

Clinicopathological investigations among recurrent camelpox outbreaks in Omanis' Arabian camels (*Camelus dromedarius*)

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ABSTRACT. Camelpox remains a widespread viral disease in camelids, with socioeconomic relevance. The present study explored the hematological, biochemical, and histopathological alterations in dromedary-racing camels from the North of Oman infected with camelpox virus diagnosed by real-time PCR. Blood and skin samples were collected from camels with clinical signs and skin lesions (n=4) and from healthy camels (n=3) from 10 different camel herds. The results indicated that the infected camels showed clinical signs, including pyrexia, lacrimation, nasal mucus discharge, affixed and swollen eyelids, emaciation, and pimples on the skin of the head, legs, and abdomen. Hemoglobin, hematocrit, and platelets were significantly greater, with a significant reduction in leukocyte and lymphocyte counts in infected camels than in healthy camels. Infected camels had higher CK and creatinine levels and hepatic-related metabolites, including AST, ALP, AST, GGT, and LDH, than the apparently healthy camels. Histopathological examination of skin scab samples revealed ballooning degeneration of epidermal cells in the presence of typical large eosinophilic intracytoplasmic inclusion bodies and suppurative dermatitis following secondary bacterial infection in all examined infected camels. Camelpox viral DNA was detected using real-time PCR in the blood and skin samples of all infected camels. These findings in dromedary-racing camels associated with a molecular diagnosis of camelpox are described for the first time in the Sultanate of Oman. Therefore, further studies are warranted.

Keywords: Camelpox; Camel; hematological; Oman; Outbreak; Molecular detection.

INTRODUCTION

Camels have socioeconomic importance in Arabian countries as food safety nets owing to their milk and meat consumption. Viral outbreaks are recurrent in camels and are triggered by pathogenic or environmental factors that can restrict vaccination competence. Camelpox virus, a highly contagious disease of camelids, is the etiological agent of this disease. This virus is a member of the genus Orthopoxvirus of the family Poxviridae. Camel keepers encounter significant losses due to morbidity and mortality in infected camels, as well as weight loss in the body and reduction in milk yield (Bhanuprakash *et al.*, 2010b). The disease has erupted in almost every region that practices camel husbandry, and many outbreaks have been reported in several countries in Africa, the Middle East, and Asia (Wernery & Kaaden, 2002), including the Oman (Fassi-Fehri, 1987; Shommein & Osman, 1987; Kumar *et al.*, 2012; Hussain *et al.*, 2015). Camelpox outbreaks have led to huge economic losses through deaths, milk loss, aborted infected camels, and the cost of supportive treatment and prevention of the spread of the disease (Wernery & Zachariah, 1999; Bhanuprakash *et al.*, 2010a).

Camelpox presents with clinical signs, including pyrexia, enlarged lymph nodes, and characteristic pox skin lesions (Bhanuprakash *et al.*, 2010b). Moreover, Camelpox disease appears in either a generalized or a localized form. In the generalized form, pox lesions spread all over the skin and extend to the esophagus, trachea, and lungs (Wernery & Kaaden, 2002; Narnaware *et al.*, 2021). In the localized form, the lesions are strictly in the skin and start as papules that transform into vesicles and pustules, followed by scab formation, which may take up to 4–6 weeks to heal. Histopathological lesions include typical cytoplasmic vacuolation of epidermal stratum spinosum keratinocytes, with large eosinophilic intracytoplasmic inclusions. Vesicles form following the rupture of swollen cells, and lesions are usually associated with the infiltration of mononuclear cells and neutrophils. Hyperplastic epithelium may be observed at the rim of the skin lesions (Balamurugan *et al.*, 2013). Rainy seasons, common watering sources, and the introduction of new camels into herds are risk factors for a higher incidence of camelpox disease. The transmission of camel poxvirus usually occurs by direct or indirect contact, as the virus is secreted in various body fluids, including nasal and ocular discharges, milk, and saliva of infected

animals. Infection is caused by inhalation, skin abrasion, and mechanical transmission through ticks (Bhanuprakash *et al.*, 2010b; OIE, 2021). Camel pox is clinically diagnosed on the basis of the identification of classical signs. However, confirmation is required to exclude similar effects such as contagious ecthyma (Orf), papillomavirus, and insect bites (Khalafalla *et al.*, 2015; OIE, 2021). Camel pox can be confirmed by several tests like transmission electron microscopy, immunohistochemistry, virus isolation, serological tests, and molecular tests like PCR (Bhanuprakash *et al.*, 2010a; Bayisa, 2019).

