Maculopapular lesions in the Central African Republic

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See Online for webappendix

In June, 2010, two boys aged 14 years and 15 years were found to have diffuse maculopapular lesions associated with pustules and crusts (figure). They had been living in a settlement in the deep forest area of the southern Central African Republic (CAR). The cases were reported to the Institut Pasteur at Bangui. The lesions developed after the boys had hunted and eaten a wild rodent (*bemba*). They both presented to a health centre 1 week after eruption of the extensive lesions, which covered the face, torso, and limbs. Both boys were systemically well, with no fever or notable lymphadenopathy.

Infection with monkeypox virus was suspected. The differential diagnoses considered were staphylococcal skin infection, chickenpox, and cutaneous syphilis. Our patients were tested for syphilis; one had a positive result and received treatment with benzathine benzylpenicillin IM 2·4 MIU as single dose. Both were also treated with cloxacillin for a possible staphylococcal skin infection. Laboratory investigations were done on serum and scabs and on a biopsy sample from the brain of a young mouse that had been inoculated 5 days earlier with scab material from the lesions. Monkeypox virus was detected by quantitative PCR based on partial haemagglutinin gene and identified by sequencing.[1] The phylogenetic tree (see webappendix) showed that the strain was identical to the Democratic Republic of the Congo (DRC, formerly Zaire) strain circulating in central Africa. This reported strain was identical to one associated with an outbreak of monkeypox in 2001 in a Bantu family living on the border between the CAR and the DRC, 480 km from the cases reported here. After eating a dead monkey, four members of one family showed typical skin lesions. Infection with monkeypox virus was identified at the Institut Pasteur of Bangui. The 2001 and 2010 strains were also tested by a resequencing microarray for detection and characterisation of a large panel of viruses and bacteria.[2,3] We were able to detect, in one step (48 h), two sequences of haemagglutinin and DNA polymerase DNA-dependent genes of the viral genome in both samples and to confirm the genotype (DRC clade). Our patients were admitted to the local health centre and kept in isolation until they were no longer infectious. When last seen a week after discharge, in June, 2010, both were well and neither showed any sequelae.

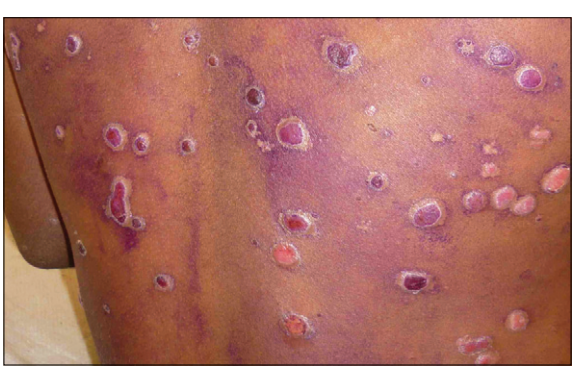

Figure: **Maculopapular lesions in the second of the two cases 1–2 weeks after initial eruption**

MPV has a wide range of hosts, so it can maintain a reservoir in wild animals while sporadically causing human disease,[4] generally in remote villages in the rainforest areas of central and west Africa. Large outbreaks, with transmission between people, occur only in the DRC. The case-fatality rate in Africa is between 1% and 10%. There is no specific treatment or vaccine although smallpox vaccination is 85% effective in preventing monkeypox. In 2003, the first human monkeypox outbreak in the western hemisphere was reported in the USA after importation of rodents from Ghana.[5] We describe the occurrence of two typical cases of monkeypox, caused by the same viral genotype, 10 years after the previous infections with this strain. Although monkeypox is rare, its differentiation from other similar presenting illnesses is important.

Contributors

EW looked after the patient; NB, EN, A-MB, and BS did the experimental studies; and NB, EW, J-CM, AG, and MK analysed the data and wrote the report.

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