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# CAMEL CONTAGIOUS ECTHYMA (PUSTULAR DERMATITIS)

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*Summary.* — In 1979, pustular dermatitis caused by a virus belonging to the family *Poxviridae*, genus *Parapoxvirus*, was observed among camels in some areas of Mongolia. The morbidity in adults ranged from 10 to 80%, of 2 to 3-month-old suckling camels between 50—70% and it reached 100% in 1-year-old animals. In the beginning, pustules developed around the mouth followed by papular elevations and scab formation. The virus designated camel contagious ecthyma (CCE) grew on the chorionallantoic membrane (CAM) of 11-day-old chick embryos (CE). The disease was transferred to the scarified skin of 2 to 3-month-old camels. Vaccination with the material containing CCE virus seemed to be promising. In contrast, camels were not protected after immunization with vaccinia virus and with a vaccine against sheep and goat contagious ecthyma.

*Key words*: *Parapoxvirus*; *pustular dermatitis*; *camel*; *contagious ecthyma*

## *Introduction*

The camel contagious ecthyma (CCE) is a rarely studied disease and descriptions of this nosologic entity are infrequent in the literature. To our knowledge, the disease was first recognized in 1967 in the district of Mangistansk (Guryev region) in the Kazakh Soviet Republic of U.S.S.R. (Buchnev *et al.*, 1969). Long time ago, it had been known among local population under the name "auzdik". The viral aetiology of the disease was described by Tulepbayev (1969). The morphological similarity of the "auzdik" agent to the sheep contagious ecthyma virus was described by Roslyakov (1972). The new virus was characterized by Khokhoo (1982). Long ago a camelpox-like disease, known under the local name "amru" has been occasionally met also in Mongolia. The disease had a self-limiting course and caused no

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greater harm to the herds of camels. By the end of 70ties and in the be beginning of the 80ties, the disease — according to its clinical signs clearly distinct from camelpox — has occurred in wide regions of south and southwest Mongolia. It showed high contagiosity and involved the animal herds considerably. The recent communication deals with the aetiology, distribution and clinical course of this disease and describes the efforts of introducing immunoprophylactic measures under laboratory as well as natural conditions.

## *Materials and Methods*

*Sampling of the material.* The clinical course of the disease was followed under harsh natural conditions in working herds of camels in cooperative farms of the Gobi-Altay region. More than 20 herds numbering about 3500 animals were inspected. For laboratory examinations, the papular elevations and scabs were collected from animals of different age categories either at the onset of disease or at the culmination of clinical symptoms. The material was shipped in 50% neutral glycerine at the temperature of —6 to —14 °C.

*Virus isolation.* The 10% suspensions prepared from papular elevations or scabs with physiological saline pH 7.3 were inoculated into the CAM of CE. The haemagglutination reaction was made in micro- and macromodification in wells of plastic dishes using 0.5%, 1% and 2% suspensions of sheep, guinea pig and cock erythrocytes, respectively. In addition, the suspensions were inoculated into primocultures of calf kidney cells (CKC), bull testicle cells (BTC), CE fibroblasts, and into stable lines of HeLa and MDBK cells. The virus was inoculated into the scarified skin of 65 camels and 20 lambs.

*Electron microscopic examinations.* The specimens from the skin of diseased camels and those from the infected CAM were examined by electron microscopy (Tesla BS 513). The material was contrasted with phosphotungstic acid.

*Immunological studies.* Three groups of camels were immunized with the following antigens: 1) scab homogenates stored in 50% glycerine and diluted 1 : 30 in physiological saline, 2) vaccinia virus (lyophilized), purchased from England., 3) dermal vaccine to sheep and goat contagious ecthyma (Biokombinat Sinkinsk, Mongolia) (1 ampulla contained 100 sheep doses in 10 ml of 50% glycerine mixture). The preparations were inoculated in 0.1—0.3 ml vol representing either 5 children's or 2—3 sheep doses. Each group (6 camels) consisted of 2 females, 2 young animals (1-year-old) and 2 suckling ones in the age of 2 to 3 months. One half of the animals received the vaccines into the scarified skin of submandibular area, the second half into the scarified skin of the lower neck region anterior to the shoulder bone.

*Testing of the specificity of immunization* was made under laboratory conditions using immunized animals for challenge. Under natural conditions, the immunizing effect of the vaccinia virus was checked in 7,000 camels, that of sheep and goat contagious ecthyma vaccine in 11,000 camels and the effect of egg-adapted CCE virus vaccine in 100 camels.

## *Results*

### *Clinical signs*

The disease was registered in March 1979 among camels of some cooperative farms in the Gobi-Altay region. The morbidity of adults was 10 to 80%, that of 1 to 4-year-old animals up to 100% and in young 2 to 3-month-old camels between 50—70%. The incidence varied in different yards: the number of sick animals was higher in those herds where the disease has not been noted for 20 years. Later on in these yards the 3 to 4-year-old

camels became ill only. Obviously, the lack of disease may be explained by long-lasting immunity developing in adults belonging to endemic herds, among which the 3 to 4-year-old individuals were most susceptible to the epizootic.

