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Some Clinicopathological Aspects of Camelpox in Saudi Arabia

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Abstract. The clinicopathological aspects of an outbreak of camelpox in an experimental camel herd in Riyadh are recorded. The morbidity rate in camel calves was 100% with a corresponding morbidity rate of 33% in pregnant females. Premonitory signs comprised fever, inappetence and listlessness, followed by localized or generalized skin eruptions typical of this disease. One of the pregnant females aborted and another gave a stillbirth. However, apart from one case of neonatal infection that terminated fatally, all of the infected camels recovered slowly. Blood analysis of the infected camel calves revealed anemia, hypoalbuminemia and elevated serum aspartate aminotransferase and gamma-glutamyl transferase activities.

Introduction

Camelpox (CP) is a highly contagious virus infection caused by an orthopoxvirus that probably existed in the dromedary (*Camelus dromedarius*) and the bacterian camel (*Camelus bactrianus*) since the Middle Ages [1]. It was first recorded in India in 1909 [2], and subsequently reported in virtually all camel raising countries world-wide [3]. In Saudi Arabia, preliminary records of acute CP were made from Al-Jouf [4], while a milder, slowly spreading form of the disease was reported from Hafof [5]. CP was also reported from several neighboring countries including Iran [6,7], Iraq [8,9], Yemen [10], the Sultanate of Oman, United Arab Emirates [11, 12] and Bahrain [13].

Previous records of CP in Saudi Arabia focused primarily on virus isolation and characterization while the clinical manifestations of the disease were either overlooked or only briefly outlined. This communication describes the clinicopathological changes

occurring during a severe, naturally acquired outbreak of CP in a herd of experimental camels housed in a farm near Riyadh.

Materials and Methods

Animals: The affected herd consisted of eleven, 4-6 years old female camels, one six years old male and ten 12-14 months old camel calves (five males and five females). The camels were kept under intensive management and fed on a balanced ration of lucerne and concentrate (16% crude protein) with *ad libitum* supply of water. They were free from endoparasites, mange mites and other ectoparasites, and none of them was previously infected with, or vaccinated against, CP.

Blood analysis: The camel calves were sampled for hematological and blood biochemical analyses on a monthly basis in connection with another study, the last readings being recorded two weeks prior to the onset of CP. When the disease broke out, two additional blood and serum samples were collected from each camel calf on days 1 and 7 of the outbreak. These were analyzed for hematological indices, serum proteins, glucose, bilirubin, urea, blood urea nitrogen concentration (BUN) and serum aspartate aminotransferase (AST), alanine aminotransferase (ALT) and gamma-glutamyl transferase (GGT) activities. The techniques used were reported previously [14-16], and the data were subjected to statistical analysis using least squares analysis procedure in SAS [17].

Histopathology: Skin biopsies were taken from affected sites for histopathological examination. These were fixed in 10% neutral formalin, mounted in paraffin, sectioned and stained with hemotoxylin and eosin.

Diagnosis: Skin biopsies and scabs were collected into sterile containers and were despatched in ice for laboratory diagnosis.

Results

The onset of the disease was sudden, involving initially two male camel calves and spreading to all of the remaining calves on the farm within 48 hours. Premonitory signs were fever, listlessness, inappetence and a watery discharge from the eyes and nostrils. Twenty-four hours later, typical pox lesions were observed, namely papules, vesicles, postules and wart-like nodules around the mouth, eyes and nostrils (Fig. 1), and to a lesser extent in the perineum, genitalia, teats, limbs and under the tail. The lips were swollen and oedematous, while the animals showed intense pruritis. Nasal discharge became thick, mucopurulent and foul smelling, resulting in labored breathing. Palpation revealed no enlargement of superficial nodes.

