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Invited review: Camelids zoonotic diseases

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Abstract

Zoonotic infections or diseases can be transmitted naturally to humans with or without arthropod intermediates. Indeed, some of these diseases can even get transmitted from camels to humans. This review intends to focus on zoonotic diseases of camelids and measures their control. Since infected animals rarely appear sick, humans frequently become exposed to and develop severe illnesses from the bacteria, protozoa, fungi, viruses, and parasites of camelid origin. People with chronic illness, or immunodeficiency, and pregnant women may be at higher risk of developing disease or complications from a zoonotic disease and should avoid contact with these animals. Zoonotic diseases associated with camelids are divided into three groups: (i) significant diseases, (ii) diseases of which Camelids are potential pathogen carriers, and (iii) minor or non-significant diseases. Therefore, anyone working with or handling camelids should be aware of the potential zoonotic threat, and precautions must be taken to minimize their risk of becoming infected.

Keywords: Camelpox, MERS-CoV, tuberculosis, ringworm, zoonotic diseases**.**

1. Introduction

Camels are humped, long-necked, even-toed ungulates, and belong to the genus *Camelus* and family *Camelidae*. They are known for their endurance and longevity, the special chemical and physical properties of their milk (Abu-Lehia, 1989), the low cholesterol content of their meat, and the quality of their wool and skin. According to FAO statistics 2008, at the global level, there are over 19 million camels, of which four million are found in Asia and 15 million in Africa. Camels are important animals to pastoralists because their ability to survive in the harsh desert environment and their high potential to transform the scanty resources of the desert into milk and meat (Gebreyohanes and Assen, 2017; Knoess, 1984; Abbas and Tilley, 1990; Schwartz, 1992). According to taxonomy and physiology or behaviour, camels are not ruminants and have a three-compartment complex stomach (Fowler, 2010). Besides, gastric digestion is alike, but different from digestion in ruminant.

Commonly, camelids are reared in the arid desert environments. Due to the harshness of the desert environment, particularly during the extended dry seasons, camels deal with severe stress situations, which in turn make them

prone to various diseases and illnesses (Abbas and Agab, 2002; Agab, 1993). In the past, and due to the scarceness of the studies that cover camel diseases, some scientists believed camels to be naturally resistant to many disease-causing pathogens and factors (Zaki, 1948; Dalling *et al.,* 1988). However, camels have been confirmed to be similarly vulnerable to the common disease-causing pathogens that affect other animal species (Wilson, 1984; Abbas and Tilley, 1990; Abbas and Agab, 2002). Some of these diseases can be communicated from camels to humans, as they are zoonotic by nature.

Moreover, although infected camels may appear healthy and exhibit no clinical signs, these diseases can cause serious illness and complications in humans, especially during pregnancy or in people suffering from immunodeficiency and chronic illness. An extensive search for publications and websites revealed few documented reports concerning zoonotic diseases of camelid origin. In addition, no systematic literature review regarding zoonotic diseases in camelids was found. Therefore, this review intends to provide an overview of the zoonotic diseases of camelids, particularly with regard to their epidemiology, pathogenesis, biology, diagnostic approaches, and control measurements.

2. Classification and importance of camelids zoonotic diseases

Several unexplained diseases with over mortalities have occurred in the last ten years, indicating an emerging disease in camelid populations. However, little is known about the pathogens circulating in camel populations or their interaction mechanism with the camel. Furthermore, validation of only very few diagnostic tests for use in camels has been carried out, and yet the response of these animals towards vaccines is not fully understood. Likewise, camels to be a source of the human disease 'Middle East Respiratory Syndrome' (MERS). The queries related to the validity of antibody tests for MERS in camels have highlighted the need to better understand disease dynamics in these animals. The most vital challenge in the raising of camel herds is the associated zoonotic disease. Typically, camelids zoonotic diseases are divided into three groups: (a) significant diseases, (b) diseases of which Camelids are potential pathogen carriers, and (c) minor or non-significant diseases.

2.1 Significant diseases

2.1.1 Camelpox

Since 2010, camelpox has been considered, an emerging public health issue due to increased reported cases and epidemics in camels. Camelpox is a contagious, often sporadic, and notifiable skin disease of camelids and it is socio-economically significant as it incurs a considerable loss regarding morbidity, mortality, loss of weight, and reduction in milk yield. It is mostly confined to camel rearing countries (Duraffour *et al.,* 2011).

The camelpox virus (CMLV) is a predominantly host specific zoonotic agent (Davies *et al.,* 1975), although indications and evidence have been released from Somalia and India of its presence in smallpox unvaccinated persons and camel handlers or attendants respectively (Jezek *et al.,* 1983; Kriz *et al.,* 1982; Bera *et al.,* 2011). Camelpox, which presents with mild skin lesions (Coetzer *et al.,* 2004), may have a public health impact. Consumption of milk from camelpox-affected animals, appears to have led to the formation of ulcers on the lips and in the mouth of individuals drinking it, though no laboratory confirmation has been done (Davies *et al.,* 1975). Meanwhile,

on certain occasions, CMLV could be pathogenic, particularly in an immune-comprised individual.