The early rash was manifested by small elevations around the mouth. Within 4—12 days, larger papules about 4 mm in diameter formed and became confluent (Fig. 1). Within 2—5 months, scabs developed about 5 to 15 mm thick, occasionally subdivided by manyfold furrows. The scabs did not come off spontaneously, their removal was usually followed by bleeding. Occasionally, the papular elevations changed into papillary, wart-like formations (Fig. 2). The rash was sometimes present also around the nostrils, in the lip corners, on the face and on upper or lower eyelids. Despite of the self-limiting course, the animals were hindered in chewing which resulted in fasting. During its long-term course, the disease was complicated by bacterial infection and in warm season by the bites of flies. All changes described were most prominent in the 1 to 4-year-old camels. Although lethal outcome was rare, death occurred in 0.1—0.6% of animals, mainly due to fatigue and exhaustion by late winter and early spring periods. Healing occurred usually in 3 months. Sometimes scarring remained interfering with the lip movements. Reinfection was never observed within 18—24 months after recovery.

*Aetiology of the disease*

The 10% suspensions of scabs and/or papular elevations were prepared in physiological saline (pH 7.3), extracted for 48 hr and clarified by centrifugation at 12 000 $\times$g. This material did not haemagglutinate the cock, sheep and guinea pig erythrocytes. By electron microscopy the morphology of virus particles resembled to sheep contagious ecthyma virions (Fig. 3), genus *Parapoxvirus*. The CCE virus did not cause lesions in rabbits neither after intracutaneous administration nor after inoculation onto the scarified skin. Guinea pigs and outbred white mice were resistant as well. Oedema, hyperhaemia, thickening of the membrane and greyish-white spots were seen in the first passage on the CAM of 11-day-old CE. In the second passage, CAM was several times thickened on the site of virus inoculation, where greyish-white, transparent round shaped spots 1—3 mm in diameter and elevated by 2—3 mm above the surface were found. The papular elevations and haemorrhages were situated along vessels (Fig. 4). In further passages, these small nodular elevations were seen in groups or single. The intensity of pathological lesions on the CAM remained unchanged from the 6th egg passage.

The CCE virus caused no cytopathic changes either in primocultures or in stabile cell lines listed in Materials and Methods. To confirm the transmissibility of the disease, 2 to 3-month-old camels were infected into the scarified lips with 10% virus suspension from naturally occurring scabs. By day 4, small papules occurred which later became confluent. On days 5 to 7, scabs were formed elevated 3—4 mm above the surrounding skin. Further

changes were characterized by formation of 6—8 mm high papillary elevations with uneven furrowed surface.

Virus titrations were made 14—16 days after experimental infection using collected papules and scabs. The 10-fold dilutions of this material were inoculated into four different areas of the scarified skin of 2-month-old camels. The developed lesions showed no tendency for dissemination. The infectious titre of the tested material was 2.3 log $ID_{50}$/0.1 ml.

This experiment has clearly demonstrated the high contagiosity of camel ecthyma. When the lips were scarified without virus inoculation, the first changes occurred 20—25 days after the natural contact. About 70% camels aged 2—6 months became ill this way. In lambs, CCE virus caused local lesions in one out of 4 scarified areas by 60—70% of infected animals without any generalization. The 2 to 3-month-old camels were also inoculated with goat ecthyma virus. The lesions seen by 4—5 days after virus administration were less evident as compared to those seen in camels inoculated with the CCE virus. The local lesions became more apparent during the next 10 to 14 days. Small papules developed into tiny scabs on some but not all scarification sites. Within 30—35 days the lesions disappeared. The same material, however, caused in lambs typical generalized sheep and goat ecthyma.

*Immunological distinction of CCE virus*

In response to inoculation of CCE virus-containing material, the reaction resembled to natural localized lesions. When the virus was administered into submandibular area of females, the reaction was more prominent than after virus inoculation into the area of shoulders. In young animals no considerable difference was observed between these two zones. Severe confluent postvaccinal reaction was observed in 2 to 3-month-old camels. In these, elevated nodules and scabs had formed but no papillary proliferations were seen. The scabs came off by 20 days post vaccination.

The reaction to vaccinia virus was characterized by occurrence of 8 to 12 papules by 4—5 days after virus inoculation; scabs showed confluency along the scarification line (Fig. 6) and lasted for at least 20 days. The sheep and goat ecthyma virus vaccine caused a slight reaction 5—6 days along the scarification line: only single papules were seen in a 2-month-old camel. The small scabs came off within 12—15 days.

The immunized animals were challenged 14 days later with CCE virus-containing material obtained from experimentally infected camels (CCE virus in its first passage in camels). Non-immunized camels were infected as controls. In adults, the lesions were rarely seen along the scarification line by 4—5 days and they were not found by 24 days. In 1-year-old camels slight scabs, which quickly disappeared, were observed along the scarification line 3—5 days after challenge. In 2 to 3-month-old camels, tiny scabs not wider than 1 mm developed by 3—5 days after challenge. Summing up, no overt disease developed in camels of either age group immunized with the CCE virus vaccine.

