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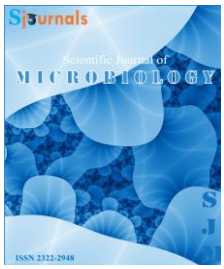

Review article

Epidemiology, zoonotic implication and diagnosis of camelpox: A comprehensive review

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ABSTRACT

Camelpox is an economically important, notifiable skin disease of camelids and could be used as a potential bio-warfare agent. The disease is caused by the camelpox virus, which belongs to the Orthopoxvirus genus of the *Poxviridae* family. Young calves and pregnant females are more susceptible. Tentative diagnosis of camelpox can be made based on clinical signs and pox lesion, but it may confuse with other viral diseases like contagious etyma and papillomatosis. Hence, specific, sensitive, rapid and cost-effective diagnostic techniques would be useful in identification, thereby early implementations of therapeutic and preventives measures to curb these diseases prevalence. Treatment is often directed to minimizing secondary infections by topical application or parenteral administration of broad-spectrum antibiotics and vitamins. The zoonotic importance of the disease should be further studied as humans today are highly susceptible to smallpox a very related and devastating virus eradicated from the globe. This review address an overview on the epidemiology, zoonotic impacts, diagnostic approaches and the preventive measures on camelpox.

1. Introduction

Camelpox is an economically important, contagious, often sporadic, and notifiable to Office Internationale des Epizootics (World organization for animal health-WOAH) skin disease of camelids (Elliot et al., 2008). The causative agent, camelpox virus (CMLV) is closely related to Variola virus (VARV), the causative agent of smallpox. Camelpox is confined to camel-rearing belts particularly in developing countries and causes economic impact due to considerable loss in terms of morbidity, mortality, loss of weight and reduction in milk yield in lactating ones (Azwai et al., 1996).

Transmission of camelpox occurs by direct contacts with sick animals through skin abrasions or via aerosols. Scab materials, saliva and secretions of affected camels may shed virus to the environment, such as water which becomes then the source of infection. Transmission of camelpox occurs by direct contacts with sick animals through skin abrasions or via aerosols (Wernery and Kaaden, 2002). Scab materials, saliva and secretions of affected camels may shed virus to the environment, such as water which becomes then the source of infection. Various studies have demonstrated that the incidence of camelpox outbreaks increases during rainy seasons with the appearance of more severe forms of the disease (Khalafalla, 2007). This may be due to the fact that moisture may enhance virus stability in the environment and increase subsequent transmission to susceptible animal. It could also be associated with the involvement of arthropods which are abundant during rainy seasons, which may serve as a mechanical vector of the virus. The latter idea is evidenced by the isolation of CMLV from Hyalomma dromedarii ticks (Wernery et al., 2000).

The disease is characterized by fever, enlarged lymph nodes and skin lesions. Skin lesions appear 1-3 days after the onset of fever, starting as erythematous macules, developing into papules and vesicles and later turning into pustules. These lesions first appear on the head, eyelids, nostrils and the margins of the ears. Later, skin lesions may extend to the neck, limbs, genitalia, mammary glands and perineum. In the generalized form, pox lesions may cover the entire body. 4-6 weeks to heal. In the systemic form, pox lesions are found associated with the mucous membranes of the mouth, respiratory and digestive tracts (Wernery and Kaaden, 2002).

Tentative diagnosis of camelpox can be made based on clinical signs and pox lesions, but will confuse with other viral diseases, such as contagious ecthyma (parapoxvirus) and papillomatosis (papillomavirus), therefore differential diagnosis is needed (Wernery and Kaaden, 2002). Five complementary techniques might be advised for camelpox diagnosis: transmission electron microscopy (TEM), cell culture isolation, standard PCR assays, immunohistochemistry and demonstration of neutralizing antibodies (Duraffour et al., 2011). Camelpox can be controlled or prevented by vaccination. Currently there are two types of vaccines, live attenuated and inactivated camelpox vaccines. A live attenuated vaccine gives longterm protection against camelpox. However, a booster vaccination is recommended for young animals vaccinated before the age of 6-9 months. When inactivated vaccine is used, the animals must be vaccinated annually (OIE, 2008).