Because of a lack of precise immunological analyses of camelpox antibodies between unvaccinated herds (Marennikova, 1975), no systematic epidemiological studies have been carried out in humans (Azwai *et al.,* 1996). However, the self-limiting nature of the camelpox virus towards humans (Duraffour *et al.,* 2011), can be associated by the previous administration of the smallpox vaccination. The first definite proof of camelpox zoonotic infections in unvaccinated smallpox humans in connection with dromedary camel epidemics was reported in India by Bera *et al.,* (2011), who were first to confirm the zoonotic nature of camelpox through laboratory investigations. They described three human cases that presenting with papules, vesicles, ulceration, and finally scabs over the fingers and hands (Figure 1). Molecular characterization of the causative agent accompanied with clinical, epidemiological, and serological tests was the basis for their confirmation of CMLV zoonosis in human cases.

Khalafalla and Abdelazim (2017) also reported the zoonotic nature of CMLV in Sudan. They described a camelpox outbreak on two camel herds in eastern Sudan followed by infection in humans, specifically in three male owners who were in direct contact with the infected camels. Their analysis of epidemiological data and CMLV genomic sequences seems to suggest possible zoonotic transmission of CMLV from camels to people. However, there was no approval of the human-to human spread. Further, the confirmation of camelpox in humans during these outbreaks indicates that, since CMLV will not only infect camels, the host range of the virus should also include humans.

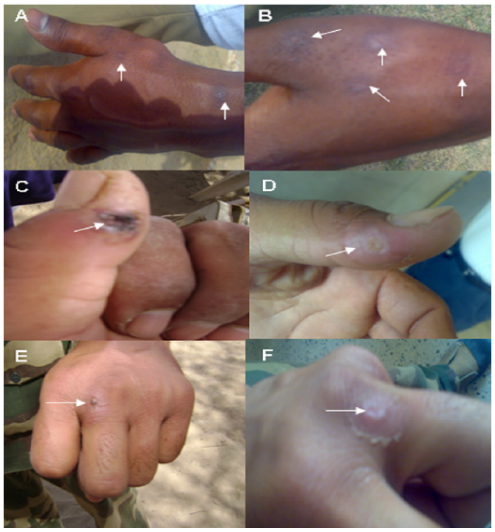

Figure 1. Skin lesions of camelpox in human cases. Case 1: (A&B) revealed disseminated cured scabs over the hand. Case 2: (C&D) Pock lesion displayed as an ulcerated open wound with central necrosis surrounded by a sharp haemorrhagic edge on the thumb. Case 3: (E&F) Typical pock-like lesions appearing as an eruption at the base of the middle finger (Bera *et al.*, 2011).

Camelpox is one of the diseases notifiable to the OIE epizootics (World organization for animal health-WOAH). CALV is strictly related to the Variola virus, the smallpox causative agent. Although the disease is restricted to camels, it is enzootic in almost every region, where camels are being raised. except from Australia. The virus belongs to the genus Orthopoxvirus (OPV), of the subfamily Chordopoxvirinae of the family Poxoviridae. Numerous CMLV strains have been isolated, from different epidemics in different parts of the camel rearing countries.

During the smallpox eradication campaign in the early 1970s, the identification of the CMLV agent caused some alarm because of its designation as a smallpox-like disease (Baxby, 1972). The CMLV genome contains a single linear double-stranded DNA molecule ended by a hairpin loop that replicates in the cytoplasm. The virus carrying genes are responsible for host immune evasion mechanisms owing to the threat posed by potential bio-warfare agents. Camelpox is a contagious disease of the *Camelus dromedarius* and *Camelus bactrianus* (Old-World camelids) and the new-world camelids (Elliot *et al.,* 2008), although it naturally infects merely the Old-World camelids. It occurs in camel breeding areas of Asia, the Middle East, Africa and north of the equator. The disease is endemic in the Middle Eastern countries [Iran, Iraq, King Saudi Arabia, United Arab Emirates, Oman, Yemen and, recently Syria (Al-Ziabi *et al.,* 2007)], Africa (Sudan, Algeria, Egypt, Kenya, Mauritania, Niger, Somalia, Morocco and Ethiopia) and in Asia (India, Pakistan and Afghanistan).

CMLV transmission occurs by indirect contact via a contaminated environment or directly between infected and susceptible animals, either by inhalation or through skin abrasion. However, the mechanical transmission mechanism may also play a role in the transmission of the virus, as infected camels may shed the virus through scab materials, milk, saliva, ocular, and nasal discharges. There is also suspicion about the role of an arthropod vector in the transmission of the disease, as the spreading of tick populations, especially *Hyalomma dromedarii* (the most predominant species during the rainy season), seems to be linked to the spread of the CMLV.

