetic reasons, for the production of wholesome milk and also for the health and comfort of those involved in milking (Francis, 1984). Teat and udder lesions are quite common and somewhat neglected problem in bovines. Pure exotic breeds are highly susceptible followed by crossbred animals, while buffalos are least affected. The lower susceptibility of local breeds is basically due to ~~its~~ better immunity levels and low production (Archana *et al.,* 2019). These lesions are not fatal to animals but can undoubtedly be classified amongst the diseases of great economic importance to dairy industry. Teat lesions may cause deterioration of animal health, severe milking problems, prolonged milking time and development of mastitis. Radostits *et al.* (2007) reported 20% decline in milk yield in cows suffering from herpes mammillitis.

Skin lesions on udder and teat may be primary diseases occurring mainly on udder/ teat skin such as bovine herpes mammillitis (BHM), pseudocowpox, cowpox, buffalopox, papillomatosis and udder impetigo or these may be secondary and incidental symptoms of a generalized disease like foot and mouth disease (FMD), bovine papular stomatitis, vesicular stomatitis, contagious pustular dermatitis (orf) and lumpy skin disease. The literature on teat skin lesions in dairy animals was reviewed by various authors (Martin *et al.,* 1972; Francis, 1984; Gibbs, 1984; Kahrs, 1985; Carter *et al.,* 1995; Wellenberg *et al.,* 2002; Waller, 2004). The occurrence of mastitis due to bacterial and fungal causes has been well established (Watts, 1988; Radostits *et al.,* 2007) but the possible role of teat skin lesions in development of mastitis is poorly understood and needs to be investigated (Wellenberg *et al.,* 2002). Under field conditions, it is difficult to differentiate between different teat skin lesions, and therefore appropriate preventive and control measures cannot be taken leading to huge economic losses. Keeping in view, the present review has been undertaken to better understand the problem and suggest curative and control measures against udder and teat lesions in bovines.

Prevalence

The prevalence of teat lesions varies according to method of sampling and conditions included in the study. Some studies were conducted at the slaughter houses (Sobari *et al.,* 1976) and others included teat injuries, mastitis, udder fibrosis and other related problems during the prevalence studies (Sobari *et al.,* 1976; Thilagar and Dewanmuthumohammed, 1992). Sobari *et al.* (1976) recorded lesions in 11.4% of slaughtered beef cows and classified lesions as cysts, leech infestation, mastitis, teat occlusion, papillomatosis, cutaneous horns, teat fistulas, teat trauma and udder fibrosis. Sieber and Farnsworth (1981) after a field study on 1213 dairy cattle that only 15.8% of the teats were free from teat-end lesions whereas Graf and Gedek (1983) recorded teat end lesions in 74.1% teats at machine milked dairy farms. Agger and Willeberg (1986) examined the herd for a year at monthly interval and found 154 teat lesions in a herd of 188 cows. Thilagar and Dewanmuthumohammed (1992) classified teat lesions as mastitis (67.5%), mastitis and thelitis (4.6%), teat obstruction (20.5%), thelitis (2.0%), wound (0.7%), abscess (2.0%), psychic agalactia (0.7%), pox (0.7%), teat stricture (0.7%) and ulcer (0.7%). Jhand *et al.* (1994) reported the incidence of teat lesions to be 17.0% and included teat injuries, supernumerary teats, blind teats, warts, teat abscess and udder skin injury. The cumulative incidence of infectious teat skin lesions in a year was 17.3% and 8.5% in cattle and buffaloes, respectively (Sharma *et al.,* 2005). The lesions recorded during the study were herpes mammillitis, pseudocowpox, cowpox, buffalopox, papillomatosis, udder impetigo and FMD teat lesions. The reason for better protection of buffalo teat skin than cattle against the inclement environment could be the more thickness of teat epithelium in buffaloes as compared to cattle; moreover, buffalo teat epidermis has comparatively higher amount of melanin than in cattle (Kumar, 1990). (Kumar *et al.,* 2012) recorded a total of 111 udder and teat lesions on examination of 494 quarters from 124 cows at machine milked farms. The prevalence of udder and teat lesions was (22.46%) at machine milked farms. However, a total of 55 udder and teat lesions were recorded on examination of 348 quarters from 87 cows at hand milked farms. The prevalence of udder and teat lesions was (15.80%) at hand milked farms. The major teat lesions reported were warts (55.8%) and dryness of teat skin (30.9%). Mir *et al.* (2014) studied udder and teat lesions in 872 quarters from 218 HF × Sahiwal cross-bred lactating cows and reported prevalence as 23 per cent on quarter basis. The type of lesions observed were warts (6.01%), dryness (5.54%),