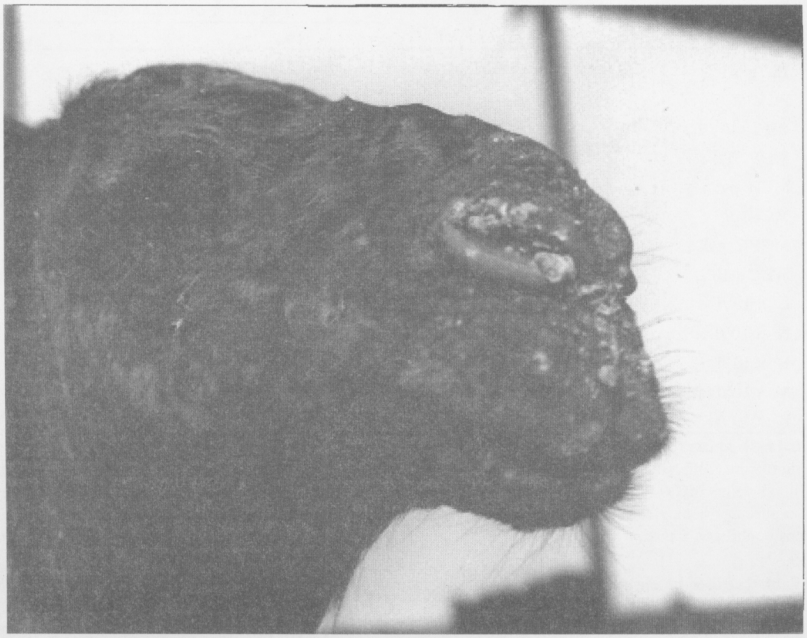

Fig. 1. Pox lesions on the face of an infected calf.

The condition reached its height during the first four days after the appearance of skin eruptions, with acute manifestations lasting for about 10 days during which the affected camels were almost completely off food. Thereafter, the clinical signs gradually subsided while the lesions started to dry and fall off, leaving thickened, hairless areas and dry scabs on the skin. However, complete recovery extended over a period of more than one month.

The blood picture was consistent with rapid loss of condition, namely, significant reduction in hemoglobin (Hb), packed cell volume (PCV), red (RBC) and white (WBC) blood cell counts, lymphocyte counts, neutrophil counts and MCHC, along with hypoproteinemia, hypoalbuminemia and increased serum ALT and GGT activities (Tables 1,2). Non-significant changes were recorded in other blood parameters.

Table 1. Haematological indices of camel calves before and after infection with camelpox[1]

Parameter	Pre-infection	Days after onset of disease	
		Day 1	Day 7
Hb g/dl	13.0±0.07[a]	12.9±0.08[a]	11.7±0.08[b]
PCV %	27.0±0.15[a]	28.0±0.26[a]	25.9±0.24[b]
RBC x10^{12}/L	9.7±0.10[a]	9.5±0.21[a]	7.7±0.21[b]
MCV fl	27.0±0.53	27.8±0.92	29.9±0.89
MCH pg	13.3±0.25	13.4±0.39	13.9±0.37
MCH pg	13.3±0.25	13.4±0.39	13.9±0.37
MCHC g/dl	47.6±0.13[a]	46.9±0.27[a]	45.3±0.28[b]
WBC x 10^9/L	9.7±0.45[a]	9.8±0.71[a]	6.9±0.55[b]
L x 10^9/L	5.9±0.34[a]	6.0±0.24[a]	4.1±0.21[b]
N x 10^9/L	4.4±0.37[a]	3.7±0.13[a]	1.8±0.28[b]
E x 10^9/L	0.2±0.05	0.3±0.07	0.2±0.05
M x 10^9/L	0.2±0.04	0.2±0.03	0.2±0.03
B x 10^9/L	0.2±0.02	0.09±0.02	0.04±0.02

1=Least squares mean (LSMean) and standard error (SE). Different superscripts in the same row indicate significant differences (P<0.005 - P<0.0005). Hb = hemoglobin; PCV = packed cell volume; RBC = total red blood cells; MCV = mean corpuscular volume; MCH = mean corpuscular hemoglobin; MCHC = mean corpuscular hemoglobin concentration; WBC = total white blood cells; L = lymphocytes; N = neutrophils E = eosinophils; M = monocytes; B = basophils.

