HORSEPOX

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Keywords: horse; horsepox; grease; vaccinia; Uasin Gishu; molluscum contagiosum

Summary
Classical horsepox is considered to occur only in Europe. The disease is transmitted by direct contact with an infected host or with contaminated grooming equipment. Two main forms of classical horsepox are recognised: the buccal form and the ‘grease’ form. A poxvirus infection of horses (Uasin Gishu) is also recognised in Africa, and poxvirus infection has been linked with a disease resembling molluscum contagiosum in North America.

Introduction
The poxviruses are a large group of DNA viruses that include Orthopoxvirus (cowpox and vaccinia), Capripoxvirus (sheep-pox, goatpox, bovine lumpy skin disease), Suipoxvirus (swinepox), Parapoxvirus (pseudo-cowpox, bovine papular stomatitis, contagious viral pustular dermatitis) and Molluscipoxvirus (molluscum contagiosum) (Scott and Miller 2003). Horsepox is a rare dermatological viral disease that can have several forms, but that is classically characterised by lesions in and around the mouth or on the legs. The horsepox virus is an epitheliotrophic, unclassified DNA poxvirus, similar to vaccinia virus and cowpox virus; indeed it is possible that horsepox and vaccinia are the same virus. There are few well-documented reports of poxvirus infection in horses, and classical horsepox is considered to occur only in Europe. However, a poxvirus infection of horses (Uasin Gishu) is also recognised in Africa, and poxvirus infection has been linked with a disease resembling molluscum contagiosum in North America.

In the past in Europe, when vaccination against smallpox was being carried out, horses could become infected by vaccinia virus from recently vaccinated human subjects (De Jong 1917; Munz and Dumbell 2004). The disease is currently much less common in Europe than it was in the first half of the 20th century (Timoney et al. 1988) following the eradication of smallpox.

The virus gains access to the body by the respiratory tract or via the skin (Timoney et al. 1988). A viraemia disseminates the virus back to the skin and other target organs. The virus causes degenerative changes in the epithelium as a result of virus replication and results in the development of vesicular lesions. Degenerative changes in the dermis or subcutis may result from ischaemia secondary to vascular damage. The virus also causes epithelial hyperplasia by stimulating host cell DNA synthesis before the onset of cytoplasmic virus-related DNA replication. Two poxviruses antigenically similar to vaccinia were isolated from horses with natural infections in Kenya (Kaminjolo et al. 1974).

Skin lesions typically begin as erythematous macules, which then become papular and then vesicular (Scott and Miller 2003). The vesicular stage is well-developed in some pox infections, but transient or nonexistent in others. Vesicles develop into umbilicated pustules with a depressed centre and a raised erythematous border. This lesion is the so-called pock. The pustules rupture and form a crust. Healed lesions may leave a scar.

Clinical features
Horsepox may affect horses of any age. The disease is transmitted by direct contact with an infected host or with contaminated grooming equipment.

Two main forms of classical horsepox are recognised: the buccal form and the ‘grease’ form. In the buccal form (which is considered to be the more
important), multiple small pox-like lesions develop on the inside of the lips and the opposing surfaces of the gums, on the frenulum of the tongue, and on the inside of the cheeks. The lesions begin as papules, change to vesicles and then become pustules. The animal may have a fever, and young horses may become systemically sick and die (Timoney et al. 1988). Affected animals are often inappetent, and saliva drools from the corners of the mouth. The horse may play and dip its mouth in water. Lesions may also develop on the skin of the lips, eyelids, face, trunk and in the nasal passages (Jayo et al. 1986). Virus isolated from lesions of horses will infect cattle, and virus from cattle will also infect horses (Timoney et al. 1988).

In the ‘grease heel’ form (also known as ‘grease’ or ‘greasy heel’), papular eruptions develop on the flexor surface of the pastern region. The papules change to vesicles, then to pustules, which finally dry up and form crusts. There may be associated pain and lameness, but affected horses are not systemically ill.

In Africa, a verrucous or papillomatous type of horsepox called Uasin Gishu disease has been recognised (Daubney 1934; Kaminjolo et al. 1974a,b; Kaminjolo and Winquist 1975). The virus(es) causing this condition are not well-determined, but it is possible that it is another manifestation of vaccinia infection (Scott and Miller 2003). The lesions can be generalised but occur mainly on the neck, face, back, flank and hindquarters. Early lesions are small nodules covered by tufts of hair, which are covered by powdery white scab-like material. These scabs detach leaving bleeding patches. When the affected parts lose their hair covering, large raised areas of skin resembling papillomas up to 20 mm in diameter are left.

Another similar disease was also described in North America and Australia in the 1940s to the 1960s, termed viral papular dermatitis (McIntyre 1948; Hutchins 1960). Comparison of the clinical, histopathological and virological features of Uasin Gishu and viral papular dermatitis, along with experimental vaccinia infections in horses suggests that these conditions are probably the same disease (Studdert 1989; Taylor 1993).

Pox viruses have also been demonstrated in skin lesions resembling a human disease called molluscum contagiosum. Molluscum contagiosum is a mildly contagious, cutaneous poxvirus infection (caused by molluscipox virus) of man characterised by small, waxy, firm papules occurring principally on the face, trunk and genital region. ‘Molluscum bodies’, brightly eosinophilic, dyskeratotic keratinocytes containing intracytoplasmic pox virions, are considered pathognomic for this disease (Raheley and Mueller 1983). A similar disease (with similar lesion morphology) has been reported in a small number of horses involving multiple small papules on the cutaneous surface of the prepuce, the penis, neck, thorax, mammary glands and the muzzle (Gribble 1980; Raheley and Mueller 1983; Cooley et al. 1987; Lange et al. 1991; van Rensburg et al. 1991). The papules are typically 2–3 mm in diameter, dome-shaped with a smooth hypopigmented or slightly roughened surface. Affected horses are generally systemically well, but in one reported case, widespread lesions of molluscum contagiosum were identified in association with granulomatous enteritis (Cooley et al. 1987); the authors of this report suggested that the widespread lesions in this horse may have been associated with immunosuppression. Mature virions with a typical pox virus morphology have been found in the keratinocytes of the stratum spinosum and stratum granulosum in this condition. On the basis of very close homology of their viral DNA sequences, the viruses of equine and human molluscum contagiosum are considered to be either identical or very closely related (Thompson et al. 1998), and it has been suggested that the disease may have been transmitted from man to the horse.

Diagnosis

The clinical features plus the presence of large, eosinophilic, intracytoplasmic pox inclusions in vacuolated keratinocytes are considered pathognomic for this disease. The virus may be demonstrated by electron microscopy. Definitive identification of the specific poxvirus requires viral isolation and its identification by serological and immunofluorescence techniques (Scott and Miller 2003).

Immunity

Recovery from horsepox leaves considerable immunity (Timoney et al. 1988). The bovine and equine diseases reciprocally immunise against each other, and both will infect people who have not been vaccinated against smallpox.
Treatment

Treatment is symptomatic, including supportive care and prevention of secondary bacterial infections (including topical treatment with antibacterial shampoos). Mildly affected horses usually recover in 2–4 weeks.

No reported treatment has been successful for molluscum contagiosum. Horses may remain affected by multiple lesions for many months to years. Some of the lesions may regress with time, but complete resolution is unlikely.

References


