

Occupational infection with herpes simplex virus type 1 after a needlestick injury

Mark W Douglas,* Jane L Walters,†
Bart J Currie‡

*Registrar, Infectious Diseases, Royal Darwin Hospital (currently at Centre for Virus Research, Westmead Millennium Institute, Westmead, NSW 2145).

† Resident Medical Officer, Royal Darwin Hospital, Casuarina, NT. ‡ Director of Clinical Research, Tropical Medicine and International Health Unit, Menzies School of Health Research, Casuarina, NT.
mark_monique@one.net.au

TO THE EDITOR: A 27-year-old hospital medical officer received a penetrating needlestick injury to her left hand, drawing blood, after using a 22-gauge needle to deroof a vesicle for diagnosis in a two-year-old patient with orolabial herpes simplex virus type 1 (HSV-1). The medical officer had no significant medical history, took no regular medication, and had no previous history of oral or genital herpes.

On Day 4, a vesicle appeared at the site of inoculation, with surrounding erythema. The medical officer first presented on Day 6, by which time the vesicle was crusting over, with several satellite lesions (see Figure). She described mild pain in her left axilla, but no fevers or sweats. A 10-day course of oral famciclovir (250 mg, three times daily) was prescribed and she was restricted from work until the lesions had completely healed (Day 16). During 12 months of follow-up there has been no clinical recurrence.

Specimens from the two-year-old child were positive for HSV-1 by direct immunofluorescence, and HSV-1 DNA was detected by polymerase chain reaction (PCR). Specimens from the medical officer on Day 6 were negative for HSV-1 by direct immunofluorescence, but positive by PCR. There was no evidence of HSV-2 or varicella zoster virus in either specimen.

To our knowledge this is the first reported transmission of HSV-1 after needlestick injury. Herpetic whitlow (HSV of the hands), a well-recognised occupational

Herpes simplex lesion of the palm six days after a needlestick injury



hazard for dentists and anaesthetists, is frequently misdiagnosed, resulting in unnecessary surgical procedures and delayed healing. In healthcare workers, pain and also work restrictions to limit cross-infection reduce productivity. Horizontal transmission can occur in the absence of clinical lesions, but latex gloves are an effective barrier.¹

There are few guidelines available for postexposure prophylaxis for HSV-1, and no controlled clinical trials in humans. The short incubation period and early establishment of latency in HSV infection remain obstacles for effective delivery of postexposure prophylaxis. HSV can establish latent infection of neurones in the absence of peripheral replication.² In an animal model, postexposure treatment with famciclovir or valaciclovir inhibited peripheral replication of HSV, reducing latent infection but not preventing it altogether.² In one case report, a patient who started taking famciclovir within one hour of a needlestick injury did not develop whitlow and remained seronegative for HSV.⁴

Famciclovir and valaciclovir have high oral bioavailability, minimal toxicity and proven efficacy in treating HSV. Available data suggest that treatment with these drugs after documented exposure to HSV reduces the severity of acute disease, limits the

number of neurones infected and may reduce the frequency of subsequent recurrences. If started early enough, postexposure prophylaxis may prevent latent infection altogether.

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Serotonin toxicity with therapeutic doses of dexamphetamine and venlafaxine

Felicity H Prior,* Geoffrey K Isbister,†
Andrew H Dawson,‡ Ian M Whyte§

* Director, Hunter Drug Information Service, Newcastle Mater Misericordiae Hospital, NSW; † Clinical Lecturer and Toxicologist, ‡ Associate Professor, and Specialist; § Associate Professor, and Specialist, Discipline of Clinical Pharmacology, University of Newcastle, and Department of Clinical Toxicology and Pharmacology, Newcastle Mater Misericordiae Hospital, Bag 7, Hunter Region Mail Centre, NSW 2310.
gsbite@bigpond.com

TO THE EDITOR: We report two episodes of serotonin toxicity (or serotonin syndrome) caused by drug interaction in one individual chronically treated with dexamphetamine. The interacting drugs were venlafaxine, then later citalopram. We are not aware of any previous reports of serotonin toxicity caused by dexamphetamine in combination with either venlafaxine or any selective serotonin reuptake inhibitor (SSRI).

A 32-year-old man presented after two days of marked agitation, anxiety, shivering and tremor. He was being treated with dexamphetamine, 5 mg three times daily, for adult attention deficit hyperactivity disorder. He had started venlafaxine (75 mg daily) two weeks previously, and this had been increased to 150 mg daily after a week. On examination, he was alert and oriented, but diaphoretic, shivering and had fine motor tremor. His heart rate was 140 bpm, blood pressure was 142/93 mmHg and temperature 37.3°C. Pupils were 3 mm diameter and reactive, with no nystagmus or ocular clonus. There was generalised hypertonia, hyperreflexia, 1-2

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