vesicles/papules (2.83%), leaky teats (2.59%), eversion (2.36%), nodules/hard tip (1.30%), wound/laceration (1.30%) and scab (0.70%).

Bovine Ulcerative Mammillitis

Bovine ulcerative mammillitis (synonym bovine herpes mammillitis, BHM) is viral disease characterized by severe ulceration of the skin of udder and teats. The causative bovine herpesvirus-2 (BHV-2) is identical with Allerton virus of lumpy skin disease and with dermatotrophic bovine herpesvirus (Radostits *et al.,* 2007). In addition to causing ulcerative mammillitis, BHV-2 is also associated with generalized skin disease in cattle known as pseudo-lumpy skin disease (Haig, 1967; St. George *et al.,* 1980; Woods *et al.,* 1996). BHV-4 has been isolated from cattle with mammary pustular dermatitis (Reed *et al.,* 1977) and chronic ulcerative mammary dermatitis (Cavirani *et al.,* 1990). The virus does not have any zoonotic significance (Radostits et al., 2007). The disease was first described as skin gangrene of bovine udder by White *et al.* (1959). Martin *et al.* (1964) isolated virus from a case of ulcerative lesion of cows in Ayrshire. Since then, outbreaks of disease has been reported by several authors in different countries of the world producing ulcerative lesions on udder and teat in cattle (Pepper *et al.,* 1966; Castrucci *et al.,* 1972; Dadiri, 1973; Gibbs *et al.,* 1972; Weaver *et al.,* 1972; Letchworth and LaDue, 1982; Muller *et al.,* 1984; Lenihan *et al.,* 1985; Scott and Holliman, 1986; Martin *et al.,* 1987; Chauhan *et al.,* 1989; Gourreau *et al.,* 1989; Moriello *et al.,* 1993).

The incidence of disease varies between 15-90% during the outbreaks but in most of cases it is about 30% (Radostits *et al.,* 2007). There are two epidemiological patterns of disease- in some outbreaks, the disease spreads rapidly within the herd and in others, the infection is confined to recently calved animals. The lesions are mainly observed during the first lactation in cattle and buffaloes and that too within first two months of lactation. According to Martin (1973), heifers become infected before the calving and at calving, stress and hormonal changes precipitate the activation of virus which results in severe clinical disease; moreover BHV-2 replicates more readily in the skin with lower than normal body temperature which occurs commonly after parturition in the edematous area around the udder (Martin, 1990). The reduction in cell-mediated immunity that occurs at advanced pregnancy and shortly after parturition allows replication of herpesvirus so lesions occur shortly after calving. The lesions occur mostly during the winter season (Martin, 1990). According to Letchworth (1980), low temperature stimulates replication of BHV-2 and reduces the production of interferon. Moriello *et al.* (1993) recorded seasonal vesicular disease similar to BHM in the state of Wisconsin but the disease had only sporadic occurrence and has non-infectious pattern of occurrence.