Even though few studies have been conducted on camelpox in Ethiopia, the occurrence of the disease is frequently reported by field veterinarians and camel herders in different camel rearing areas of the country; indicating that it is one of the important infectious viral diseases of camel, the epidemiology of the disease is not well studied. Therefore, keeping in view the importance of disease, the current review is done which is helpful to provide updated epidemiological data and bases to adopt the control measures against the diseases in camels, therefore the objective of this review paper were:

- ✓ To compile literature and comprehend a note on camelpox epidemiology and public health significance;
- ✓ To highlight some possible approaches for camelpox diagnosis as a basis for designing further effective control strategies.

2. Etiological description

Camelpox virus (CMLV), the causative agent of camelpox, belongs to the genus *Orthopoxvirus* (OPV), of the subfamily *Chordopoxvirinae* of the family Poxviridae (Moss, 2007). The other members of the genus include several pathogens of veterinary and zoonotic importance. These are VARV, Monkeypox virus (MPXV), Vaccinia virus (VACV), Buffalopox virus (BPXV, a variant of VACV), Cowpox virus (CPXV), Ectromelia virus (ECTV), Rabbitpox virus (RPXV), The camelpox virus that causes camelpox is an Orthopoxvirus that is very closely related to the Vaccinia virus that causes smallpox. It is a large, brick-shaped, enveloped virus that ranges in size from 265-295 nm. The

viral genetic material is contained in double stranded linear Deoxyribo Nucleic Acid (DNA) consisting of 202,182 tightly packed base pairs. The DNA is encased in the viral core. Two lateral bodies are found outside the viral core, and are believed to hold the enzymes required for viral reproduction (Gubser et al., 2001). Camelpox viruses' ether resistant and chloroform sensitive. The virus is sensitive to pH 3-5 and pH 8.5-10. The virus can be destroyed by autoclaving, boiling for 10 minutes and is killed by ultraviolet rays (245 nm wave length) in a few minutes (Coetzer, 2004).

3. Epidemiology

3.1. Host range

Camelpox virus is a member of the genus Orthopoxvirus of the family *Poxviridae* (Afonso et al., 2002). In contrast to other OPV members, such as vaccinia virus (VACV), cowpox virus (CPXV) or monkey pox virus (MPXV), CMLV remains poorly studied, although it is, genetically, the closest virus related to variola virus (VARV) (Gubser and Smith, 2002). While other OPVs can infect various hosts, including rodents, zoo animals, monkeys and humans, VARV and CMLV are restricted to a single host, humans for VARV and camels for CMLV, in which they induce a severe disease. Old World (dromedary and Bactrian) camelids have been recognized as the reservoir hosts of CMLV, although New World camelids, such as guanacos, may be experimentally infected. And also, arthropod vectors involved in the transmission of the disease could be infected (Wernery and Zachariah, 1999).

In general, strains of CMLV have an extremely limited host range. Intra-dermal inoculation of the virus into sheep, goats, rabbits, guinea pigs, rat, hamsters and mice have not been successful (Bhanuprakash et al., 2010).

3.2. Mode of transmission

The incubation period varies from 4 to 15 days with an initial rise in temperature followed by papules on labia, vesicles, pustules, and finally formation of scabs (Balamurugan et al., 2009). During an outbreak of camelpox in United Arab Emirates in 1995-1996, twenty ticks were collected from five camels with generalized camelpox. Ticks, processed for electron microscopic and cell culture analyses, were found to contain CMLV. However, the question remains whether ticks might transmit CMLV mechanically or whether they might be a true reservoir of the virus. In the last case, the maintenance and spread of camelpox would be explained by transstadial transmission (the pathogen is maintained in the vector from one developmental stage to subsequent stages) or transovarial transmission (the female vector passes the infectious agent through her eggs to the next generation). Risk factors associated with higher incidence of camelpox have been defined and include the average age of the animals (less than four years old), the rainy season of the year, the introduction of new camels in a herd and the common watering. Transmission of camelpox occurs by direct contacts with sick animals through skin abrasions or via aerosols. Scab materials, saliva and secretions of affected camels may shed virus in the environment, such as in water which becomes then the source of infection (Khalafalla and Ali, 2007).