In 1-year-old camels immunized with vaccinia virus, typical disease developed by 7—9 days. Similarly, in 2 to 3-month-old camels the disease had a course similar to that in non-vaccinated controls. The disease was relatively mild in those adults only which previously received the vaccinia virus. The clinical course of the lesions in challenged animals showed that vaccinia virus did not efficiently prevented the growth of CCE virus in the skin of camels.

In adult camels immunized with the vaccine against sheep and goat contagious ecthyma, small scabs formed along the scarification line by 5—6 days after challenge and later on quickly disappeared. Similar scabs were seen 7—9 days after challenge of 1-year-old camels; they came off within 24 days. Slight scab formation was observed also in 2 to 3-month-old animals about 5 days after challenge. In the latter, the reaction culminated by 8—9 days and lead to formation of single papular elevations. Thus, camels vaccinated with the sheep and goat contagious ecthyma vaccine were partially protected confirming some antigenic cross-reactivity between CCE virus and sheep contagious ecthyma virus.

*Prophylactic measures*

During the epizootics in 1979 we recommended vaccination with the vaccinia virus. However, this had no effect, and in the course of 1—4 months all vaccinated camels developed signs of camel ecthyma. Later on, we used the lyophilized vaccine against sheep and goat contagious ecthyma in a dose corresponding to 5—6 sheep doses. The latter vaccine had no significant effect either. In contrast, very good results were observed with the egg-adapted strain of CCE virus. Camels receiving this vaccine were resistant to reinfection for at least 6 months and did not acquire the disease when kept together with the sick animals in the same yards.

## *Discussion*

The aetiology of several infections of camels remains still unclear. Especially viral diseases — camelpox and contagious ecthyma — are recently discussed due to the similarity of clinical manifestations, pathological lesions and epizootologic features. In our study we aimed at distinction of CCE as a defined nosologic entity along with the description of properties of the virus.

CCE was first noticed in M.P.R. in May 1979. Since that it spread to several regions in south and southwest of the country showing all epizootological features. The clinical symptoms of the disease were similar to those described by Tulepbayev (1969) in Soviet Kazakhstan under the name "auzdik", which was shown to be viral in origin (Buchnev *et al.*, 1969; Roslyakov, 1972). The dermotropic poxvirus showed a morphology similar to sheep and goat contagious ecthyma. Electron microscopic examination of cutaneous lesions from the mouth of sick camels in the above mentioned

regions of Mongolia by inoculation of the scab suspensions into the CAM of 11-day-old CE has shown that according to morphological criteria, the virus resembled to viruses of genus *Parapoxvirus*, family *Poxviridae*.

CCE virus 320 × 160 μm in size is the smallest poxvirus described to now. According to its biological properties, it can be regarded for a distinct species — contagious ecthyma virus of camels — *Parapoxvirus camelli*. CCE differs from goat and sheep ecthyma virus not only by its size, but also by its host spectrum and in part by its antigenic structure. In cross-protection tests, both viruses showed a slight degree of reactivity. In practice, only homologous parapoxvirus has proved suitable for immunoprophylaxis of the disease. It seems that the vaccine can be prepared by adaptation of CCE to CE. The virus isolated by Roslyakov (1972) during the epizootics in Kazakhstan (U.S.S.R.) was of different size. However, it is difficult to assess, whether they differ in other properties, because no direct comparison was made. Rabbits are resistant to CCE when inoculated into the scarified cornea or skin, which also confirms the species specificity of the virus. In contrast, both vaccinia and camelpox (subfamily *Orthopoxviridae*) cause local changes when administered to the scarified squamous epithelium in rabbits. This test can be used to distinguish CCE from camelpox virus.

Because of similar clinical symptoms of CCE and camelpox, it is important to use rapid tests enabling the diagnosis in the beginning of the epizootics. In addition to inoculation of rabbits, it is important to examine the skin lesions by electron microscope and to inoculate the virus into the CE chorioallantoic membrane. CCE and camelpox have a quite distinct ultrastructure, they differ in haemagglutination activity, in host specificity and pathogenicity, in their ability to grow on the CAM of CE and in cell cultures as well as in their antigenic composition.

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*Explanation of Figures* (Plates XXI—XXIII):

*Fig. 1.* Confluent papules (days 14—45 of the disease).
*Fig. 2.* Formation of multiple wart-like elevations and initial nodules or papules (2—5 months).
*Fig. 3.* CCE virion, contrasted with phosphotungstic acid, × 120,000.
*Fig. 4.* Changes on the chorionallantoic membrane of 11-day-old CE inoculated with CCE virus.
*Fig. 5.* Clinical features of CCE in experimentally infected 2-month-old camel.
*Fig. 6.* Postvaccinal reaction 14 days after inoculation with vaccinia virus.