Table 2: Blood biochemical parameters of camel calves before and after infection with camelpox[1]

Parameter	Pre-infection	Days after onset of disease	
		Day 1	Day 7
TP g/L	68.5±1.13[a]	69.0±1.42[a]	62.9±1.38[b]
ALB g/L	2.6±0.94[a]	43.1±0.89[a]	39.1±0.86[b]
GLB g/L	23.9±1.52	25.9±1.40	23.8±1.42
A:G	1..9±0.15	1.7±0.27	1.7±0.19
ALT U/L	13.1±0.55[a]	13.9±0.38[a]	15.6±0.24[b]
AST U/L	98.0±1.00	102±0.84	103±1.00
GGT U/L	6.3±0.43[a]	6.4±0.38[a]	14.5±0.30[b]
GLU mg/dl	68.4±1.67	67.2±1.83	63.8±1.77
BIL mg/L	2.3±0.78	2.6±0.85	2.2±0.70
CHO mg/L	0.8+0.64	0.5±0.45	0.5±0.46
Urea mmol/L	11.5±1.1	10.1±1.06	10.7±1.16
BUN mmol/L	5.4±0.03	4.7±0.04	5.0±0.03

1=Least squares mean (LSMean) and standard error (SE). Different superscripts in the same row indicate significant differences (P<0.005 - P<0.0005). TP = total proteins; ALB = albumin; GLO = globulins; A:G = albumin:globulin ratio; ALT = alanine aminotransferase; AST= aspartate aminotransferase; GGT= gamma glutamyl transferase; GLU = glucose; BIL = bilirubin; CHO = total cholesterol; BUN = blood urea nitrogen.

Histolopathological examination of the skin lesions revealed typical proliferative and vacuolar degenerative changes in epithelial cells in the affected sites.

Two of the camel calves were particularly severely affected showing generalized cutaneous eruptions (Fig 2), erosions of the buccal mucosa and diffuse swelling of the extremities. These two calves also showed more marked anemia, leucopenia and hypoalbuminemia than the remaining calves.

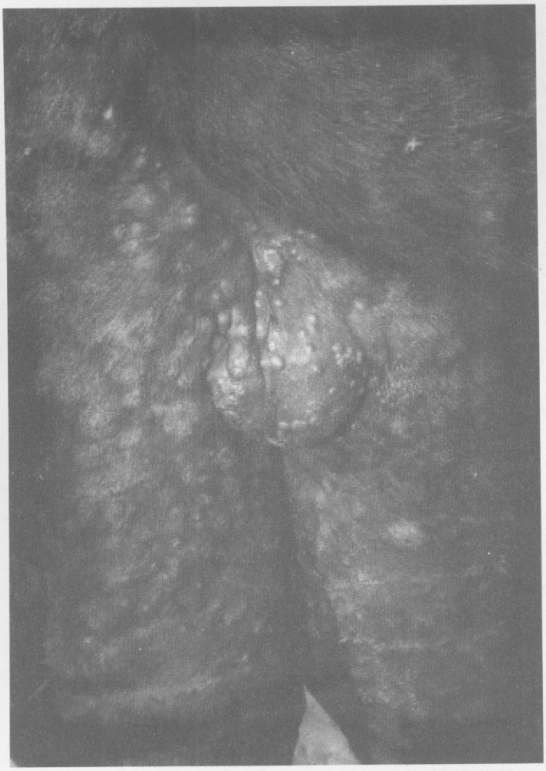

Fig. 2. Generalized pox lesions in an infected camel calf.

Four adult females in late pregnancy contracted the disease five days after the appearance of CP in the camel calves. One of them developed a severe, generalized condition with consequent abortion, while the remaining three exhibited a moderate infection, yet one of them gave a stillbirth. The remaining two camels delivered normally during the course of the disease. However, one of the newborns rapidly developed a fatal

infection characterized by high fever, facial oedema, dyspnoea, lethargy and generalized eruptions. Besides, it showed severe buccal erosions that prevented it from suckling, and eventually developed a profuse diarrhoea and died at the age of ten days. Post-mortem examination revealed anasarca, diffuse cutaneous and subcutaneous nodules and lymphadenopathy.