In cattle, initial vesicles rupture within 24 hours exposing the congested dermis. The vesicles are thin walled, 1-2 cm in diameter and often present at the base of teat. In most of cases, vesicles are not commonly seen. In severe cases, entire teat becomes swollen and painful, exudation of serum leaving a raw ulcer covering most of the teat. In later stages, the exudate dries over the lesion to form a flat smooth scab. There are no signs of systemic illness in the affected animals (Martin, 1973; Gibbs and Rweyemamu, 1977). In buffaloes (*Bubalus bubalis*), the typical ulcerative lesions on udder-teat junction had been named as necrotic thelitis, allergic mammitis, non-specific mammillo-mammitis on the basis of clinical symptoms (Sankaram and Kotayya, 1977; Nauriyal and Randhawa, 1982; Mouli, 1991; Mouli, 1992; Sreeramulu, 1993; Sundaresh and Sundaresh, 1997). Later on, these lesions were found to be associated with herpesvirus (Sharma *et al.,* 1998; Rao *et al.,* 2003; Lokanadhamu *et al.,* 2005). The disease in buffaloes start with the formation of localized swollen plaques (2 to 5 mm diameter) within the thickness of teat wall surrounded by inflammatory zone that ruptures within 48 hours and produce very deep ulcers. The vesicle formation is not observed in buffaloes and the deep raw ulcer or ulcer covered with thick blue-black scab is most common presenting lesion.

Histopathological examination in early cases reveals hydropic degeneration, necrosis and intercellular edema along with marked thickening of epidermis. Multiple syncytia formation containing basophilic or acidophilic inclusion bodies in their nuclei is observed. The formation of syncytia containing intranuclear inclusions is a characteristic feature of BHV-2 infection (Pepper *et al.,* 1966; Martin *et al.,* 1969; Rweyemamu *et al.,* 1969b; Gibbs *et al.,* 1970a; Johnston *et al.,* 1971; Gibbs and Collings, 1972; Chauhan *et al.,* 1989; Sharma *et al.,* 1998). Intradermal inoculation of suspected material from early vesicles in the rabbits produces dermonecrosis (Martin, 1990; Rao *et al.,* 2003; Lokanadhamu *et al.,* 2005). Electron microscopy is best for the rapid diagnosis of BHV-2 infections (Sharma *et al.,* 1998). Serological tests can be employed for the diagnosis of BHV-2 infections. The presence of high virus-neutralizing antibody titers in paired serum samples may support the diagnosis (Rweyemamu and Johnson, 1968;

Martin and Scott, 1979; O'Connor, 1987; O'Connor *et al.,* 1994; Janeett *et al.,* 2000). The serum neutralization test (Rweyemamu *et al.,* 1969a; Engels *et al.,* 1979; Pastoret *et al.,* 1983; St. George, 1983; Scott and Holliman, 1984; Tabbaa and Liebermann, 1989) and ELISA (Geiger *et al.,* 1990) can be employed to know seroprevalence of antibodies against BHV-2. Imai *et al.* (2002) used shuttle PCR for the detection of BHV-2.

Pseudocowpox

Two forms of cowpox were recognized since ancient times; 'clinical cowpox', that confers immunity in humans against smallpox and 'spurious cowpox', that does not provide immunity. The later form of disease is now known as pseudocowpox (Daniel, 1970). Pseudocowpox virus is member of the genus *Parapoxvirus* and is related to viruses of contagious ecthyma and infectious pustular stomatitis. The virus was first time isolated in fetal bovine testes tissue culture from pseudocowpox lesions from cows' teat and from lesions in calf mouth (Moscovici *et al.,* 1963). Friedman-Kien *et al.* (1963) isolated virus with similar characteristics from a case of milker's nodules in man. The disease is of zoonotic significance (Lauder *et al.,* 1966; Naginton *et al.,* 1966; Naginton *et al.,* 1967; Neale and Calvert, 1967; Tuaillon *et al.,* 1975; Peisslinger, 1982). The viruses of pseudocowpox, contagious pustular dermatitis and bovine popular stomatitis have been classified into subgroup paravaccinia viruses (Peter *et al.,* 1964; Naginton *et al.,* 1967; Carter *et al.,* 1968). Pseudocowpox is recognized as common teat skin lesion in many countries of the world (Huck, 1966; Naginton *et al.,* 1966; Cheville and Shey, 1967; Johnston and Shorey, 1967; Morita *et al.,* 1967; Carter *et al.,* 1968; Gagnon *et al.,* 1973; Gibbs and Osborne, 1974; Moussa *et al.,* 1983; Sharma, 1997) and found to be endemic in Britain affecting up to 10% of cows' teats (Francis, 1983). There is only one report concerning the isolation of poxvirus from the milk (Dawson *et al.,* 1968).