The incubation period of the disease ranges from 9-13 days, and it is followed by enlarged lymph nodes, skin lesions, and prostration. Variations in the clinical signs of camelpox range from mild local to severe systemic disease, depending on the CMLV strains involved in the infection. The typical lesion is a rash that undergoes all the stages of pox lesions development, including as papules on labia, macules, papules, pustules, vesicles, and scabs. Conversely, the generalized form lesions may spread over the body, particularly the head and limbs, with occasional swelling of the neck and abdomen and even the appearance of multiple pox-like lesions on the mucous membrane of the mouth, respiratory and digestive tracts. When these are detected, the outcome is most likely fatal (Figure 2).

Infected camels may show salivation, anorexia, lacrimation, a mucopurulent nasal discharge, and diarrhea. Abortion may occur in pregnant animals due to septicaemia caused by a secondary bacterial infection such as *Staphylococcus aureus* (Wernery and Kaaden, 2002). The diagnosis of camelpox infection depends on the clinical signs found in affected animals. Tissue samples from the skin lesions or organ biopsies are most useful in recognizing the infectious agent. For this purpose, the utilization of different diagnostic approaches for making a confirmatory diagnosis is imperative. For

camelpox diagnosis, the use of several complementary tools such as transmission electron microscope, virus isolation using cell culture, standard PCR assays, immune-histochemistry, and demonstration of neutralizing antibodies, might be recommended (Bhanuprakash *et al.,* 2010).

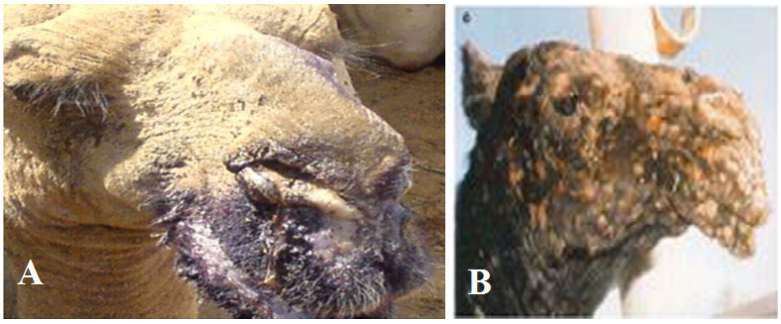

Figure 2. Clinical signs of camelpox A. Nasal discharge, B. Severe skin lesions on the head and face of the infected camel.

There is very little information regarding Camelpox treatment; however, the administration of antibiotics and supportive medication is essential for reducing the severity of the disease. The use of antiviral drugs/agents may also be of choice as an alternative treatment, especially in young camels. Similar to Variola, CMLV depends on a single host, which in turn indicates that the disease could potentially be eliminated through the combination of surveillance, vaccination, and quarantine (Duraffour *et al.,* 2011). Moreover, to curb the spread of camelpox in enzootic countries, prophylactic methods have been developed. A camelpox virus vaccine was developed in the former Soviet Union (Borisovich, 1973), and the little information available on vaccination efficacy originates from field investigation using commercialized CMLV-based vaccines. Traditionally, Bedouins in Arab countries use lactotherapy as a vaccination method for uninfected camels during an outbreak. This method involves a mixture of milk and scarification of camelpox infected crust. However, some countries, like Saudi Arabia, are administering an attenuated prepared vaccine. The intradermal or subcutaneous injection of the passage level 78 of CMLV (Jouf -78strain) propagated in camel kidney cell cultures has been found to be safe and effective at 10 3 TCID50 (Hafez, 1992).

Globally, camelpox is considered to be a severe zoonotic disease. As a result, the application of skills, knowledge, and veterinary public health resources required to protect public health from pathogenic zoonotic infections. Besides, the demand~~s~~ for control procedures for emerging and re-emerging pathogens is increasing with an increase in the population. The application~~s~~ of new molecular genomic and proteomic tools, apart from the traditional diagnostic techniques is also needed in the identification of the CMLV. Therefore, prophylactic, therapeutic, and prevention processes should be applied well in advance.

2.1.2 Rabies in camels

Rabies is an acute and deadly viral infection. This encephalitic disease is widespread in many areas of the world and affects camelids as it does all other mammals ~~are~~. The rabies virus

belongs to the Rhabdoviridae family, which includes the genera *Lyssavirus* and *Vesiculovirus*. This virus has been studied widely because of its zoonotic feature and its high mortality rate following the appearance of clinical signs (Wernery and Kaaden, 2002; Jackson, 2003).

Generally, the rabies virus spreads through bites and mucous membrane released from an infected rabid animal. The virus has been detected~~,~~ in saliva and other body excretions, but it cannot enter the non-injured skin. Rabies has been observed~~,~~ in camels in many African and Asian countries (Richard, 1980), including ~~such as~~ Morocco (Chevrier, 1959), Somalia (Arush, 1982), Niger (Bloch and Diallo,1995), Mauritania (Bah *et al.,* 1981), Oman (Ata *et al.,* 1993; Body *et al.,* 2015), the UAE (Wernery and Kumar, 1993), and Iraq.