It is hypothesized that CMLV strains of different virulence may explain the differences in pathogenicity seen between dry and wet seasons, but this has never been assessed. Another possibility could be the involvement of arthropod populations which, abundant during rainy seasons, may exert a greater virus pressure onto camel populations. This idea is supported by the isolation of CMLV from *Hyalomma dromedarii* ticks (Wernery et al., 2000).

3.3. Morbidity rate and mortality rate

The morbidity rate of camelpox is variable and depends on whether the virus is circulating in the herd. Serological surveys taken in several countries reveal a high prevalence of antibodies to camelpox. The incidence of disease is higher in males than females, and the mortality rate is greater in young animals than in adults. Morbidity rate High percentage may reach to 100% in pregnant camel (33%). Mortality rate (5-25%) for adult and (25-100%) for young. The mortality rate in adult animals is between 5% and 28% and in young animals between 25% and 100% (Werney and Kaaden, 2002).

3.4. Geographical distribution of camelpox

Camelpox occurs in almost every country in which camel husbandry is practiced apart from the introduced dromedary camel in Australia and Tylopods (llama and related species) in South America. Outbreaks have been

reported in the Middle East (Bahrain, Iran, Iraq, Oman, Saudi Arabia, United Arab Emirates and Yemen), in Asia (Afghanistan and Pakistan), in Africa (Algeria, Egypt, Ethiopia, Kenya, Mauritania, Morocco, Niger, Somalia and Sudan) and in the southern parts of Russia and India. The disease is endemic in these countries and a pattern of sporadic outbreaks occurs with a rise in the seasonal incidence usually during the rainy season (Megerssa, 2010).

3.5. Clinical manifestation

The disease is characterized by an incubation period of 9-13 days with an initial rise in temperature, followed by enlarged lymph nodes, skin lesions and prostration. The clinical manifestation of camelpox varies from mild local to severe systemic disease depending on the CMLV strains involved in the infection (Wernery and Kaaden, 2002). The typical skin lesion/rash will pass through all the stages of pox lesions progression, i.e. development of papules on labia, macules, papules, pustules, vesicles and scabs. Skin lesions appear 1-3 days after the onset of fever with erythematous macules to papules and vesicles, and pustules and then crusts from ruptured pustules. In general, the lesion takes 4-6 weeks to heal. The lesion is usually localized in skin but occasionally, it leads to generalized form. The later form is frequently seen in young animals aged 2-3 years in a herd associated with weaning and poor nutrition. Eruptions are mainly localized on the head, nostrils, the margins of the ears and eyelids, as well as on the mucous membranes of the lips, the nose and also in the oral cavity. Later, lesions may extend to the neck, limbs, genitalia, mammary glands and perineum or scrotum (Duraffour et al., 2011). The animals may show salivation, systemic form of the disease. Pregnant females may abort. Death is usually due to septicemia caused by secondary bacterial infections such as *Staphylococcus auras* (Wernery and Kaaden, 2002).

3.6. Pathogenesis

The CMLV enters commonly through skin. However, the oro-nasal infection is also reported. After local replication and development of a primary skin lesion, the virus spreads to local lymph nodes leads to a leukocyte-associated viremia, which may be associated with pyrexia. During this period, virus can be isolated from various tissues, including the skin, turbinates, lungs and also lymphoid organs. Widespread secondary skin lesions appear a few days after the onset of viremia, and new lesions continue to appear for 2-3 days, at that time the viremia subsides (Bhanuprakash et al., 2010). The incubation period varies from 4 to 15 days with an initial rise in temperature followed by papules on labia, vesicles, pustules, and finally formation of scabs. Skin lesions appear 1-3 days after the onset of fever, starting as erythematous macules, developing into papules and vesicles, and later turning into pustules. Crusts develop on the ruptured pustules. These lesions first appear on the head, eyelids, nostrils and the margins of the ears. In severe cases, the whole head may be swollen. Later, skin lesions may extend to the neck, limbs, genitalia, mammary glands and perineum (Werney and Kaaden, 2002).