The source of infection and spread to cattle occur through milker's hands, wash cloths and teat cups. Recently calved animals are most susceptible to disease. There may be pyrexia in the affected animals (Gibbs, 1984) whereas cowpox is not associated with fever (Heidrich and Renk, 1967). Initially erythematous eruptions of 2 to 4 mm occur on the teats, which are followed by extensive scab formation within 48 hours. Naginton *et al.* (1966) and Daniel (1970) reported the formation of small vesicles during the disease. The healing started from the centre and a characteristic 'horseshoe or signet ring' can be observed within 8 to 10 days in some of the recovering animals. The ring scab is pathognomonic lesion for the diagnosis of pseudocowpox (Naginton *et al.,* 1966; Daniel, 1970; Gibbs, 1984).

Histopathological examination reveals hydropic degeneration, vesiculation and ballooning in the prickle cell layer of epidermis together with the presence of small eosinophillic intracytoplasmic inclusion bodies characteristic of pox infection (Moscovici *et al.,* 1963; Daniel, 1970). Inoculation of suspected material on chorioallantoic membrane of chicken embryo or on rabbits does not produce any lesions (Moscovici *et al.,* 1963; Heidrich and Renk, 1967; Daniel, 1970; Gagnon *et al.,* 1973) differentiating it from cowpox, which produces large, clear and raised white pocks with hemorrhagic spots in the centre (Sambyal *et al.,* 1983; Silva *et al.,* 1986). Electron microscopy can be employed for the rapid confirmation revealing parapoxvirus with 9 to 10 'crossovers' of surrounding spiral thread (Johnston and Shorey, 1967; Carter *et al.,* 1968). Inoshima *et al.* (2000) developed PCR for the diagnosis of pseudocowpox.

Cowpox/ Buffalopox

Cowpox and buffalopox is caused by *orthipoxvirus* in the family Poxviridae. The disease is very rare in European and developed countries (Mayr and Czerny, 1990), however outbreaks of disease continue to occur in most of developing nations including India. Various outbreaks of pox have been reported in the developing countries (Ganiev and Farzaliev, 1964; Mallick *et al.,* 1990; Al-Gaabary and Rawash, 2004). The transmission mostly occurs from cow to cow by milker's hands. Human cowpox is not common and usually consists of one or few lesions on the hand and face with minimal systemic reaction. Milk from the affected cows is suitable for human consumption (Radostits *et al.,* 2007).

In cowpox/ buffalopox, the five stages of typical pox can be observed. Initial erythema is followed by firm, raised papules light in colour but with a zone of hyperemia around the base. Vesicles are yellow coloured blisters with a pitted centre followed by pustular stage and then development of thick, red tenacious scab 1-1.5 cm in diameter (Radostits *et al.,* 2007). Initially, typical pox lesions are seen mainly on the udder and teats, but later on, the lesions may spread to base of horns, inside thighs and on perineal region. Variable reports are available in the literature concerning the localization of pox lesions. Ghosh *et al.* (1977) observed buffalopox lesions restricted to udder and

teats only. Karmakar and Saha (1989) and Muraleedharan *et al.* (1989) reported buffalopox outbreaks localized to ears and eyes only whereas a case of generalized buffalopox involving udder, teats, medial aspects of thighs, lips, nostrils, eyes, neck and abdomen had also been documented (Anantwar *et al.,* 1985). Ghosh *et al.* (1977), Mathew *et al.* (1978) and Kumar *et al.* (1987) observed pox lesions on the hands and fingers of human beings during the outbreaks of buffalopox. The cases of mastitis usually develop following cowpox virus infection (Sambyal *et al.,* 1983). The cowpox virus produces typical pock lesions on CAM of chicken embryo and can be transmitted to rabbits (Reis *et al.,* 1970; Gibbs *et al.,* 1973; Mathew, 1976; Tantawi *et al.,* 1976; Chandra *et al.,* 1986; Silva *et al.,* 1986); cultivated on pup kidney cell culture (Singh *et al.,* 1984) and confirmed by electron microscopy (Skalinskii and Borisovich, 1978).