Domestic animals can get infected via interaction with reservoir hosts in a particular geographic location, wildlife such as bats, skunks, and raccoons. In the USA, rabies is controlled in dog and cats by vaccination, but wild species serve as a reservoir. In Peru, from a herd of 160 heads, twenty alpacas were bitten by a rabid dog, and thirteen of these animals died after a short incubation period of six to eight days from the progress of clinical signs (Franco, 1968). Rabies has been documented in dromedary camels of all dromedary raising countries. The reservoir and transmitting hosts, while not always known, are assumed to be dogs and red foxes (*Vulpes vulpes*) in the UAE (Wernery and Kumar,1993).

Affected camelids often display neurological symptoms and unusual behaviour. Lameness, ataxia, and posterior paresis are the initial signs of rabies in camelids, followed by either an aggressive syndrome (furious rabies) or a paralytic syndrome (dumb rabies) (Peck, 1966; Higgins, 1986; Fowler, 2010 & 1998). In the aggressive form, no fever usually occurs until the animal becomes aggressive, which leads to an increase in the muscular activity. Aggression in this form manifests as attacks on people, offspring, housemates and objects. Other indications of this stage are vocalization changes, including alarm cries without cause. The characteristic features of terminal stages of rabies in camels are yawning (Figure 3). Other signs include bloating, pruritus, muscle tremors, aimless running, and sexual hyperactivity, and, later on, recumbence, convulsions, coma, and death that occurs within four days.

The characteristics of camelids paralytic rabies are anorexia, depression, ear droop, ptosis, tenesmus, salivation, circling, facial paralysis, mild fever, flaccidity of face, anus and bladder muscles, and pharyngeal / laryngeal paralysis (Afzal et al., 1993; Kumar and Jindal, 1997).

Figure 3. The terminal stages of rabies in camel: prior to death, the dromedary attempts to yawn continuously.

It is vital to make an accurate diagnosis of camelids' rabies to enable the authorities to enforce a preventive procedure. The rabies virus causes a non-suppurative encephalitis with perivascular cuffing by mononuclear cells. The presence of Negri bodies can be confirmed using immunofluorescence, a characteristic of rabies in camelids (Wernery and Kaaden, 2002) (Figure 4). A very large number of rabies virus particles of varying sizes were observed in the brains of all examined rabid dromedaries. Active immunization is possible with inactivated vaccines, with data showing that one cattle dose of inactivated rabies vaccine induces satisfactory but short-term serological conversion in dromedary camels. A booster dose of vaccine 6 to 8 months after primary vaccination is, therefore, necessary.

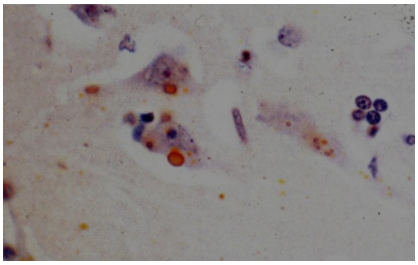

Figure 4. *Negri bodies confirmed by immunofluorescence (Wernery and Kaaden, 2002).*

2.2 Diseases for which camelids are potential pathogen carriers

2.2.1 Middle east respiratory syndrome coronavirus (MERS-CoV)

The novel Middle East Respiratory Syndrome coronavirus (MERS-CoV) was first recognised as the cause of respiratory infections in Saudi Arabia in 2012, and approximately 330 of those cases proved to be fatal (Crameri *et al.,* 2015; Gossner *et al.,* 2016). Coronaviruses are a large family of viruses that can cause diseases ranging from the common cold to severe acute respiratory syndrome (SARS) (Zaki *et al.,* 2012). Typical MERS symptoms in humans vary from mild respiratory symptoms to severe acute disease and death. The typical presentation of MERS-CoV starts with fever, coughing, abnormal rapidity of breathing, and gastrointestinal symptoms including diarrhea (Zaki *et al.,* 2012). Pneumonia is most common, but not always present. Respiratory failure occurs with severely affected patients and it requires hospitalization in ICU (intensive care units) and mechanical ventilation. Older people, immunosuppressed individuals, people suffering from chronic diseases including chronic lung diseases and diabetes are more susceptible to the virus and the development of more serious symptoms. . Further, some researchers have proven that cases of MERS-CoV infection may appear as asymptomatic. The majority of these asymptomatic cases have been determined following aggressive contact tracing of a laboratory-confirmed case. About 35% of recorded cases of patients affected by MERS have died.

Even though the majority of human cases of MERS have contributed to human-to-human infections in healthcare settings, current scientific evidence suggests that dromedary camels are the primary reservoir host for MERS-CoV and an animal source of MERS infection in humans (Crameri *et al.,* 2015; Gossner *et al.,* 2016). Although MERS-CoV is a zoonotic virus and some studies reveal that infected dromedaries infect people via direct or indirect contact (Gossner *et al.,* 2016), the exact role of

dromedaries in the spread of the virus and the exact route(s) of spreading are unknown. The virus does not seem to pass easily from person to person except if there is close contact with the affected person like when providing care for an undiagnosed patient. It is worth mentioning that healthcare-related occurrences have happened in several countries, and a high epidemic has been documented in Saudi Arabia, the United Arab Emirates, and Korea (Crameri *et al.,* 2015).