3.7. Diagnostic approaches

The presumptive diagnosis of camelpox infection can be made on the basis of clinical signs. However, infections of camels in the early clinical stages and in mild cases should be differentiated from contagious ecthyma (Orf), which is caused by a parapoxvirus (PPV), papillomavirus infections and insect bites. Para poxviruses and papillomaviruses have also been associated with skin infections of camel similar to camelpox lesions. Therefore, these infections cannot be differentiated solely on clinical signs. However, camelpox is diagnosed based on clinical signs, epizootiological and pathological findings, virus isolation, electron microscopy and genus-specific antigen capture Enzyme-Linked Immunosorbent Assay (ELISA) (Al-Ziabi et al., 2007). CMLV has also been detected by isolation and transmission electron microscopy (TEM) of the camel tick, *Hyalomma dromedary* (OIE, 2008).

3.7.1. Postmortem lesions

There is limited information on the pathology of camelpox. The lesions observed on postmortem examination of camels that die of severe camelpox infection are multiple pox-like lesions on the mucous membranes of the mouth, respiratory (mainly on the trachea and lungs) and digestive tracts. The size of the lesions in the lungs may vary between 0.5 and 1.3 cm in diameter and occasionally may reach up to 4-5 cm in diameter. Smaller lesions may have foci of hemorrhagic center on the surface of the lungs. In addition, infection of the heart and liver has also been observed in fatal forms of camelpox infection (Pfeffer et al., 1998).

3.7.2. Transmission electron microscopy

Transmission electron microscopy (TEM) is a rapid method to demonstrate OPXV in scabs or tissue samples. The laboratory confirmation of camelpox occurs through the demonstration of the characteristic brick-shaped orthopoxvirions in skin lesions, scabs or tissue samples. The virus is distinct from ovoid-shaped PPV, the etiological agent of camel contagious ecthyma (Orf). High concentration of virus in the sample is required for positive diagnosis and it is not possible to differentiate CMLV from other OPXV species. However, TEM is currently the best method for distinguishing clinical cases of camelpox and Orf caused by camelpox and PPVs, respectively, although the viruses can be differentiated by serological and Polymerase Chain Reaction (PCR) assays (Al-Ziabi et al., 2007).

3.7.3. Immunohistochemistry

Camelpox can be confirmed by immunohistochemical demonstration of the camelpox antigen in scabs and pox lesions in tissues. It is a fast method and can be used in lieu of TEM to establish a tentative diagnosis (Wernery and Kaaden, 2002). In addition, the paraffin-embedded samples can be stored for a long period, enabling future epidemiological and retrospective studies. Monoclonal and polyclonal antibodies can be used. However, almost any polyclonal antibody against VACV is likely to produce results in this test, as there is greater degree of similarity between VACV and CMLV (Nothelfer et al., 1995).

3.7.4. Virus isolation

Camelpox virus was first isolated by Buchnev and Sadykov (1969) in Russia. Since then, many countries have reported the isolation (Bhanuprakash et al., 2010). The most useful systems that can be considered for CMLV isolation are described below.

Embryonated Chicken Eggs: Camelpox virus can be isolated on the Chorioallantoic Membrane (CAM) of 11-13-day-old embryonated chicken eggs. The eggs should be incubated at 37^{o}C, and after 5 days characteristic dense, greyish white pock lesions are observed on the CAM. CMLV does not cause the death of inoculated embryonated chicken egg. The optimum temperature for the formation of pock lesions is 38.5^{o}C. If the eggs are incubated at 34.5^{o}C, the pocks are flatter and a hemorrhagic center may develop. Opaque white proliferative pock lesions of approximately 0.5-0.6 mm diameter were demonstrated on the CAM on the fifth day without any hemorrhagic lesions but with stunted growth. Characteristic long, opaque, white proliferative pock lesions have been produced when Vero cell-adapted virus was inoculate onto CAM (Marodam et al., 2006).

