Varicella zoster virus infection causing urinary retention in a child with HIV infection

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Varicella zoster virus (VZV) of the human herpes virus family causes childhood chickenpox, becomes latent in sensory ganglia and re-activates years later in immunocompromised and elderly persons to produce shingles (herpes zoster). The annual incidence of herpes zoster in children aged <10 years is reported to be 0.74 per 1 000 children per year. The association of VZV infection and neurogenic bladder dysfunction is rare and mostly seen in adults, with only one reported case in a child.

Severe and debilitating zoster-associated dermatological, ophthalmological and neurological complications may occur in patients with HIV infection. We describe the case of an HIV-positive child who presented with acute urinary retention secondary to VZV infection.

Case description
An 11-year-old boy receiving antiretroviral therapy for HIV infection and antibacterial therapy for pulmonary tuberculosis presented with urinary retention due to varicella zoster virus infection involving the sacral nerves, confirmed on serological testing. The perineum over dermatomes S2 - S4 on the left was involved with a vesicular and superficially erosive rash. A transurethral catheter was inserted and the patient was treated with acyclovir (300 mg 6-hourly for 5 days). At follow-up 4 weeks later, the perineal skin lesions had healed, the catheter was removed and the patient was able to pass urine.

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Fig. 1. Blistering and superficially erosive skin lesions due to varicella zoster virus infection involving the sacral nerves (S2 - S4) on the left side.

An 8F Foley catheter was inserted transurethrally and 1 500 ml of clear urine was drained. The boy was admitted to hospital and treated with 300 mg acyclovir 6-hourly (intravenous), 4 drops of oral tilidine 6-hourly and 1 000 mg paracetamol 8-hourly.

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The incidence of herpes zoster in aged 2 - 14 years in South Africa is approximately 2.5%. The incidence of herpes zoster in children aged <10 years is approximately 0.74 per 1 000 per year. This incidence is higher in HIV-positive children (164 per 1 000 per year) and possibly even higher in children with a low CD4 count. Bladder dysfunction secondary to herpes zoster is uncommon, affecting 3.5 - 4.2% of people with VZV infection, but occurs more often when the lumbosacral dermatomes are involved (28.6%).

Voiding dysfunction caused by herpes zoster may be classified as cystitis-associated, neuritis-associated, or myelitis-associated. Neuritis-associated dysfunction leads to an acontractile bladder and hypoesthesia. In cystitis-associated bladder dysfunction, the neurological examination is normal, whereas overflow incontinence and neurological abnormalities occur with myelitis-associated dysfunction, according to the level of spinal involvement. It is important not to ascribe urinary retention to the pain of genital ulceration.

The prognosis is favourable with acyclovir therapy and intermittent or indwelling catheterisation. The usual time to recovery of voiding function is 8 weeks. Antiviral therapy decreases the duration and number of vesicles, but there is no evidence that it reduces the incidence of neuropathic bladder dysfunction. It is uncertain whether starting acyclovir therapy after the vesicles have formed alters the outcome.

Viruses associated with neurological complications that affect bladder function are HSV types 1 and 2 (most common), VZV, cytomegalovirus and Epstein-Barr virus. Radiculomyelitis causing transient urinary retention and sensory lumbosacral symptoms is known as Elsberg syndrome.

The most common diagnostic pitfall with VZV is its confusion with HSV infection. HSV lesions may appear in a dermatomal pattern, especially when involving the thighs or buttocks. The major difference between the two diseases (when HSV occurs in belt-like patterns) is the significantly higher re-activation frequency of HSV.

Laboratory tests may be required to differentiate HSV from VZV. A definitive diagnosis is made by isolation of the virus in cell cultures inoculated with body fluids. Polymerase chain reaction techniques may be used to detect viral DNA in the cerebrospinal fluid. Heterologous antibody responses to HSV and VZV may occur in some patients because the two viruses share common antigens.

In our patient, the clinical picture was in keeping with VZV rather than HSV infection, and the serological tests were compatible with a diagnosis of acute VZV infection.

**Discussion**

The prevalence of HIV infection in children aged 2 - 14 years in South Africa is approximately 2.5%. The incidence of herpes zoster in children aged <10 years is approximately 0.74 per 1 000 per year. This incidence is higher in HIV-positive children (164 per 1 000 per year) and possibly even higher in children with a low CD4 count. Bladder dysfunction secondary to herpes zoster is uncommon, affecting 3.5 - 4.2% of people with VZV infection, but occurs more often when the lumbosacral dermatomes are involved (28.6%). Voiding dysfunction caused by herpes zoster may be classified as cystitis-associated, neuritis-associated, or myelitis-associated. Neuritis-associated dysfunction leads to an acontractile bladder and hypoesthesia. In cystitis-associated bladder dysfunction, the neurological examination is normal, whereas overflow incontinence and neurological abnormalities occur with myelitis-associated dysfunction, according to the level of spinal involvement. It is important not to ascribe urinary retention to the pain of genital ulceration.

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Acknowledgement. Written informed consent was obtained from the patient's mother to take clinical photographs of the perineal skin lesions at presentation and follow-up.