The First Outbreak of Camelpox in Syria

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ABSTRACT. In this study we report the first outbreak of camelpox in two provinces in Syria. Clinical symptoms started with fever, salivation and general exanthema. The main features were facial and legs oedema, pustules on the mucosa of the lips and a high rate of abortion. Lesions may also occur on the whole body including scrotum and udder. Specimens were investigated by electron microscopy, virus isolation in cell culture and embryonated eggs and by immunohistochemistry. The causative agent was identified as camelpox virus by polymerase chain reaction and sequencing of the hemagglutinin gene.

KEY WORDS: camel, outbreak, pox virus.

Camelpox occurs in almost every country in which camel husbandry is practised. The disease is endemic and outbreaks have been reported in the Middle East (Bahrain, Iran, Iraq, Oman, Saudi Arabia, United Arab Emirates and Yemen), in Asia (Afghanistan and Pakistan), in Africa (Algeria, Egypt, Ethiopia, Kenya, Mauritania, Morocco, Niger, Somalia and Sudan) and in the southern parts of Russia and India [1–4, 7, 9, 14–16]. Clinical manifestations of camelpox range from unapparent and mild local infections confined to the skin to moderate and severe systemic infections [2]. The disease is characterised by fever, enlarged lymph nodes and skin lesions [17]. Lesions appear 1–3 days after the onset of fever, starting as erythematous macules, developing into papules, vesicles, and pustules later turning into crusts. Lesions first appear on the head, eyelids and nostrils and the whole head may be swollen. Later, skin lesions may extend to the neck, limbs, genitalia, mammary glands and perineum. In the generalised form, pox lesions may cover the entire body [11, 17]. Skin lesions may take up to 4–6 weeks to heal. In the systemic form, pox lesions can be found in the mucous membranes of the mouth, respiratory and digestive tract. The morbidity rate of camelpox is variable and depends on whether the virus is circulating in the herd [17]. Transmission is by either direct contact or indirect infection via contaminated environment. Virus is secreted in milk, saliva, and ocular and nasal discharges [13]. The role of an arthropod vector in the transmission of the disease has been suspected. Camelpox virus belongs to the genus orthopoxvirus of the family Poxviridae [16, 14] and is indistinguishable from the prototypic vaccinia virus with respect to size, shape, structure, physico-chemical properties and replication [8] Transmission to humans is very rare but has been reported in non-vaccinated humans in Somalia [10]. Here we report the first outbreak of camelpox in Syria which occurred in the provinces Hama and Duma.

In spring and summer of 2005, blood samples and specimens of vesicles and pustules from affected camels were collected, in addition to crusts and scabs from recovered camels. Clinical findings and post mortem changes were recorded to trace the source of the infection. Skin biopsy samples were homogenized with the FastPrepTM System (Q-BIOgene, Hilden, Germany). Briefly, about 100 mg of the biopsy sample were placed into a 1.5 ml screw-cap tube containing a ceramic medium. After addition of 0.9 ml minimal essential medium (MEM) with antibiotics (500 unit/ml penicillin G sodium, 500 µg/ml streptomycin sulfate, and 1.25 µg/ml amphotericin B) the tube was shaken vigorously for 20s in the FastPrep instrument. After a brief centrifugation step (1 min at 1,000 × g), the supernatant was used for further analysis. Homogenized supernatants were used for electron microscopic inspection. Negative counter staining was performed using standard procedures. Total DNA was directly isolated from 100 µl of supernatant of the homogenized skin biopsy using the Magna Pure Compact System (Roche, Mannheim, Germany) according to the manufacturer’s instructions.

A polymerase chain reaction (PCR), spanning the entire coding sequence of the hemagglutinin (HA) gene was performed as described [5]. Prior to sequencing amplicons were purified using the QIAquick PCR Purification Kit (Qiagen, Hilden, Germany). Both strands of the PCR amplicons were sequenced using the same primers as for.