Camel pox has been reported in many developing countries in Asia and Africa (Mohammadpour *et al.*, 2020). The disease has been reported among camels in the Sultanate of Oman; however, there is a lack of reports regarding clinicopathological data on the disease. This is the first study to report hematological, biochemical, histopathological, and molecular investigations related to camel pox infection in camels from the Sultanate of Oman.

MATERIALS AND METHODS

Samples collection

In October 2020, an outbreak of skin lesions in camels was reported on ten farms in the Sultanate of Oman. Samples were collected from camel herds reared in Aswad, Shinas Province, Al Batinah North Governorate, Sultanate of Oman. The animals were kept under a closed rearing system of a dromedary camel herd. Infected camels were male and female, aged between 1 and 10 years. The infected camels showed clinical signs. Blood samples ($n=7$) of both apparently healthy ($n=3$) and infected ($n=4$) camels from each farm were collected to study hematological and biochemical alterations and DNA extraction. Skin scabs were collected and deposited in 10% neutral buffered formalin for histopathological examination.

Hematological and plasma biochemical assessment

Blood samples (5 ml) were collected from the jugular vein in heparin tubes and stored at 4 °C until examination. Once in the laboratory, the samples were further separated into two subsamples. The first was used for hematological examinations. The second sub-tube was centrifuged (3000 rpm for 20 min) for plasma separation and stored at -20 °C for blood metabolites. Using the automated Haematology Veterinary Analyser (ABX Micros ES 60®, Hori-ba Company, UK), the total of white blood cells (WBC), differential leucocyte (DLC), red blood cell count (RBC), hemoglobin (Hb), hematocrit (PCV), and platelets count were measured.

The plasma components of the protein fractions (total protein and albumin), creatinine, creatinine kinase, alkaline phosphatase (ALP), lactate dehydrogenase (LDH), gamma-glutamyl transferase (GGT), aspartate aminotransferase (AST), and alanine transaminase (ALT) were assessed using commercially available kits (Pars Azmun Corporation

Ltd., Tehran, Iran) according to the manufacturer's instructions, using auto-analyzer biochemistry (Chem 299, Gesan, Italy).

Histopathological examination

To assess the histopathological alterations, specimens were fixed in 10% neutral buffered formalin and then dehydrated with increasing concentrations of alcohol (65%, 75%, 90%, and 100%). Fixed specimens were embedded in paraffin using an automated processor (MTM-SLEE, Germany), sectioned with a Leica® microtome into 5 µm sections, and routinely stained with hematoxylin and eosin according to the method described by Suvarna *et al.* (2018). Images of tissues were captured using a Nikon ECLIPSE E200 microscope.

DNA extraction and real-time PCR

DNA was extracted from whole blood samples of healthy and infected camels. DNA was isolated using a DNeasy Blood & Tissue kit (Qiagen, Germany) following the manufacturer's instructions (Alhaddad *et al.*, 2019). The quality and quantity of DNA were then determined. Extracted DNA from blood and skin scab tissue samples was tested by real-time PCR using a commercial Genetic PCR Solutions (GPSTM) Camel pox virus MONODOSE dtec-qPCR kit (Alicante, Spain), following the manufacturer's protocol. The thermal cycling conditions were 95°C for 1 min, followed by 40 cycles of denaturation at 95°C for 10 s, extension, and data collection at 60°C for 1 min.

Statistical analysis

The differences between the means of the two groups (infected and apparently healthy) were tested for significant differences using a t-test (Proc T-test; SAS Institute Inc., 2012). Statistical significance between means was set at a p -value less than 0.05. The results are expressed as the mean \pm SEM.

RESULTS

Clinical signs and treatment

Our data revealed that the infected camels exhibited clinical signs of different severity, including anorexia, pyrexia, lacrimation, swollen eyelid, nasal mucus discharge, and pock lesions at various skin parts such as nostrils, mouth, head, legs, and abdomen, especially in the end periods (Figure 1 A, B, and C). No mortality was recorded from any of the camels during data collection from the camel herds. Clinically infected camels were isolated and treated successfully with three doses of long-acting oxy-tetracycline (20 mg/kg every two days) with a topical antibiotic spray (chloramphenicol/gentian violet).

