Acta Tropica 158 (2016) 32–38

Contents lists available at ScienceDirect

Acta Tropica

journal homepage: www.elsevier.com/locate/actatropica

Camelpox: A brief review on its epidemiology, current status and challenges

Shyam Singh Dahiya[a,*], Sachin Kumar[b], Sharat Chandra Mehta[a], Shirish D. Narnaware[a], Raghvendar Singh[a], Fateh Chand Tuteja[a]

[a] National Research Center on Camel, Jorbeer, Bikaner, Rajasthan 334001, India
[b] Department of Biosciences and Bioengineering, Indian Institute of Technology Guwahati, Assam 781039, India

ARTICLE INFO

Article history:
Received 17 December 2015
Received in revised form 12 February 2016
Accepted 18 February 2016
Available online 20 February 2016

Keywords:
Camelpox
Zoonosis
Outbreak
Vaccine
Immune evasion

ABSTRACT

Camelpox caused by a Camelpox virus (CMLV) is a very important host specific viral disease of camel. It is highly contagious in nature and causes serious impact on health even mortality of camels and economic losses to the camel owners. It manifests itself either in the local/mild or generalized/severe form. Various outbreaks of different pathogenicity have been reported from camel dwelling areas of the world. CMLV has been characterized in embryonated chicken eggs with the production of characteristic pock lesions and in various cell lines with the capacity to induce giant cells. Being of *Poxviridae* family, CMLV employs various strategies to impede host immune system and facilitates its own pathogenesis. Both live and attenuated vaccine has been found effective against CMLV infection. The present review gives a comprehensive overview of camelpox disease with respect to its transmission, epidemiology, virion characteristics, viral life cycle, host interaction and its immune modulation.

Contents

1. Introduction

Camelpox is a highly contagious disease of camels with an exception of dromedary camel in Australia and tylopods (llama and related species) in South America (Mosadeghhesari et al., 2014; Pfeffer et al., 1998; Wernery and Kaaden, 2002). The disease can cause high mortality and morbidity in camels leading to greater economic impact in the countries like India and Middle East. The camelpox causes higher mortality in young animals than in adults (Kriz, 1982). The disease can manifest itself in two distinct forms, i.e. milder and localized in old camels or severe and generalized in young camels which may cause heavy mortality (Wernery and Kaaden, 2002). The disease causes a decrease in milk production in lactating animals, weight loss and debilitating condition in all the infected animals (Kinne et al., 1998). Outbreaks have often been attributed to weaning or poor nutrition, which may sometimes result into severe fatality (Bhanuprakash et al., 2010a). Since 1978, the smallpox vaccination has been stopped and the concern has been shown as the human population has become susceptible

* Corresponding author.
E-mail address: dshsing04@gmail.com (S.S. Dahiya).

http://dx.doi.org/10.1016/j.actatropica.2016.02.014
0001-706X/

to emerging or re-emerging of any virus from the poxvirus group (VARV, monkeypox, CMLV or taterapox virus) (Gubser and Smith, 2002). Camelpox is important not only to control the economic losses in camel rearing areas (Otterbein et al., 1996) but to deal with it in a logistic and holistic approach to check its spread to other nearby areas also (Bhanuprakash et al., 2010a).

Present review gives a comprehensive overview of camelpox disease with respect to its epidemiology, mode of transmission, virion characteristics and its immune modulation.

2. Global scenario

In the list of many primordial viral infections known to mankind, poxviruses have its own important place among the most feared viruses of livestock animals and humans (Essbauer et al., 2009). As per the United Nation Food and Agriculture Organization, there are approximately 20 million camels in the world (http://faostat.fao.org). Camelpox was first reported from the Punjab province in India in the year 1909 (Wernery and Kaaden, 2002). Camelpox incidences have been reported from the Middle East, Asia, Africa, Southern Russia and India (Sharawi et al., 2011; Wernery and Kaaden, 2002). OIE has declared camelpox as a reportable disease (Bray and Babiuk, 2011). Sporadic outbreaks of camelpox occur in the camel dwelling areas and their incidences increases seasonally especially, during the rainy season (Anonymous, 2008). From an economic point of view, camelpox is possibly the most important orthopoxviral disease (Azwai et al., 1996). In camel rearing countries, camel provides a mode of transport, milk, meat and wool for both nomadic as well as non-nomadic people (Duraffour et al., 2011b). The presence of camelpox in the camel herd has great significance because of its economical impact over the camel owners (Bett et al., 2009; Pfeffer et al., 1998; Wernery and Kaaden, 2002) in terms of loss of health condition, secondary infections, loss of weight and milk production (Duraffour et al., 2011b). Moreover, camelpox is considered important in camel trade and is viewed carefully in order to avoid its transmission to the adjacent camel harbouring countries (Bhanuprakash et al., 2010a). Further, death of the camel by camelpox deprives camel owners of their milk and makes camel calf undernourished which brings greater threat to dreadful diseases and higher infant mortality (Kriz, 1982). Camelpox has been designated as very contagious and extremely transmissible skin disease of camels. CMLV and variola virus (VARV)—causative agent of smallpox, share genome co-linearity and are also unique in infecting a single host species (Fenner, 1989). Exploring the genomics and biological aspects of CMLV can help us to understand the VARV (Duraffour et al., 2011b) as there is a possibility of smallpox-like disease in some immune-compromised human population following its infection (Baxby, 1975).

