

A NOTE ON EXPERIMENTALLY INDUCED SEVERE CAMEL ORF (AUZDYK DISEASE) IN DROMEDARY CAMELS

E.M.E. Abu-Elzein, F.M.T. Housawi, A.I. Al-Afaleq, A.A. Gameel and R.O. Ramadan

College of Veterinary Medicine and Animal Resources,
King Faisal University, Hufuf - 1757, AL-Ahsa 31982, SAUDI ARABIA

It is rather difficult to differentiate, clinically, between camel orf and camel-pox infection in countries where both diseases exist (Wernery and Kaaden, 2002). However, such differentiation becomes more difficult when the camel orf takes the generalised form (Mahnel and Munz, 1987).

Although the etiological agents of camelpox and camel orf belong to the same virus family (Esposito *et al*, 2002), still they are not related and there is no cross-immunity as such between them (Wernery and Kaaden, 2002).

Scanty information is available regarding cross, mixed or super infection between camelpox and camel orf viruses. However, Wernery and Kaaden (1995) reported a mixed infection of camelpox and parapox virus, where they artificially infected the camels with camelpox virus. These camels got natural infection by parapox virus. The authors could see both viruses situated next to one another upon electron microscopy. They suggested that the situation might have been due to super infection with the contagious ecthyma virus or that the camels were latent carriers of the virus.

The aim behind the present study, was to extend the observations of Wernery and Kaaden (1995), by experimentally infecting dromedary camels, which were convalescent from natural generalised camelpox virus infection, with camel contagious ecthyma virus. Results of these experiments are expected to help the field veterinarians in better understanding of the clinical forms of these two diseases and that they should be aware of the probable complications which can occur between them, under the field conditions, such as that reported by Wernery and Kaaden (1995).

In Saudi Arabia, both camelpox and camel orf do exist (Hafez *et al*, 1986; Hussein *et al*, 1987; Abu Elzein *et al*, 1998). Although camelpox is endemic in

the country and is causing great losses in camel calves (Hafez *et al*, 1986; Hussein *et al*, 1987), camel orf has only recently been identified (Abu Elzein *et al*, 1998). The generalised camel orf, as described by Mahnel and Munz (1987) has not yet been reported in Saudi Arabia.

Four locally-bred, dromedary camels that aged 3 to 4 years were used in the present study. These camels had previously suffered from a natural generalised skin infection, which was diagnosed as camelpox (Abu Elzein *et al*, 1999). A month following complete healing of the pox lesions, the four camels were experimentally infected with camel orf scab material, in the form of 50% homogenate, in phosphate buffered saline (PBS), pH 7.4, as described by Abu Elzein *et al* (1998). The used scabs were collected from naturally infected dromedary camels (Abu Elzein *et al*, 1998). The inoculated camels were kept in isolation, provided with food and water, *ad libitum* and observed daily for clinical signs.

The inoculated camels developed severe typical clinical signs of camel orf infection as described by Abu Elzein *et al* (1998). The whole span of the disease took six weeks. Scab materials were collected and the virus was identified as contagious ecthyma virus, as described by Abu Elzein *et al* (1998).

Results, in this report, indicated that the inoculated camels, which were at convalescence from generalised camelpox infection, developed severe camel orf infection. This had confirmed that, there is no cross-protection between camelpox and camel orf viruses and that cross or super infection can occur between the two viruses.

The present results and those of Wernery and Kaaden (1995), proved that clinical mix-up between camelpox and parapox infection, in the field, could not be ruled out. This is a situation for which attention of the field veterinarians must

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be drawn; particularly when performing clinical differential diagnosis and in case of recommending vaccination against camelpox.

Acknowledgements

The authors would like to thank Mr.A.AI-Khars for technical assistance.

