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People with asymptomatic or unrecognised infection potentially contribute to monkeypox virus transmission

The ongoing global mpox (formerly known as monkeypox) outbreak has focused attention on methods of virus transmission. Studies published in 2022 have shown monkeypox virus genome detection in multiple body sites from infected patients.^{1,2} Furthermore, multiple PCR-positive samples have been reported in asymptomatic individuals.³ The contribution of people with asymptomatic or subclinical infection to the monkeypox virus transmission chain should be established.

In our laboratory at IRCCS Sacro Cuore Don Calabria Hospital (Verona, Italy), a subclinical infection was detected in a man aged 35 years who was receiving pre-exposure prophylaxis for HIV and attending the outpatient clinic for sexually transmitted diseases at Verona University Hospital; he provided his informed medical consent.

The man had household contact with a different man who was diagnosed with mpox 7 days after contact. On clinical observation, he presented with a single painless ulcer at the base of the penis with a clear base, no hardened edges, no surrounding erythema and oedema, and no local or generalised lymphadenopathy. Ulcer swab was negative to monkeypox-virus-specific

PCR and specific serological tests were negative as well. Common sexually transmitted disease testing gave negative results, except for the presence of *Neisseria gonorrhoeae* DNA in anal swab. Over the following 4 days, the ulcer healed completely; no residual signs or scars were present at the 8-day follow-up visit. The lesion was considered traumatic due to self-reported extensive use of a circular band wrapped around the penis base to enhance sexual performance. Mpox diagnosis was based on seroconversion with specific IgA, IgM, and IgG detection, observed in a day-30 follow-up serum sample (appendix). IgA development, so far not described for mpox, is reported in other human orthopoxvirus infections and might be relevant for diagnosis as its kinetics are usually earlier than IgM and IgG. PCR tests of previously collected samples revealed mpox DNA in anal swab and plasma. Anogenital swabs are reported to be the second highest viral load site in mpox, after skin lesions.¹ Viral load in the anal swab of the patient (PCR cycle threshold 26.8) was consistent with the presence of replication-competent virus.⁴ Moreover, monkeypox virus isolation from a urethral swab of an asymptomatic patient was reported, supporting contagiousness of asymptotically infected people via sexual contacts.³ Therefore, our case could probably transmit the infection. Asymptomatic infection can substantially contribute to the transmission chain and should be clearly addressed in public health policy to contain monkeypox virus transmission. We emphasise the need

to further investigate possible role of asymptomatic or unrecognised infections in transmission chains of monkeypox virus, even in unusual routes.

CC, MC, and ET conceptualised the study design, analysed and discussed data, and wrote the manuscript. CC and ET designed the study. SA and EP collected samples and did diagnostic tests. MC enrolled patients and collected and managed clinical data. All authors read, revised, and approved the manuscript. We thank Maria Rosaria Capobianchi (IRCCS Sacro Cuore Don Calabria Hospital, Verona, Italy) for advice and support. We declare no competing interests.

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See Online for appendix