Cell Cultures: Various cell lines are susceptible to CMLV including HeLa, GMK-AH1, BSC-1, WISH and Vero. CMLV can also be propagated in MA-104 and MS monkey kidney and baby hamster kidney cells. Primary cell cultures such as lamb testis, lamb kidney, camel embryonic kidney, calf kidney and chicken embryo fibroblast can also be used (Tantawi et al., 1994). CMLV shows typical cytopathic effects on a wide variety of cell cultures. Intra-cytoplasmic eosinophilic inclusion bodies, characteristic of poxvirus infection, may be demonstrated in infected cells using hematoxylin and eosin staining. Poxviruses are epitheliotropic, and both VACV and CPXV have the ability to infect raft cultures of human keratinocytes. Interestingly, CMLV also has the ability to infect human keratinocytes even though the CMLV is restricted to camels. Unlike VACV or CPXV, which have wider host specificity, CMLV has also been shown to infect human embryonic lung fibroblasts (Duraffour et al., 2011). The characteristic cytopathic effect includes rounding, vacuolization, multinucleated giant cell formation, syncytia and cytolytic changes in Vero cells (Marodom et al., 2006).

3.7.5. Serological tests

Poxvirus antibodies can be detected in animal sera much more frequently than isolation of the virus from clinical samples. All viruses in the genus OPXV are serologically cross-reactive. PPVs and CMLV do not cross-react and so infections of camelpox and camel Orf can be distinguished serologically. Most of the conventional serological tests are time consuming and labor intensive. However, these could be used as an adjunct for confirmation and retrospective epidemiological studies in those areas where vaccination against camelpox is not practiced. A wide range of serological tests are available to identify camelpox. Conventional serological tests such as hem agglutination, hemagglutination inhibition, neutralization, complement fixation and fluorescent antibody tests have been used to detect CMLV antibodies (Al-Hendi et al., 1994).

A set of serological tests, including haemagglutination (HA), haemagglutination inhibition, virus neutralization test (VNT), indirect ELISA, complement fixation, and fluorescent antibody tests/assays are available for the

detection of antibody to CMLV (Balamurugan et al., 2013). VNT and ELISA are the most commonly used and sensitive tests. VNT test is based on a reaction between the virus and specific antibody in the test serum. Virus and products containing a neutralizing antibody were mixed under appropriate conditions and then inoculated into cell culture. The presence of unneutralized virus was detected by plaque formation (cytopathic effect). A loss of infectivity was caused by interference by the bound antibody with any of the steps leading to the release of the viral genome from the host cells including attachment, infection, or viral release. On the other hand, ELISA is developed for the detection of total IgG and IgM antibodies to camelpox virus in camel sera and for identifying the seroreactive antigens of the virus. It is a simple method which can successfully be applied for retrospective and also for epidemiological investigations. The test is more sensitive than virus neutralization (VNT) (Azwai et al., 1996).

3.8. Polymerase Chain Reaction (PCR)

Since the advent of PCR, insufficiencies in the quantity of DNA were no longer a limitation in diagnostic procedures (Viljoen et al., 2013) PCR method has also been adopted for the detection of camelpox virus DNA and it is rapid and sensitive method which can detect even a few copies of viral DNA from the clinical samples (Nagarajan et al., 2011). DNA can be extracted from cell culture samples and clinical material using numerous commercial kits. A reliable and low-cost two-step extraction procedure has been developed for isolating CMLV DNA from skin samples (Yousif et al., 2010). The PCR assays available to identify CMLV are based on the detection of different target genes using different specific oligonucleotides/primers. Regardless of the application of PCR for the diagnosis of CMLV with an excellent sensitivity and specificity, it is not commonly used for large scale epidemiological studies because it requires very expensive sophisticated equipment's and skilled personnel. Further, they are not suitable for on-spot detection at field situations or primitive clinical laboratories particularly in developing countries (Meyer, 1997).