The following clinical findings were observed: Infected camels showed exhaustion in the whole body with fever and salivation. This was accompanied with loss of appetite and subsequent loss of weight. Vesicles and pustule formation were present on the lips, nose, eyelids, hard palate, dental pad, udder, the inner surface of the thigh and on other mucous membrane. In severe cases pustules were spread all over the body including the genital and anal regions. Facial and leg oedema were often seen in infected camels. Pregnant females showed high percentage of abortion (80–90%). Most of the infected camels suffered from nasal discharge and pneumonitis. Some infected camels died due to the complications of secondary infections. In general, young calves and pregnant females were severely infected; the
morbidity ranged between 30–90% and the mortality was about 1–15% (Table 1). Some camels recovered very quickly whereas in other cases it took more than 3 weeks to recover. The source of the infection was traced to camel herds which were grazing in the desert east of the suburbs of Damascus (province of Duma). Subsequently transportation of infected animals introduced the agent to the province of Hama.

Coverslips with Vero cells displayed a plaque-type cytopathic effect (CPE) within 4–6 days in the first passage and within 2–3 days in the second and third passages which led to isolation of camelpox virus strain CP-Syria. The distinctive features of the CPE were foci of rounded cells, cell detachment, and the formation of massive numbers of large syncytia (data not shown). The large syncytia showed eventually fragmentation and resulted in rounding of cells with pyknotic nuclei. Chorioallantoic membrane (CAM s) were harvested on day 6 post inoculation and characteristic dense, grayish-white pocks were present (data not shown).

Table 1. Data collected from herds in two provinces

<table>
<thead>
<tr>
<th>Herds</th>
<th>Total animals</th>
<th>Camels with symptoms</th>
<th>Aborted/Pregnant Abortion%</th>
<th>Morbidity-Mortality</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dumal females</td>
<td>98</td>
<td>60</td>
<td>30/26</td>
<td>61%–7%</td>
</tr>
<tr>
<td>Duma2 mixed</td>
<td>136</td>
<td>50</td>
<td>24/22</td>
<td>91%</td>
</tr>
<tr>
<td>Duma3 mixed</td>
<td>180</td>
<td>90</td>
<td>36/32</td>
<td>15%–50%</td>
</tr>
<tr>
<td>Duma4 males</td>
<td>67</td>
<td>19</td>
<td>–</td>
<td>30%–1%</td>
</tr>
<tr>
<td>Hama1 mixed</td>
<td>79</td>
<td>40</td>
<td>10/8</td>
<td>50%–12%</td>
</tr>
<tr>
<td>Hama 2 mixed</td>
<td>210</td>
<td>134</td>
<td>28/25</td>
<td>63%–14%</td>
</tr>
<tr>
<td>Hama 3 mixed</td>
<td>105</td>
<td>96</td>
<td>31/27</td>
<td>11%–90%</td>
</tr>
</tbody>
</table>

Inspection of homogenized scabs revealed Orthopoxvirus particles with a typical brick-shaped appearance and irregularly arranged clear tubular surface proteins (Fig. 1).

Template DNA derived from homogenized skin biopsy and from infected Vero tissue cultures by hemagglutinin (HA)–specific PCR led to amplification of a product with the expected size of about 1100 bp (Fig. 2). Sequencing of the amplicon and subsequent data analysis resulted in identification of the hemagglutinin open reading frame (ORF) which accounted for 948 bp (the sequence has been submitted to GeneBank with the accession number DQ853384). The HA ORF was compared to sequences of other Orthopoxviruses published in GeneBank. At the nucleotide level the sequence obtained from the skin biopsy was 100% identical to camelpox virus strain Somalia and differed in one nucleotide only as compared to the HA sequence of camelpox virus strain Saudi Arabia.

In this study we investigated an outbreak of camelpox. Camelpox can be diagnosed by clinical symptoms in combination with PCR, electron microscopy. Our findings resemble those studies which reported camelpox infections in
various countries [1, 2, 6, 7, 11–13, 17, 18]. However, camelpox virus is host-specific and intradermal inoculation of the virus into cattle, sheep, goats, rabbits, guinea pigs, rats, hamsters and mice were no successful [13]. Our observations revealed that sheep and cows with directed contact with infected camels were healthy and had no signs of infection or sickness. Moreover, there was no evidence of any signs of human infections of those people who took care of their infected camels. The eradication of camelpox infection in camel husbandry is of great importance by developing attenuated proactive vaccines. Attempts to produce an attenuated camelpox vaccine have been undertaken in Morocco [6], in Saudi Arabia [9], and in the United Arab Emirates [18] an attenuated live vaccine has been used to protect camels from infection.

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REFERENCES