The virus of the disease has been identified in several camel-rearing countries, including Saudi Arabia, Qatar, Oman, and Egypt. Moreover, MERS-CoV specific antibodies have been found in dromedaries in the Middle East, Africa, and South Asia, which indicates that the camels had been previously either exposed or infected by the virus (Figure 5).

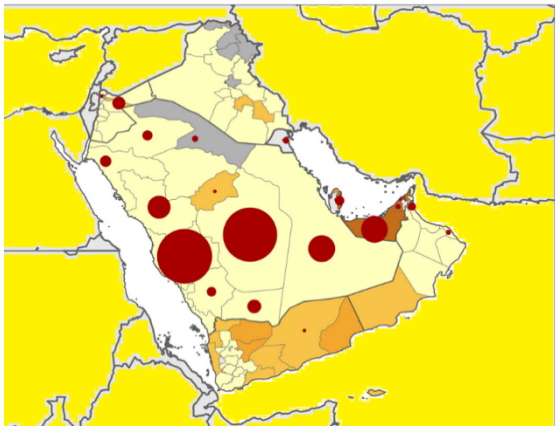

Figure 5. Distribution of the Camelidae in Middle East and MERS-CoV human cases from 2 March 2012 to 23 July 2014 (n =695) Showing cases for which the probable region of infection is available. The map was created using data from: World Health Organization for Animal Health. World Animal Health Information Database (WAHID), Animal population, Camelidae, 2011–2013 (Available from: *http://www.oie.int/wahis_2/public/wahid.php/Countryinformation/Animalpopulation). ECDC line listing: data compiled from WHO and Ministries of Health websites around the world.*

Indeed, the source of the virus is not fully understood. Nonetheless, according to the analysis of different virus genomes, it is assumed that it may have originated in bats, which was further transmitted to camelids a long time ago. However, evidence remains weak and unconvincing (Ithete *et al.,* 2013; Memish *et al.,* 2013).

In dromedaries, the MERS-CoV infection is either asymptomatic or causes mild respiratory symptoms (Hemida *et al.,* 2014 A; Hemida *et al.,* 2014 B; Nowotny and Kolodziejek, 2014), which would indicate that epidemics in camel herds are likely to go undiagnosed. Previous studies carried out in Jordan and Saudi Arabia on sheep, goats, and cattle were unable to corroborate a previous infection (Reusken *et al.,* 2013; Alagaili *et al.,* 2014). Moreover, in the United Arab Emirates, the examination of stored sera collected in 2005 also revealed a negative output for MERS-CoV antibodies (Alexandersen *et al.,* 2014). In contrast, serological investigations, in Jordan, Oman, Qatar, Saudi Arabia, and UAE revealed a high antibodies rate against MERS-CoV (Reusken *et al.,* 2013; Alagaili *et al.,* 2014; Meyer *et al.,* 2014), thereby

pointing to wide-spread distribution of the virus in the Arabian Peninsula. Egypt, Ethiopia, Kenya, Nigeria, Sudan, South Sudan, Tunisia, and the Canary Islands have also revealed a titer of antibodies against MERS-CoV in dromedaries camelids (Perera *et al.,* 2013; Reusken *et al.,* 2013; Corman *et al.,* 2014). It has also been proved that as early as 1992, MERS-CoV was spreading in camels in Saudi Arabia (Alagaili *et al.,* 2014), and in the year 2003 it reached to UAE (Meyer *et al.,* 2014), while the new MERS-CoV ancestor was a sample from humans in 2011 (Rambaut, 2013). Additionally, to manage the zoonotic implications of MERS-CoV from dromedary camels, control measures were implemented in the Arabian Peninsula. Ultimately, these processes appear to be efficient and successful, as the number of reported cases has decreased significantly.

2.2.2 Rift valley fever (RVF) in dromedary camels

Rift valley fever (RVF) is an acute, arthropod-borne fever-causing viral disease affecting domestic animals such as cattle, buffalo, sheep, goats, and camels. This zoonotic disease can also affect humans in contact with contaminated items (Hoogstraal, 1979), causing haemorrhagic fever, encephalitis, blindness, and severe liver damage. RVF outbreaks cause severe economic damage to animal owners due to lack of milk production and fatalities, which is intensified by the 100% abortion rate at all stages of pregnancy. The weather seems to play a crucial role in the development of RVF epidemics; all outbreaks were reported to occur after heavy rainy seasons, which would generate a steep increase in insect population, a vector requirement (Huebschle, 1983). The occurrence of RVF in very arid areas has not been reported.