3. Epidemiology

Housawi, (2007) demonstrated CMLV specific antibody prevalence rate of 0%, 6% and 10% in cattle, sheep and goat respectively, in Saudi Arabia (Housawi, 2007). Based on these results, Duraffour et al., suggested the potential adaptation of camelpox in enzootic areas to hosts other than camel (Duraffour et al., 2011b). CMLV is host specific and isolated reports of some skin lesions in humans have been reported (Bera et al., 2011; Ramyar and Hessami, 1972). The movement of large animal herds facilitates the continued propagation of CMLV. Arthropod vector may also help in propagating the infection and the virus may get transmitted directly from infected camel by skin injuries or through inhalations (Anonymous, 2008; Bera et al., 2011). CMLV has been isolated from the camel tick-*Hyalomma dromedarii* harboring camelpox infection (Pfeffer et al., 1996). CMLV may get into the body secretions including milk and may survive in the dried scab for as long as four months. CMLV was confirmed in the skin of the camels through PCR and on the CAM of specific-pathogen-free (SPF) embryonated chicken eggs (ECE) even after one year of their previous infection (Yousif and Al-Naeem, 2012).

The incubation period of camelpox varies from 3 to 15 days (Anonymous, 2008; Moss, 2007). Depending upon the virus strain, the infection may range from a mild type of skin lesions to moderate and serious systemic infections (Wernery and Kaaden, 2002). Moreover, pregnant animals may abort and the animal dies due to secondary infections and septicaemia (Kriz, 1982). The morbidity rate depends upon the circulation of the virus in the herd (Wernery and Kaaden, 2002). The incidence of camelpox is more in the male camels as compared to the female counterpart while the mortality is higher in the young ones than in the adults (Kriz, 1982). The disease is often associated with poor nutrition (Bhanuprakash et al., 2010a). Besides other factors, the virus gets transmitted from infected to healthy camel through shared animal husbandry practices.

4. Virion and genome characteristics

The etiological agent of camelpox disease in camels is an epitheliotropic DNA virus (Salem et al., 2008). It is a member of the genus *Orthopoxvirus* (OPV) under subfamily *Chordopoxvirinae* and the family *Poxviridae* (Essbauer et al., 2009). The brick shaped virion is 265–295 nm in size and surrounded by an outer membrane studded with irregularly arranged tubular proteins (Moss, 2007). CMLV replicates in the cytoplasm and carries a large number of viral encoded enzymes, associated within the virion. CMLV is ether resistant and chloroform sensitive (Moss, 2007). CMLV remained unaffected between a pH range of 3–8.5 for one hour and resists heat at 56 °C for one hour; however, its infectivity completely disappeared at 70 °C after 30 min (Falluji et al., 1979). The genome of CMLV is composed of a single linear double-stranded DNA molecule of 205,719 bp and contains 211 putative genes (Afonso et al., 2002). The genome of CMLV consists of a central region bound by identical inverted terminal repeats of approximately 7 kbp. Although the genome of CMLV shares close structural and functional similarities with other OPVs, it contains unique region of about 3kbp encoding for three ORFs (CMLV185, CMLV186, CMLV187) which are absent in other OPVs (Afonso et al., 2002). The genes in the middle of the orthopoxviruses genome are conserved while towards either terminal are variable, which encodes proteins involved with host tropism, virulence or immunomodulation (Gubser and Smith, 2002). Based on nucleotide sequence analysis, the CMLV is most closely related to VARV. The CMLV-CMS ITR just like variola virus encodes proteins within 650 bp of its terminus (Massung et al., 1994; Shchelkunov et al., 1995, 2000). At nucleotide level, CMLV and VAR shares 96.6–98.6% identity. The DNA distance matrix also showed lower genetic distance between CMLV and VAR than between CMLV and vaccinia virus. The percentage amino acid identity of CMLV with other poxviruses showed that CMLV is more closely related to VAR than any other viruses. The camelpox genome is composed of 66.9% A + T and has a distinctive *Hind* III restriction map (Gubser and Smith, 2002). Till date about 45 serotypes have been reported for CMLV and three serotypes (CMLV1, CMLV2 and CMLV-Hyd 06) are more prevalent in Indian subcontinent while serotypes 19 and 16 are prevalent in the Middle East and Africa respectively. These strains have different physicochemical properties and manifest themselves differently in various cells and in embryonated chicken eggs (Duraffour et al., 2011b).