Papillomatosis

Bovine papillomatosis (warts) caused by bovine papillomas virus (BPV) may be recognized as hyperplasia or benign neoplasms of epidermis (Lancaster and Olson, 1982). The papillomas may be present on cutaneous body parts and udder/ teats. There are 6 different types of BPV producing distinct types of papillomas grosally as well as microscopically and have predilection for particular site of body (Olson, 1990). The virus may produce papillomas on cutaneous body parts viz. head, neck, shoulders and ventral abdomen (BPV-2,3), alimentary tract (BPV-4) and udder/teat skin (BPV-1,5,6). There are distinct types of papillomas viz. frond form having filiform projections on them and approximately one cm in length; flat papillomas are round type, usually multiple and always sessile and up to 2 cm in diameter; and rice grain papillomas are elongated structures (Radostits *et al.,* 2007). Wettimuny and Panangala (1972) observed teat papillomas in 14% of cattle surveyed. Meischke (1979) classified papilloma types as frond (27%), flat and frond (20%) and rice grain like (53%). Olson *et al.* (1982) recorded the frequency of papillomas types as atypical flat papillomas (62.1%), atypical filiform papillomas (30.5%) and typical fibropapillomas (7.4%). Sharma *et al.* (2004a) classified teat papillomas as flat type (53.4%), filiform type (33.3%) and cauliflower like (13.3%) on the basis of macroscopic appearance. In most of the cases, only single teat is affected (Olson *et al.,* 1982; Sharma *et al.,* 2004a) and there may be concurrent papillomas on the udder in some cases (Lindholm *et al.,* 1984; Sharma *et al.,* 2004a). Samir *et al.* (2014) reported 1.65% incidence of teat papilloma and its incidence was higher in right hind quarters (0.67 %), followed by right forequarters (0.32%). Teat warts may regress during the dry period and recur in the next lactation. Teat warts usually show increased frequency with the age (Radostits *et al.,* 2007).

The histopathological changes in cutaneous papillomas are typical of fibropapilloma revealing acanthosis and hyperkeratosis in the epidermis along with elongated growths of epidermis into the dermis. The dermis is also hyperplastic along with marked proliferation of connective tissues (Wadhwa *et al.,* 1996, Sharma *et al.,* 2003). However, in udder/teat papillomas, typical fibropapilloma is not observed and only epithelial hyperplasia is seen in most of cases without any fibromatous proliferation (Cappellaro *et al.,* 1979; Olson *et al.,* 1982; Sharma *et al.,* 2003). Barthold *et al.* (1974) recorded atypical warts in cattle which were differentiated from typical cutaneous papillomas on the basis of lack of fibromatous dermal component. The papillomaviruses cannot be cultured, and the differentiation of types is based on the histological features, electron microscopy (Moussa *et al.,* 1983; Gerdes and Lugt, 1991) and DNA identification by hybridization or PCR (Radostits *et al.,* 2007).