The RVF virus belongs to the *Phlebovirus* genus of the family *Bunyaviridae*. It has a spherein shape, with a diameter of 80 to 120 nm, and possesses a host cell-derived bilipid layer envelope where virus-coded glycoprotein spikes project. RVF virus strains have revealed no significant antigenic differences. However, demonstration of variations in virulence, have been carried out. The infection originated in eastern and southern Africa and went endemic in indigenous forests because of the availability of mosquito vectors after heavy rains. The first description of this disease was found among livestock in the Rift Valley in Kenya in the early 1900s (Scott *et al.,* 1963). Subsequently, Imam *et al.,* (1978), Eisa (1981), Slama, (1984), Davies *et al.,* (1985), Saluzzo *et al*., (1987), and Olaleye *et al.,* (1996) reported the disease in Egypt, Sudan, Tunisia, Kenya, Mauritania, and Nigeria, respectively.

RVF antibodies have been found in 45% of the examined dromedaries during epizootics, with a drastic increase in abortions (Scott *et al.,* 1963); the high abortion rate was also reported in in Egypt). Recently, an unusual RVF outbreak with high mortality rates and severe clinical signs typically observed among dromedaries was reported in Mauritania, at a northern latitude and in an extremely arid region (Figure 6, El Mamy *et al.,* 2011). According to the WHO, a high mortality rate in camels during this RVF outbreaks was reported.

Camelpox or parapox (Ecthyma contagiosum) has been suggested as the cause of high mortality and morbidity. Clinical symptoms typically include ballooning of head and upper neck, swollen eyes and giant mucoid membrane sloughs in the mouth covering some ulcers. Ultimately, all RVF outbreaks in camels are characterized by signs such as (i) fever, (ii) abortion and (iii) sometimes early neonatal death and jaundice.

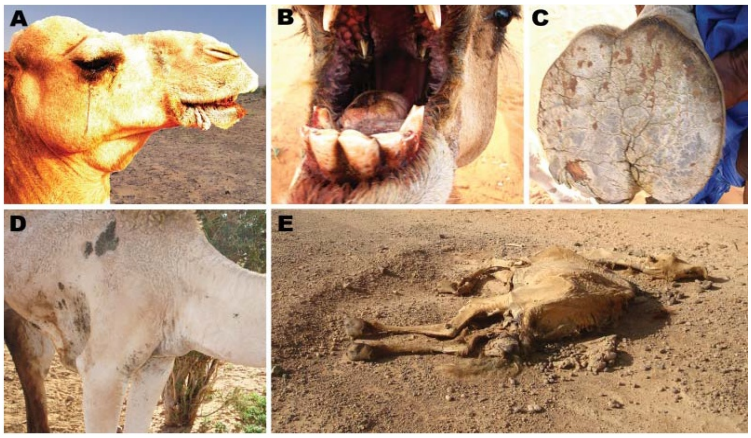

Figure 6. Clinical signs of RVF outbreaks in camels in Mauritania. A. Conjunctivitis, B. Haemorrhage of Gums, C. Foot lesions (cracks in the sole) with secondary myasis, D. Oedema at the base of the neck, E. Dead camel with signs of abortion, convulsions and arching of the neck (El Mamy et al., 2011).

The definitive diagnosis of RVF relies on virological and serological investigations. Specimens for laboratory confirmation include heparinised blood, liver, spleen, kidney, lymph nodes, and brain from aborted foetuses which are collected for virus isolation on Vero and BHK21 cells or suckling and weaned mice. Complement fixation test, Agar gel diffusion test, and ELISA can be used to detect RVF antibodies. Immunofluorescence on impression smears from infected tissue can also be used. Immunization of susceptible animals is the best and most effective method to protect livestock against RVF, since chemical control of vectors is not a practical method (El Mamy *et al.,* 2011).

2.3 Minor or non-significant diseases

2.3.1 Brucellosis

Brucellosis, a global infectious zoonotic disease affecting animals including camels, as well as humans, is typically caused via Gram-negative bacteria of genus Brucella that are facultative intracellular. These bacteria can persist in the host cell causing a chronic disease and may stay throughout the lifetime of the infected animal. Brucellosis is considered to be a highly severe disease as it can be transmitted to people, thereby exerting an effect on public health (Al-Salihi, 2013). Even though the infection has got special consideration from researchers, scientists, and governments, it has not been well investigated in camels to date.

Camel brucellosis was reported for the first time in 1931 (Solonitsuin, 1949), and it was subsequently detected in all camel-keeping countries (Gwida *et al.,* 2012). Various factors make camels prone to brucellosis, the most crucial one being the raising of camels with another species of animals like sheep and goats. Although camels are not primary hosts of Brucella, camelids are susceptible to both *Brucella abortus* and *Brucella melitensis* (Cooper, 1991). Subsequently, the incidence is influenced by the infection rate in primary hosts being in contact with them.