Haematological and biochemical profile

The hematological values of the infected and apparently healthy camels are presented in Table 1. Hb, PCV, WBC,

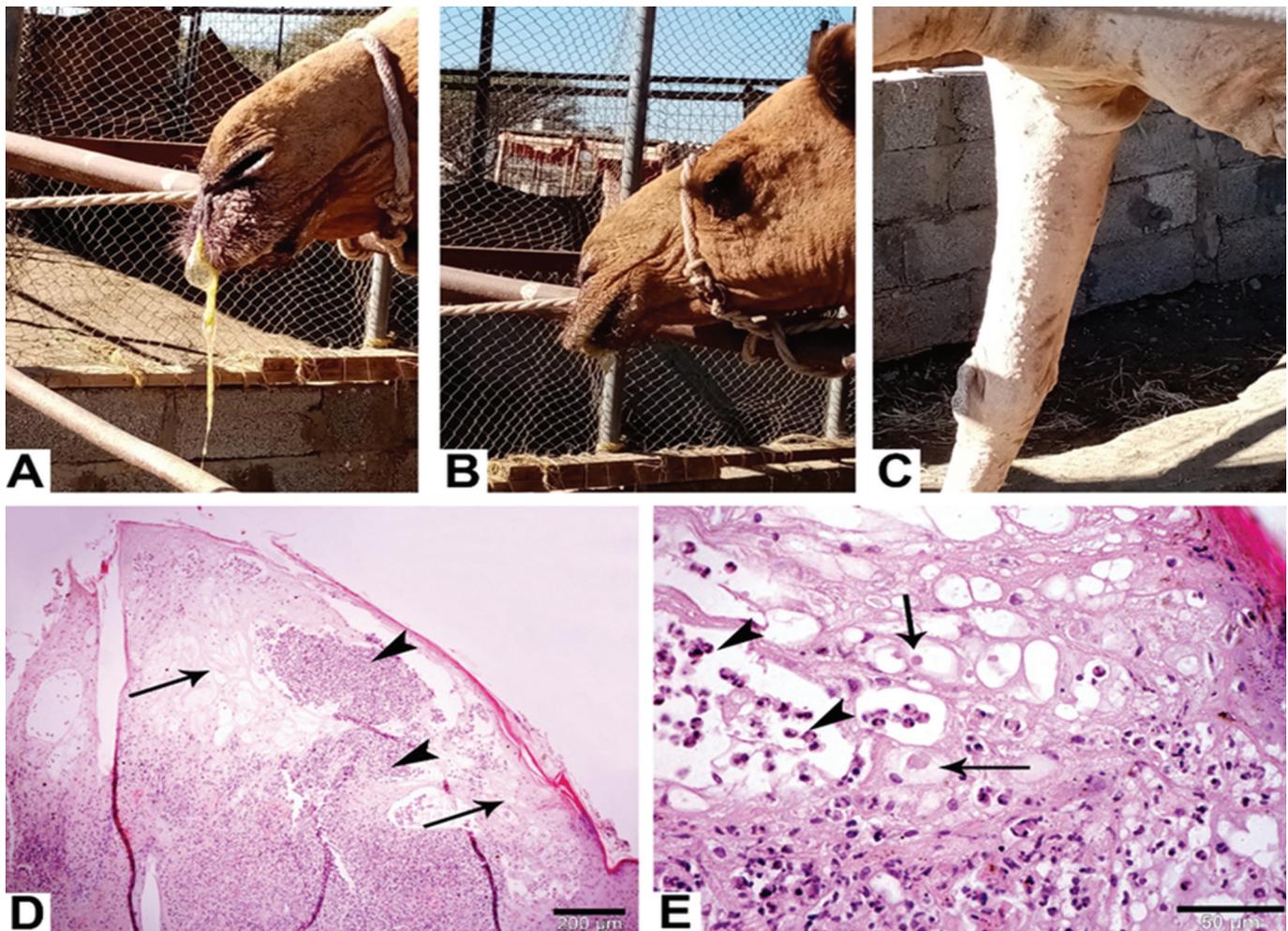


Figure 1. An adult racing dromedary camel showing nasal discharges along with papules over the head and the forelimb (A, B, C); Skin scabs from camelpox infected animal showing marked hydropic and ballooning degeneration (arrows) of the epidermal cell layer with large aggregates of dead and living neutrophils forming microabscesses (arrowheads) (D) (H&E stain, Bar= 200µm); Large eosinophilic intracytoplasmic inclusion bodies (arrows) in the degenerated cells of the stratum spinosum along with aggregates of neutrophils (arrowheads) (E) (H&E stain, Bar= 50µm).

platelet, and neutrophil levels were significantly different between the two groups. Hb, PCV, and platelet values significantly increased by 24.8%, 27.6%, and 5.0%, respectively, in the infected group compared to those in the healthy group. Infected camels had lower ($p < 0.05$) WBC and lymphocyte counts by 33.05% and 14.4%, respectively, than apparently healthy camels (Table 1). The plasma biochemical values of infected and healthy camels are shown in Table 2. The values of creatinine and CK were significantly increased ($p < 0.05$) in infected camels by 55.5% and 41.08%, respectively, compared with those in the healthy camel group. In contrast to the healthy camel group, all hepatic-related enzymes, ALP, LDH, AST, and GGT, were significantly increased in the infected camels by 70.4%, 48.9%, 58.3%, and 51.7%, respectively. No significant effects were detected for ALT levels.