FMD Teat Lesions

Foot and mouth disease (FMD) is caused by aphthovirus of the family Picornaviridae. Lesions on muzzle and feet characteristic of FMD are present along with systemic signs. In addition, thin walled vesicles filled with serous fluid on the teat are seen in some cases which rupture within 24 hours leaving bright red painful erosions. The FMD needs to be differentiated from more common viral teat infections as pustular lesions appear on teats before their presence in the mouth (Hillerton *et al*, 2000). The virus replicates in the secretory epithelial cells of mammary gland and is isolated from the milk of affected cows (Burrows, 1968; Ray *et al.,* 1989; Fuchs, 1994). There is evidence of virus replication in the mammary gland following systemic infection (Blackwell *et al.,* 1981; Blackwell *et al.,* 1983), aerosol route (Blackwell and Yilma, 1981) or after intramammary inoculation (Burrows *et al.,* 1971). From the udder and teat lesions, FMD virus serotype Asia-1 (Firoozi *et al.,* 1974), serotype O (Sarma and Boro, 1984), serotype C (Uppal *et al.,* 1984) and serotype A_{22} (Sharma *et al.,* 2004b) has been identified. An increased incidence of mastitis with secondary bacterial pathogens is reported after an infection with FMD virus (Singh *et al.,* 1981; Ray *et al.,* 1989; Seinhorst *et al.,* 1991; Saini *et al.,* 1992).

Udder Impetigo

The lesions of udder impetigo start with the appearance of numerous small, creamish-yellow and 2-4 mm diameter pustules on the udder. The lesions are mostly observed on the lateral sides as well as in the well of the udder, but sometimes also present on hairless skin at the base of the teats (Francis, 1984; Faull *et al.,* 1991). Yeruham *et al.* (1996) reported lesions of contagious impetigo on the vulvar region, perineum as well as on udder. The cases are mostly reported in the summer season and *Staphylococcus aureus* organism is mostly isolated from the lesions (Johnston, 1972; Yeruham *et al.,* 1996).

Teat End Hyperkeratosis

Teat-end hyperkeratosis is a thickening of the skin that lines the teat canal and surrounds the external teat orifice. The condition is variously described as teat rings, teat flowers, teat erosion, callus formation, callosity, certification or teat-end roughness. Mir (2009) observed that teats with smooth ring were found to be the most common 352 (40.55%) followed by teats rough ends 220 (25.35%), normal teats with no ring 208 (23.96%), teats with very rough ends 61 (7.03%) and teats with open lesions or scabs 27 (3.11%). Moret-Stalder *et al.* (2009) also observed the higher percentage of teats with normal to smooth rings followed by rough to very rough rings. Bade *et al.* (2007b) in a survey of teat-end condition on commercial farms indicated that the percentage of cows with rough or very rough teat ends averaged about 50% with some farms exceeding 70% and some farms less than 20%. The ability of the teat to prevent infection is associated with the condition of the teat-end (O'Shea *et al*., 1987). Therefore, teat-end condition, often measured in terms of teat-end callosity (TEC) or teat end score, is an important physiological parameter. The recent histological studies have observed that these changes result from an increase or buildup of callous tissue around the orifice orifice rather than an 'erosion' of teat tissue or the orifice (Neijenhius *et al*., 2001). Changes in teat-end tissue result from mechanical forces exerted by vacuum and the collapsing liner during machine milking. The magnitude of forces depends on milking vacuum, pulsation, machine-on time, liner type and teat shape (Mein and Thompson,1993). Teats with a smooth thin callosity ring have been observed to be at the lowest risk of mastitis. On the other hand, thicker and rough callosity teats have the highest incidence of mastitis. Also, teat ends with severe erosions that are raw and ulcerated show higher prevalence of mastitis (Farnsworth, 1995). A hyperplastic stratum corneum, found in teats with an increased TEC score, leads to a roughened surface to which bacteria can adhere, making disinfection of the teat after milking more difficult and limiting its effectiveness (Neijenhuis *et al*, 2000).