The mishandling of isolated species of Brucella from camels, drinking of milk, and consuming meat has led to a high number of human brucellosis cases, arousing acute public health problems (Kiel and Khan, 1989). The greatest farmers from nomadic areas have a belief that camel milk is a healer for various

diseases. Thus, they drink raw camel milk and completely neglect the disease-causing effect of the non-pasteurized milk. In Mogadishu/ Somalia, researchers found the existence of camel brucellosis using various serological tests, in addition to mRBPT which was as sensitive as SAT and c ELISA. Moreover, they found that RBPT is a highly sensitive test and further validated its antigen consistent for bovine brucellosis (Kadle *et al.,* 2017). Very little research has been published concerning camel brucellosis in Iraq (Al-Ani *et al.,* 1998).

Additionally, in a serological study using the Rose Bengal test on 104 serum samples collected from different age groups, the percentage of camels testing positive for brucellosis was 6.73 % (Al-Rodhan *et al.,* 2006).

Several complications appear in the diagnosis of camel brucellosis, as this disease displays only insufficient clinical signs in parallel to its clinical appearance in cattle (Mousa *et al.,* 1987). Moreover, camel herds are usually reared in remote areas, where the proper infrastructure required for veterinary care and support is missing. Therefore, in all camel raising countries, Brucellosis needs more attention and research and, for effective planning of a vaccination program, the isolation and identification of the causative agents in camels are imperative. Additionally, educational programs and brochures to make Bedouins aware of the risks of brucellosis should reduce the human infection percentage.

2.3.2 Tuberculosis

Tuberculosis (TB) is a contagious, granulomatous chronic disease caused by a mycobacterial species in the Mycobacterium tuberculosis complex (MTC) (Thoen *et al.,* 2006). *M. tuberculosis*, *M. bovis*, *M. caprae,* and *M. microti* have been isolated in camelids (Barlow *et al.,* 1999; Bush *et al.,* 1990; Dinkla *et al.,* 1991; Elmossalami *et al.,* 1971; García-Bocanegra *et al.,* 2010; Lyashchenko *et al.,* 2007; Lyashchenko *et al.,* 2000; Ryan *et al.,* 2008; Twomey *et al.,* 2007; Waters *et al.,* 2006; World Organization for Animal Health, 2008). *M. kansasii,* a member of atypical mycobacteria (non-MTC) has also been isolated from classical TB lesions (Johnson *et al.,* 1993). In Iraq, PCR tools have proven TB to have affected *Camelus dromedaries* (Al Salihi, 2016).

Primarily, the disease affects the lungs and lymph nodes of many vertebrate animals and humans. Previously, camels did not seem to show high susceptibility towards TB (Mason, 1917; Fowler, 2010). Nonetheless, more recent publications have proven the importance of TB in New World Camelids (NWCs), especially llamas and alpacas reared in countries other than their native South America. In the United Kingdom, a severe emerging disease is gradually spreading among the NWC population (Twomey *et al.,* 2010; Oevermann *et al.,* 2004). Tuberculosis has also affected Old World Camelids (OWCs), comprising dromedaries and Bactrian camels (Mustafa, 1987).

Tuberculosis is one of the top ten causes of death globally. According to the WHO, in 2016, 10.4 million people contracted TB, of which 1.7 million died, including 0.4 million with HIV (http://www.who.int/news-room/fact-sheets/detail/tuberculosis). Low- and middle-income countries constituted over 95% of TB deaths.

The disease is zoonotic, and the causative agent, Multi-resistant tuberculosis (MDR-TB), continuously develops resistance to rifampicin, the most effective first-line drug. Epidemics of bovine TB, particularly among people working in zoos and private herds, are consequently of considerable concern to public health (Pate *et al.,* 2006; Bush *et al.,* 1990;

Dekker, 1962; Dinkla *et al.,* 1991; Moser *et al.,* 2008; Twomey *et al.,* 2010).

Although *M. tuberculosis* is responsible for most human cases, bovine TB, caused by *M. bovis*, is an essential zoonosis that can be transmitted to humans through the ingestion of non-pasteurized milk and dairy products as well as via inhalation of infectious droplets (Thoen *et al.,* 2006). The number of *M. bovis*-related human TB cases has dropped significantly in developing countries as a result of eradication programs and pasteurization of milk (Thoen *et al.,* 2009). However, the zoonotic hazard of *M. bovis* still occurs for people who are in close contact with infected animals. In Russia, *M. bovis* has been isolated in camel milk, which indicated that camel milk, typically consumed without boiling, is also a potential source of infection.

A diagnosis of TB in camelids, which is generally carried out via post-mortem examination, reveals typical gross lesions, followed by the usual histopathological features. A bacterial culture is necessary for confirming the presence of these low-growing organisms, which require a unique selective media, such as Lowenstein- Jensen (Figure 7). Recently, PCR has been used as a rapid confirmatory diagnosis test (Taylor *et al.,* 2007; Thomson, 2006). The diagnosis of TB in living camels is highly problematic due to the absence of characteristic signs. Therefore, additional tests such as the traditional tuberculin skin test (Figure 8) and serological tests are required to reach a diagnosis. According to the World Organization for animal health (OIE), testing of camelids should follow particular guidelines (Wernery *et al.,* 2007).