Histopathological examination

As shown in Figure 1(D) and 1(E), examination of the skin scab tissues revealed marked hydropic and ballooning degeneration of the epidermal cell layer, with large eosinophilic intracytoplasmic inclusion bodies. The scabs also exhibited large aggregates of dead and living neutrophils that formed microabscesses.

Molecular detection of camelpox virus

Camelpox viral DNA was successfully detected using a commercial kit for real-time PCR from the blood and skin scab tissue samples of camels that exhibited clinical signs and only one of the apparently healthy camels.

Table 1. Haematological values of camels infected by campelpox virus showing skin lesions and apparently healthy. Results are expressed as means \pm SE.

Variable ¹	Infected	Apparently healthy
Erythrocytes		
Hb (g/dl)	15.1 \pm 0.83*	12.1 \pm 0.72*
RBC (10 ⁶ / μ l)	9.9 \pm 0.62	7.7 \pm 0.69
PCV%	41.3 \pm 1.14*	32.4 \pm 1.07*
Platelets	607.5 \pm 2.46*	578 \pm 2.17*
Leucocytes		
WBC (10 ³ / μ l)	8.7 \pm 0.80*	13.1 \pm 0.57*
Neutrophil (%)	74.8 \pm 0.75*	65.4 \pm 0.54*
Lymphocyte (%)	17.3 \pm 0.76	19.4 \pm 1.38

Hb: hemoglobin; RBC, red blood cell; PCV, packed cell volume; WBC, white blood cells. * $p \leq 0.05$ is statistically significant.

Table 2. Plasma biochemical values obtained from camels infected with the Camelpox virus showing skin lesions and apparently healthy. Results are expressed as means \pm SE.

Variable ¹	Infected	Apparently healthy
Protein fraction		
Total protein (g/dl)	7.8 \pm 1.09	6.55 \pm 0.27
Albumin (g/dl)	2.92 \pm 0.51	3.54 \pm 0.92
Cardiac indices		
CK (U/L)	179.6 \pm 1.14*	115.5 \pm 1.32*
Kidney indices		
Creatinine (mg/dL)	2.21 \pm 0.05*	1.57 \pm 0.1*
Hepatic indices		
ALP (U/L)	154.4 \pm 0.95*	90.6 \pm 1.04*
LDH (U/L)	1176 \pm 6.92*	591 \pm 4.83*
AST (U/L)	79.4 \pm 3.73*	50.15 \pm 2.28*
ALT (U/L)	6.24 \pm 0.270	6.4 \pm 0.31
GGT (U/L)	11.5 \pm 1.75*	7.58 \pm 1.95*

¹CK, creatinine kinase; ALP, alkaline phosphatase; LDH, lactate dehydrogenase; GGT, gamma-glutamyl transferase; AST, aspartate aminotransferase; ALT, alanine transaminase. * $p \leq 0.05$ is statistically significant.

DISCUSSION

This study investigated outbreaks of camelpox disease in camel herds in Oman. All infected camels had localized skin lesions on their head, neck, abdomen, and limbs. No mortality was recorded for any of the affected animals. The outbreak occurred in October, a hot and humid month in the coastal Al-Batinah North Governorate, which may

have played a role in initiating the infection. Our data are in agreement with previous investigations, where a higher incidence and more severe form of camelpox outbreaks were observed in the rainy season than in the dry season, in addition to its association with calf weaning and poor nutrition (Wernery *et al.*, 1997; Wernery & Kaaden, 2002; Bhanuprakash *et al.*, 2010; Mohammadpour *et al.*, 2020).

As reported in our study, infected camels typically show

clinical signs of localized camelpox infection. These clinical signs, including pyrexia, anorexia, lacrimation, swollen eyelids, and pock lesions on the skin of the head, neck, legs, and abdomen, which form pustules and scabs, are in agreement with other reported camel pox outbreaks (Narnaware *et al.*, 2021; OIE, 2021; Mohammadpour *et al.*, 2020). Moreover, similar studies (Wernery & Kaaden, 2002) have mentioned that skin lesions initially appear as erythematous macules, develop into papules and vesicles, and later turn into pustules. Lacrimation and excess salivation in camels have been previously described by several authors (Aregawi & Feyissa, 2016; Dahiya *et al.*, 2017; Narnaware *et al.*, 2018). Additionally, we detected camelpox viral DNA in all camels showing clinical signs and in one of the apparently healthy camels that could be carriers or during the incubation period.