3.9. Loop Mediated Isothermal Amplification (LAMP) assay

LAMP is a rapid, accurate, simple and cost-effective novel nucleic acid amplification method under isothermal conditions (60-65°C), with great potential application in developing countries for diagnosis without requiring sophisticated equipments and skilled personnel (Notomi et al., 2000). The LAMP since its first report by Notomi and his coworkers, it has been used widely for the diagnosis of various diseases (Dhama et al., 2014). LAMP assay based on the highly conserved region of ankyrin repeat protein gene (*C18L*), which is specific only for CMLV, has been developed for the diagnosis of CMLV and evaluated using field clinical samples. The amplicon size of the LAMP product is 198 bp. The amplified LAMP product can identified by agarose gel electrophoresis and subsequent direct visualization under UV light or observation by naked eye for the presence of turbidity and color change following the addition of Green I dye and hydroxynaphthol blue (HNB) (Meyer, 1997). This assay appears to be potential as rapid and sensitive diagnostic tool for its application in less equipped rural diagnostics laboratory settings in developing countries.

3.10. Antiviral therapy

Camelpox virus is one of the viruses closely related to VARV. Post-exposure treatment of camelpox infection has also not yet been described. Treatment of severe cases includes minimization of secondary infections by topical application or parenteral administration of broad-spectrum antibiotics and vitamins (Wernery and Kaaden, 2002). Alternative treatments include the use of antiviral agents such as cidofovir and ST-246. Cidofovir inhibits CMV and poxvirus DNA polymerase (Duraffour et al., 2011). It is effective against DNA viruses particularly papillomaviruses, polyomaviruses, adenoviruses, herpes viruses and poxviruses. Among the poxviruses, it is effective against VARV, VACV, CPXV, CMLV and MPXV. The antiviral activity is long-lasting owing to the long half-life of its metabolites. Some of the derivatives of cidofovir have oral bioavailability. In some cases, resistant viruses are difficult to treat with cidofovir but their virulence can be attenuated (Smee et al., 2002).

3.11. Control and prevention

Camelpox can be controlled or prevented by vaccination. Currently there are two types of vaccines, live attenuated and inactivated camelpox vaccines. A live attenuated vaccine gives long-term protection against camelpox. However, a booster vaccination is recommended for young animals vaccinated before the age of 6-9 months. When inactivated vaccine is used, the animals must be vaccinated annually (OIE, 2008). The ability to

confirm a clinical diagnosis of camelpox through rapid molecular testing would be of critical importance in efforts to control and eradicate the disease. Since camelpox affects only camel, its causative agent has no wildlife reservoir and availability of diagnostic tests and vaccines to diagnose the disease and block its transmission; camelpox meets the basic requirements to be a candidate for eradication (Aylward et al., 2000). The camelpox virus is sensitive to a number of common disinfectants. It can also be destroyed by autoclaving, short term exposure to Ultra Violet light, and boiling for at least 10 minutes. These methods may be used by camel herders to minimize risk of environmental contamination just like smallpox in humans; the disease could be eliminated by isolating sick camels and vaccinating the rest, using either the traditional vaccinia virus vaccine or a more recently developed camelpox virus vaccine (Bray and Babiuk, 2011).

