



## CASE REPORT

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

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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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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.<sup>1</sup> The association of VZV infection and neurogenic bladder dysfunction is rare and mostly seen in adults, with only one reported case in a child.<sup>2</sup>

Severe and debilitating zoster-associated dermatological, ophthalmological and neurological complications may occur in patients with HIV infection.<sup>3</sup> 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 was referred with urinary retention. He complained of difficulty passing urine, lower abdominal discomfort, and a painful rash over the perineum for 5 days. His mother had noticed that he had lower abdominal swelling.

The patient was HIV-positive and receiving treatment accordingly (300 mg zidovudine twice daily, 250 mg didanosine daily and 200 mg/50 mg lopinavir/ritonavir twice daily). He was also receiving treatment for pulmonary tuberculosis, diagnosed 6 months prior (300 mg rifampicin and 150 mg isoniazid daily). The boy had no previous history of chickenpox.

On examination, the patient was pyrexial (temperature 39°C) and appeared acutely ill. His bladder was distended, easily palpable and mildly tender. His penis, scrotum and perineum over dermatomes S2 - S4 on the left were involved with a vesicular and superficially erosive rash. Severe swelling of the prepuce caused the appearance of phimosis (Fig. 1). On digital rectal examination, his anal tone was normal.

The bulbocavernosus reflex was not tested due to severe tenderness in the perineal area. No abnormalities were found on neurological examination of his lower limbs.



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 transurethraly 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.

Urine dipstick testing showed a trace of blood. Urine microscopy showed leukocytes  $<1\ 000$  cells/ $\text{mm}^3$  and erythrocytes  $<1\ 000$  cells/ $\text{mm}^3$ , and was negative for bacterial cultures. The patient's absolute CD4 count was  $159 \times 10^6/\text{l}$  with a CD4% of lymphocytes of 9.42% and CD45 positive white cell count of  $6.18 \times 10^6/\text{l}$ . Blood tests revealed 136 mmol/l sodium, 4.3 mmol/l potassium, 2.8 mmol/l urea, 32  $\mu\text{mol/l}$  creatinine, a  $5.9 \times 10^9/\text{l}$  white cell count, 11.5 g/dl haemoglobin and  $303 \times 10^9/\text{l}$  platelets.

Serological tests were IgG-positive and IgM-negative for human simplex virus (HSV) types 1 and 2, and IgG-positive and IgM-positive for VZV. Smears for viral culture were negative for HSV 1 and 2 and negative for VZV; however, the smears were taken after the blisters had ruptured and there was already scab formation. Due to the clinical picture and the serology results, treatment for herpes zoster was continued.

The boy was discharged after 7 days with the transurethral catheter *in situ*. At follow-up one week later, the preputial swelling had resolved but phimosis was present due to scarring. Circumcision was performed under general anaesthesia. A trial without catheter was attempted. After 3 hours the bladder was palpable, but he had no urge to urinate. A 12F Foley catheter was re-inserted and 600 ml of urine was drained. At follow-up 2 weeks later, the perineal skin had healed and a trial without catheter was repeated. He was able to urinate 250 ml with a post-void residual volume of 124 ml urine on ultrasound. He appeared well and was urinating without difficulty at last follow-up 2 months later.

## Discussion

The prevalence of HIV infection in children aged 2 - 14 years in South Africa is approximately 2.5%.<sup>4</sup> The incidence of herpes zoster in children aged  $<10$  years is approximately 0.74 per 1 000 per year.<sup>1</sup> 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.<sup>5</sup> 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%).<sup>5</sup>

Voiding dysfunction caused by herpes zoster may be classified as cystitis-associated, neuritis-associated or myelitis-associated.<sup>5</sup> 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.<sup>5</sup>

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 duration of neuropathic bladder dysfunction.<sup>6,7</sup> 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.<sup>8</sup>

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.

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