References

- Abu Elzein EME, Coloyan ER, Gameel AA, Ramadan RO and AL-Afaleq AI (1998). Camel contagious ecthyma in Saudi Arabia. *Journal of Camel Practice and Research* 5(2):225-228.
- Abu Elzein EME, Gameel AA, Ramadan RO and Housawi FMT (1999). An eruptive moderate form of camelpox infection in dromedary camels (*Camelus dromedarius*) in Saudi Arabia. *Revue Scientific Technical Office International Epizootic* 18:749-75.
- Esposito JJ, Baxby D, Black DN, Dales S, Darai G, Dumbell KR, Granados RR, Joklik G, Moss B, Moyer RW, Pickup DJ, Robinson AJ and Tripathy DN (2002). Family: Poxviridae. [http:// www.ncbi.nlm.nih.gov/ICTVdb/Ictv/fs_poxvi.htm](http://www.ncbi.nlm.nih.gov/ICTVdb/Ictv/fs_poxvi.htm)
- Hafez SM, Eissa AM, Amjad AM, Al-Sarif AK and Al-Sukayran A (1986). Preliminary studies on camelpox in Saudi Arabia. *Proceedings of 9th Symposium Biological Aspects of Saudi Arabia*, 24-27 March, 1986, Riyadh, King Saud University Press, 9 Saudi Biological Society. pp 7-14.
- Hussein MF, Hafez SM, Gar EL and Nabi M (1987). A clinico-pathological study of camelpox in Saudi Arabia. In the 10th Proceeding:Symposium on Biological Aspects of Saudi Arabia, Riyadh 20-24 April 1987. King Saud University Press (Saudi Biological Society, Ed.). pp 8-14.
- Mahnel H and Munz E (1987). Zur derzeitigen epizootologischen Lage bei den Tierpocken. *Tierarztl. Umschau* 42:5-14.
- Wernery U and Kaaden OR (1995). In: *Infectious Diseases in Camelids*. 1st Ed. Blackwell Wissenschafts-Verlag, Berlin.
- Wernery U and Kaaden OR (2002). In: *Infectious Diseases in Camelids*. 2nd Ed. Blackwell Wissenschafts-Verlag, Berlin.

Lack of gender effect on the pharmacokinetics and pharmacodynamics of dexamethasone in the camel after intravenous administration

N. A. Al-Khatheeri, I. A. Wasfi, M. Lambert and A. Saeed

The pharmacokinetics and pharmacodynamics of dexamethasone were studied in six male and six female camels after a single intravenous dose (0.05 mg kg⁻¹ body weight) of dexamethasone. The pharmacokinetics parameters of the two-compartment pharmacokinetic model for female and male camels, respectively (mean ± SEM) were as follow: terminal elimination half-lives were 8.02 ± 1.15 and 7.33 ± 0.80 h, total body clearances were 95.5 ± 16.0 and 124.5 ± 11.9 ml h⁻¹ per kg, volumes of distribution at steady state were 0.72 ± 0.08 and 0.87 ± 0.14 litre kg⁻¹ and the litre kg⁻¹. There was no significant difference in any pharmacokinetic parameter between female and male camels. Pharmacodynamic effects were evaluated by measuring endogenous plasma cortisol, circulating lymphocytes and neutrophils numbers and were analysed using indirect pharmacokinetic/pharmacodynamic models. The estimated IC₅₀ of (0.05 mg kg⁻¹ body weight) of dexamethasone for cortisol and lymphocytes for female and male camels were 3.74 ± 0.99 and 2.28 ± 1.09 and 2.63 ± 0.71 and 2.41 ± 0.79 ng ml⁻¹, respectively. The EC₅₀ for neutrophils for female and male camels were 24.5 ± 5.83 and 20.2 ± 3.82 ng ml⁻¹, respectively. There was no significant difference in any pharmacodynamic parameter between female and male camels. Dexamethasone in urine could be detected for 4 - 5 days by enzyme-linked immunosorbent assay and for 3-4 days by liquid chromatography/mass spectrometry after an intravenous dose of 0.05 mg kg⁻¹ body weight.

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Research in Veterinary Science (2004) 77(1): 73-81.