Significant changes in Hb, PCV, WBC, neutrophil, and platelet counts were observed between the infected and healthy camels. In this study, infected camels had higher levels of Hb, PCV, and platelets, and lower levels of WBCs and leukocytes. In agreement with previous reports (Kachhawa-ha *et al.*, 2014; Narnaware *et al.*, 2018), hyperproteinemia in camels infected with the camelpox virus in India was attributed to anorexia related to mouth lesions. Moreover, Bhanuprakash *et al.* (2010) indicated that camels infected with camelpox exhibited an increase in Hb and PCV and a significant reduction in WBC and lymphocyte counts. In addition, hepatic metabolites, such as GGT and AST, were higher in camelpox-infected camels (Hussein & Al-Mufarrej, 1999). This may be associated with the attachment of the virus to blood cells and prevention of their mobility and functionality. However, other studies have indicated that camelpox has no considerable impact on biochemical and hematological parameters (Bhanuprakash *et al.*, 2010). In line with our data, De *et al.* (2020) reported that leukocyte counts were significantly reduced in camelpox-infected camels than in healthy camels. During the initial period of infection, leukopenia has been detected in ruminants and is associated with augmented tissue requests and neutrophil margination (Hussein & Al-Mufarrej, 1999). The presence of other antigens in systemic infectious syndromes may lead to lymphopenia due to the lymph node confiscation of peripheral blood lymphocytes (Bhanuprakash *et al.*, 2010; Du *et al.*, 2020). Moreover, exposure of camels to *Trypanosoma evansi* infection, as evidenced by the detection of specific antibodies against the parasite, might have a role in suppressing animal immunity and hence be vulnerable to camelpox virus infection (OIE, 2021). However, our results partially agree with those of Narnaware *et al.* (2015), who reported that the blood and biochemical parameters in camel calves infected with para-poxvirus (camel contagious ecthyma) were not significantly affected.

In addition to the clinical signs and haematological changes induced by camelpox, it is critical to explore the histopathological changes in camel skin lesions to select a more appropriate treatment strategy. In the current study,

marked hydropic and ballooning degeneration of the epidermal cell layer, with large aggregates of dead and living neutrophils, formed microabscesses in addition to large eosinophilic intracytoplasmic inclusion bodies in the degenerated cells of the stratum spinosum. Histopathological changes associated with camelpox infection have been previously described (Dahiya *et al.*, 2017; Narnaware *et al.*, 2018). Moreover, marked acanthosis, ballooning degeneration, and vacuolation of the prickle cell layer, in addition to homogenous eosinophilic inclusion bodies, were reported by Khalafalla *et al.* (1998), as indicated in our present investigation. Similarly, Narnaware *et al.* (2021) revealed marked epithelial hyperplasia in infected camel's tongue with noticeable vacuolation and hydropic/ballooning degeneration with the presence of oval-shaped intracytoplasmic eosinophilic inclusion bodies in large numbers.

Very few records are available on the review of results related to molecular detection by PCR of camelpox viral DNA in camel species. PCR revealed the presence of genomic virus in all infected camels. Consistent with our data, many authors (Nagarajan *et al.*, 2013; Khalafalla *et al.*, 2015; Dahiya *et al.*, 2017) have demonstrated that PCR amplification of camelpox viral DNA from blood samples is a rapid, sensitive, and specific assay for the early detection of camelpox infection. Moreover, the assay detected viral particles in an apparently healthy camel. Molecular identification of Camelpox virus DNA is reported in this study to provide more evidence for clinical, histological, and blood biochemical changes. This molecular diagnostic method should be applied, especially in dromedary racing camels. In this regard, we expect that this study will incentivize the use of molecular techniques for the diagnosis of viral or bacterial infections in dromedary-racing camels.

The detection of camelpox virus in dromedary-racing camels using molecular and histopathological techniques ensures early intervention by veterinarians for better treatment and prevention of disease occurrence. This could also help in selecting a suitable treatment and prevention strategy, and invite other researchers to develop new therapies for this disease.

Ethics statement

All procedures in this study were in accordance with the regulations of the Ethics Committee for Animal Use in Research, Sultan Qaboos University, Sultanate of Oman.

Conflict of interest

The authors declare no conflicts of interest regarding the publication of this manuscript.

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