The disease was tentatively diagnosed as camelpox. However, to distinguish it from other pox-like diseases of camels, skin scabs were sent to the Veterinary Research Laboratories in Riyadh. The diagnosis was confirmed by electron microscopy of the scabs, which revealed orthopoxvirus, and by inoculation into chicken egg embryos via the chorioallatoic membrane in which typical whitish pocks lesions were produced.

No specific treatment is known but the animals were given broad-spectrum antibiotics and multivitamins during the course of the disease to control secondary infections.

Discussion

With global eradication of smallpox, CP stands as economically the most important disease caused by an orthopoxvirus [18, p.303-309]. While this disease was reported from some parts of Saudi Arabia, its epidemiology, pathogenesis and economic significance in indigenous camels are still unknown, and further detailed studies are needed to elucidate these aspects.

The symptoms and lesions recorded in the present camels were essentially similar to those reported in camels elsewhere [3; 10] and the lesions around the mouth and eyes also tended to resemble the verrucoid form of sheepox. During the present outbreak, the morbidity was 100% in young camels and around 33% in adult camels. Although the case fatality rate in both instances was 0%, the intensity of the disease was marked in camel calves, while three of the four affected adult females lost their offsprings due to abortion or neonatal infection. By contrast, a morbidity rate of only 10% was encountered during CP outbreak among range camels in Hofuf [5], which probably indicates that virus strains of different virulence existed in the field. These results are consistent with the literature. In most cases, CP was reported to be relatively benign, especially in adult camels [3]. However, severe infections associated with a high case fatality and blindness are not uncommon in young camels, while abortions and stillbirths, weight loss and reduction in milk production are sometimes recorded in adult animals [6; 13; 19-22]. The occurrence of a severe, fatal form of CP in one of the newborn camels in the present herd is noteworthy since it was previously maintained that offsprings born during an outbreak to infected dams acquired a relatively high level of maternal immunity [20]. Also the occurrence of a severe and generalized form of CP in an adult she-camel indicates that previously unexposed camels of any age are probably highly susceptible to infection. In this particular instance, the severity of the condition might have been precipitated by pregnancy.

Finally, the present study showed that CP produced significant changes in the blood constituents of affected camels, the most important of which being anemia and hypoproteinemia which could be attributed to the marked inappetence and, hence, under-nourishment during the acute phase of the disease. On the other hand, the increased level of some serum enzymes might suggest systemic involvement; but further studies into the enzyme changes during the course of this disease need to be carried out before making any specific inferences. To our knowledge, these changes in blood constituents of camels during the course of camelpox disease were not studied previously.

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بعض السمات السريرية والباثولوجية لمرض جدري الإبل في المملكة العربية السعودية

منصور فارس حسين و سعود بن إبراهيم المفرج
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(قدم هذا البحث للنشر في ١٤١٨/١١/٩هـ؛ وقبل للنشر في ١٤١٩/٢/٧هـ)

ملخص البحث. يتناول هذا البحث الأعراض والتغيرات الباثولوجية لوباء الجدري في قطيع من الإبل النجدية في الرياض، إذ بلغت النسبة المرضية في الحواشي ١٠٠٪ تقابلها نسبة تتجاوز ٣٣٪ في النوق الحوامل، وكانت أولى بادرات المرض ارتفاع في حرارة الجسم، وفقد الشهية والخمول، أعقب ذلك ظهور بثرات الجدري المميزة بصورة موضعية أو منتشرة في الجلد. وقد أجهضت إحدى النوق الحوامل، بينما وضعت أخرى مولودا ميتا كامل النمو. كما حدثت إصابة واحدة مميتة في حاشي حديث الولادة. أما بقية الإبل فقد تماثلت للشفاء ببطء. تم تحليل المكونات الخلوية والكيميائية للدم في الحواشي، وشملت التغيرات المرافقة للمرض فقر الدم وانخفاض نسبة الزلال فيه مع زيادة في نشاط أنزيم اسبارتات أمينو ترانسفراز وأنزيم جاما جلوتاميل ترانسفراز في مصل الدم.