Therapy

Papillomatosis

Different therapeutic regimens have been tried against papillomatosis with variable success. Papillomas can be removed by surgery or cryosurgery, but surgical removal is difficult when multiple papillomas are present on the teat/ body. Surgical removal can be followed by vaccination with autogenous vaccine (Ssenyonga *et al.,* 1990). Barthold *et al.* (1976) found that formalin inactivated BPV was capable of inducing precipitin antibody and observed that repeated vaccination can produce best serological response. The autogenous vaccine can cause complete regression of papillomas within 20-40 days of treatment (Sharma, 1997). Wadhwa *et al.* (1995) reported 71% efficacy of autogenous vaccine against papillomatosis. Rajguru *et al.* (1988) and Makay (1989) also reported autogneous vaccine to be highly successful in treating both cutaneous and teat papillomas. The efficacy of autogenous vaccine against cutaneous and teat papillomatosis was found to be 80-85% and 60.0%, respectively (Radostits *et al.,* 2007). The reason for lower efficacy of autogenous vaccine against teat papillomas could be that typical fibropapillomas occurring commonly on head and neck is seen rarely on the teats and viral capsid antigen is present in lower concentration in teat papillomas as compared to typical fibropapilloma (Olson *et al.,* 1982). The rejection responses induced by autogenous vaccine might be caused by virus antigens or other antigens expressed in or on tumour cells (Lindholm *et al.,* 1984). The autogenous vaccine has to be prepared separately for each animal and care should be taken during selection of tissues from the affected animal. The tissue has to be selected on the basis of tumour type, location and histological composition or one can include many types of tissues in a vaccine (Radostits *et al.,* 2007). The stage of development is also important and virus is present in much greater concentration in the epithelial tissue of older warts than young ones (Jarrett *et al.,* 1984). Cimtay *et al*. (2003) observed that vaccine prepared from the warts is quite effective against bovine papillomatosis but recovery in autogenous and herd specific vaccination is earlier than in species specific vaccination groups.

The efficacy of lithium antimony thiomalate was 75.0 and 57.1 per cent against cutaneous and teat papillomas, respectively (Sharma, 1997). Wadhwa *et al.* (1992) could treat two out of three cows with lithium antimony thiomalate having teat papillomas. Wadhwa *et al.* (1996) observed 55.5% efficacy of lithium antimony thiomalate against papillomatosis. However, in contrast Prasad *et al.* (1980) and Gupta *et al.* (1989) could not observe the complete removal of papillomas by lithium antimony thiomalate. Besides, some authors employed other treatment protocols against bovine papillomatosis with variable success viz. levamisole (Cihan *et al.*, 2004), fig tree latex (Hemmatzadeh *et al.*, 2003), autohemotherapy (Bajric *et al.*, 1974; Biricik *et al.*, 2003).

Other Skin Lesions

Post-milking teat dips can be used for the treatment of teat skin lesions besides systemic and intramammary antibiotic therapy for the treatment of mastitis. As most of teat skin lesions are viral in origin, therefore teat dips are applied mainly to speed up the recovery of teats and preventing mastitis and bacterial dermatitis as most of teat dips are bactericidal and repellent to insects (Francis, 1984; Gibbs, 1984). They also recommended the addition of glycerin to rehydrate teat skin or lanolin to prevent drying of teat skin in the post-milking teat dips. The iodophor solution is more efficient in destroying the infectivity of bovine mammillitis virus and pseudocowpox virus than a hypochlorite solution (Martin and James, 1969; Puddle *et al.*, 1986). Fox (1994) reported commercial iodine dip to be highly effective in preventing *Staphylococcus aureus* colonization of teat skin. However, no form of treatment was of value in shortening the healing of BHM lesions (Pepper *et al.*, 1966). Martin *et al.*, 1966) recommended the use of emollients before milking to reduce trauma and bleeding of ulcerated areas. Smith (1966) recommended removal of scabs and application of tincture for treatment of pseudo cow pox.

Conclusion

Udder and teat skin lesions are quite common but not properly addressed in dairy animals. Mostly pure exotic breeds are prone to these lesions rendering them susceptible for increased risk of mastitis. These lesions are not fatal to animals but are of great economic importance to dairy industry. Teat lesions may cause deterioration of animal health, severe milking problems, prolonged milking time and development of mastitis. Skin lesions on udder and teat may be primary diseases or secondary and incidental symptoms of a generalized disease. Their possible role in development of mastitis is poorly understood and needs to be investigated. There is utmost need to differentiate between different udder and teat skin lesions so that appropriate prevention and control measures can be taken to avert huge economic losses.

Conflict of Interests

There is no conflict of interest.

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