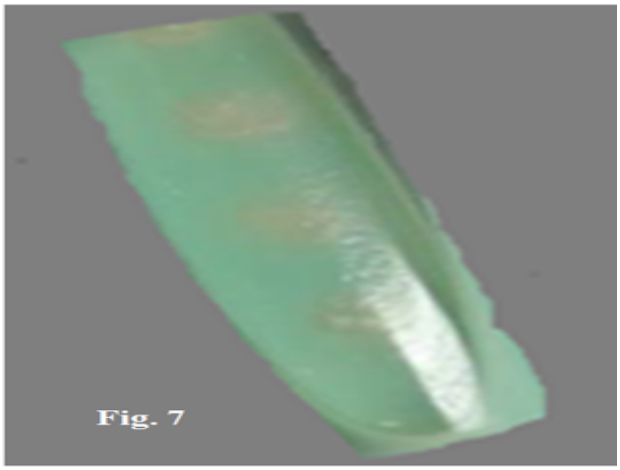

Figure 7. Colonies of Mycobacterial on Lowenstaein-Jensen medium

Figure 8. Measuring a dromedary's axillary skin thickness during intradermal tuberculin testing

Tuberculosis is an intercontinental disease that requires control regulations with the culling of infected animals. Vaccinations are not hitherto available for camelids, and treatment of infected animals is not feasible, even though some treatment measures using anti-TB drugs in captive wild animals have been taken (Thoen *et al.,* 2009). As an example, prophylaxis using isoniazid combined into pelleted feed at a dose of 2.4 mg/kg, fed ad libitum, was attempted in healthy Bactrian camels after the diagnosis of TB in two of the camels in a herd (Bush *et al.,* 1990),; bone marrow suppression due to isoniazid toxicity led to the death of several camels.

Each country should develop a national control program based on intradermal tuberculin testing combined with ante-mortem examination of camelids, removing infected animals and preventing further introduction of infected animals into the herd. Nonetheless, TB will not be fully eradicated until the infection is controlled in reservoir hosts, such as in wildlife (Thoen *et al.,* 2006). .

3. Other causative zoonosis of camelids

Other causative agents with a potential zoonosis risk associated with camelids include orf, ringworm, Q fever, chlamydiosis, leptospirosis, campylobacteriosis, salmonellosis, yersiniosis, listeriosis, pathogenic *E. coli* infections, cryptosporidiosis, and giardiasis.

Orf, or contagious ecthyma, is a viral infection that causes red, raised skin lesions around the face and mouth of young animals and the udder on nursing females. Humans can get infected and develop similar pox-like lesions if they come into direct contact with an animal's lesions (Abu Elzein *et al.,* 1998).

Dermatophytosis, a fungal skin infection commonly known as "ringworm", can be seen in both animals and humans as scaly round areas of hair loss. Both ringworm and orf are transmitted via direct

contact with an infected animal (Al-Ani *et al.,* 1995).

Q fever, *Chlamydophila psittaci* and *Chlamydophila abortus* are agents accompanying with abortion in pregnant camelids that may also be carried by normal animals. Typically, a high concentration of these agents occurs at the time of birth, requiring some care while handling newborn animals, placental tissues, and birth fluids. These agents can be acquired by exposure to placental membranes and foetuses from infected animals using the atomizer. Chlamydophila infections in pregnant women has been related to infectious abortions or miscarriages (Zaher *et al.,* 2017).

Leptospirosis, which is typically shredded in the urine of infected animals, causes reproductive failure and liver and kidney disease in animals. Humans are infected by oral ingestion and by coming in contact with contaminated urine, placenta, and foetal tissues. The organism can also infect through abraded skin.

Salmonellosis, Campylobacteriosis, listeriosis, pathogenic *E. coli* infections, yersiniosis, cryptosporidiosis, and giardiasis are acquired by either direct contact or oral ingestion of faecal material from infected animals. Clinical signs in animals infected with these diseases typically include diarrhea, but some animals may be asymptomatic. Any animal with diarrhea should be suspected of having a zoonotic disease. Individuals in contact with these animals and their environment may develop allergic reactions to animal proteins (allergens) that could lead to asthma. Risk factors for developing an allergic reaction include a history of previous allergies to animals. Symptoms of an allergic reaction include nasal discharge and congestion, conjunctivitis, tearing and eye itching, skin redness, rash or hives and lower airway symptoms (coughing, wheezing and shortness of breath). Exposure to allergens occurs through breathing and by coming in contact with the skin, eyes, and mucous membranes of infected animals. Animal allergens may get detected in animal dander, hair, wool, skin, urine, saliva, serum, and any contaminated feed or bedding materials.

In conclusion, this review has focused on camelids' zoonosis pathogens and conditions that develop primarily via contact with camelids as contaminated bedding or materials, oral ingestion or inhalation of aerosolized fluids. Different health safety precautions should be taken to avoid the risk of exposure to, development of disease, and complications related to camelid pathogens (zoonotic disease).

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