SOME BIOLOGICAL PROPERTIES OF CAMELPOX VIRUS ISOLATED FROM CAMELS (CAMELUS DROMEDARIUS) IN KENYA

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Summary

Camelpox virus was isolated from two outbreaks of camelpox. In Turkana camel calves were involved, while in Samburu both adults and camel calves were affected. The lesions were much more severe in adults in which there were pox lesions on the mouth, lips and muzzle and also neck oedema and lacrimation. All the viral isolates had the same characteristic plaque morphology in embryonated eggs and sheep kidney cells. The virus was shown to be infective in camels producing typical lesions that progressed through the stages of papule, pastule, scab and peeling off. In some cases, there was secondary bacterial infection associated with haemolytic staphylococcus aureus. Since the camelpox isolate caused a mild rather than a generalised infection it is a potential candidate for vaccine production.

Introduction

Camelpox is a systemic disease with a cyclic pattern characterised by a pox exanthema over the entire body. It was first described in Punjab, India (7), and the causal poxvirus has since been isolated in Iraq (3, 9), Russia, (6), Somalia (5), and in Kenya (1).

The disease mostly affects young camels two to three years of age and herd outbreaks are often associated with the stress of weaning or poor nutrition (8).

Calf mortality is one of the main problems in camel production and can at times reach fifty per cent (12). Although the role of disease is poorly understood, it is known that camelpox is the most serious viral infection of camels (14). The spread of the virus is by contact and within a few days of infection pustules appear on the skin of lips, nose, eyelids, legs and other parts of the body. Corneal opacity and facial oedema occur (11). Death is a sequel to generalised infection and secondary bacterial infection.

In Kenya, the virus was isolated and biochemical characteristics of the virus studied (1). The virus does not agglutinate chick erythrocytes; is not sensitive to ether but is sensitive to chloroform, and does not produce a dermal response in rabbits or sheep after intradermal inoculation. Serological evidence from virus neutralisation indicated that the condition is enzootic in Kenya, and it was found in five out of six herds of camels reared in the nomadic areas of Kenya (2). An ELISA test also demonstrated the presence of antibodies in camels reared in Kenya and neighbouring countries such as Sudan and Somalia (10). The biological characteristics and pathogenic potential in camels were not studied. At the moment, there is no available camelpox vaccine in production although there are current efforts to produce one (4). The clinical disease has not been described in Kenya although it is known that there are camelpox virus strains with variable pathogenicity even in the same region, while the mortality rates may range from 10 to 50% (5).

In this study, the clinical presentation in Kenya and the plaque morphology of the virus in embryonated eggs and tissue culture were studied. The pathogenic behaviour of the camelpox virus strain from Kenya in camels was also studied.

Materials and Methods

Epidemiology

Two districts, Turkana and Samburu were chosen as they represented two different agro-ecological zones that camels inhabit. Turkana is an arid district receiving less than 500 mm of rainfall per year. About 100,000 camels are kept by Turkana herdsmen in herds ranging from a few to 70 animals per herd. Several distant areas in the district were visited in the dry months of September 1992, and 600 camels in 25 herds were examined. Samburu district is a semi-arid area receiving about 600-750 mm of well distributed rainfall. About 66,000 camels are reared in herds ranging from a few to 70 animals per herd. The district was visited in the dry month of October and 500 camels in 20 herds were examined. Scabs were collected from the different outbreaks and sera obtained from sick camels. Sera was also obtained from apparently healthy animals in both districts.

Electron microscopy

Skin scabs from infected camels were ground and re-suspended in a minimum volume of phosphate buffered saline (pH 7.2). A formvar electron microscope grid was floated on a drop of virus suspension for two minutes, removed and bloteted with the edge of blotting paper, and placed on a drop of sodium phosphotungstate (pH 6.6). After 90 sec, the grid was bloteted, air dried and examined in a Zeiss EM 10 C/R transmission electron microscope operating at 60,000 V.