4. Zoonotic implication

Human cases of camelpox have been described as rare or inexistent. Indeed, few articles reported individuals with lesions on the arms, or ulcers on the lips and in the mouth (from drinking milk of infected animals), but they all remained unconfirmed. However, recently, camelpox has been described as a possible zoonosis with three human cases identified and laboratory confirmed in India (Bera et al., 2010). These camel handlers, in direct contact with camelpox-infected animals, developed skin lesions localized on the fingers and the hands. Identification of CMLV as the causative agent was made (i) based on the detection of camelpox neutralizing antibodies in serum samples of the three suspected cases, (ii) by amplification of a CMLV specific gene (*C18L*), and (iii) by further amplification and sequencing of other genes whose sequences were confirmed to match those of CMLV. These findings should be taken into consideration in the actual context of increasing number of OPV infections in humans and animals. However, camelpox appears largely restricted to camels, and seldom produces clinical disease in humans. Reports of human camelpox cases either confirmed or not by virological tests, have suggested a mild course of disease even though CMLV is genetically more closely related to VARV than to other OPVs (Al-Ziabi et al., 2007). Mild skin lesions in humans associated with camelpox have been reported by (Coetzer, 2004), indicating camelpox may be of public health impact. Among the human cases, people drinking milk from camelpox-affected animals have been reported to develop ulcers on the lips and in the mouth, but these observations could not be visualised or laboratory confirmed (Yousif et al., 2010). However, under certain conditions, the virus could be pathogenic for human like that of cowpox and monkeypox especially in immune-compromised individuals (Marennikova, 1975). However, no systematic epidemiological studies have been undertaken on human cases due to the lack of immunological surveys for specific camelpox antibodies among unvaccinated herds (Azwai et al., 1996). Discontinuation of smallpox vaccination has rendered the present day human population more vulnerable to smallpox and other related infections. There is a great threat of variola virus being used as a biological warfare or bioterrorist agent (De Clercq, 2001).

5. Status of camelpox in Ethiopia

Camelpox is one of the most important infectious diseases of camels in Ethiopia. The disease has been reported from Oromia, Afar, Somali, and Amhara regional states. In Ethiopia, a clinical prevalence ranging from 0.45% to 14.2% has been reported in different parts of the country (Ayelet et al., 2013). Similarly Tefera and Gebreah (2001) indicated that camelpox prevalence based on clinical cases as it was 3%, 14.2% in Dire Dawa, Harar Zuria, Jijiga, Gewane. Most of these studies have reported that the disease commonly affects young immature camels and the incidence is higher during the rainy season.

Recently, the national veterinary institute has developed a new attenuated vaccine which provides a protection for a year. Currently, about one million dose of the vaccine is distributed to major camel producing parts of the country, including Somali, Borena and Afar (Ayelet et al., 2013). In Borena (the southernmost zone of Oromia region), based on clinical diagnosis, the prevalence of camelpox was 0% during dry season, 0.3% during major wet season, and 14.2% during minor wet season (Megersa, 2010). Recently, studies have indicated that the individual level prevalence of camelpoxvia serological test as reported by (Weldegebrial, 2015) was 21.6 and 16.7% at Amibara and Awash FentaleAfar region, North Eastern Ethiopia.

6. Conclusion

Camelpox is an economically important contagious skin disease of camelids caused by camelpox virus and is characterized by mild local skin infection and less common severe systemic infections. The disease is confined to camel-rearing belts particularly in developing countries like Ethiopia and causes economic impact due to considerable loss in terms of morbidity, mortality, loss of weight and reduction in milk yield. Recently, camelpox has been described as a possible zoonosis with three human cases identified and laboratory confirmed in India. Although the disease can be diagnosed based on clinical signs, the similar confounding skin lesions necessitate identification of infection by molecular biology based diagnostic techniques. Both inactivated and live attenuated vaccines are available in some countries. However, live vaccines are preferred as they provide long-lasting immunity. Considering the emerging nature of the virus and the economic impact caused in the camel industry and pastoralists, the control and eradication of the disease from camel-rearing countries is of paramount importance. Based on the above conclusion the following recommendations are forwarded:

- ✓ Regular vaccination of camels particularly the young ones which are very susceptible to the disease and reducing the predisposing factors are important to reduce the impact of the disease in endemic areas.
- ✓ Awareness of camel herders should be raised as to the zoonotic importance and means of transmission of the disease.
- ✓ Researches that help in deepening the understanding of epidemiology of the disease and developing easy, rapid, cheap and accurate diagnostic technique that can be used at field condition is needed for earlier preventive and control responses.
- ✓ Further study on zoonotic aspect of this disease is highly encouraged as camelpox virus behaves very similarly to the virus that causes small pox, a dreaded disease.

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