

Acute urinary retention as a complication of primary varicella-zoster infection of childhood – a second reported case

To the Editor: We discuss the case of a child with acute urinary retention and constipation following primary varicella-zoster infection (chickenpox). To our knowledge, this unusual complication has only been reported once before.¹

An 8-year-old boy presented 2 days after developing urinary retention. Chickenpox had been diagnosed 2 weeks previously and treated with paracetamol and topical calamine lotion. There was no similar preceding history, trauma, use of anti-cholinergic medication, or other urinary or neurological signs or symptoms. The child was HIV-negative. He was fully ambulant, well hydrated and afebrile. His urinary bladder was abdominally palpable and was catheterised at the referring hospital, draining clear urine. The trunk and extremities had healing primary varicella-zoster (chickenpox) skin lesions. No evidence of sacral or perineal shingles rash (secondary varicella-zoster infection) or neurological deficit was found. Urine dipstick, renal function tests, blood electrolytes, full blood count and C-reactive protein were normal. Lumbar puncture and serology for varicella-zoster virus were not done.

After excluding urethral and bladder outlet obstruction, anti-cholinergic use, urinary tract or bladder infection and transverse myelitis, primary varicella-zoster virus-related urinary retention and constipation was tentatively diagnosed. He was admitted and given oral acyclovir (400 mg 8-hourly for 10 days) and laxatives. After 3 doses of acyclovir, the catheter was temporarily removed and the child gradually began to urinate and resumed passing stools. There were no further similar complaints after discharge. Informed consent for publication was obtained from the patient.

The varicella-zoster virus is an exclusively human virus belonging to the Alphaherpesvirinae subfamily of the Herpesviridae.² It is neurotropic and very contagious, and is spread mostly by virus-filled respiratory droplets and, to a lesser degree, from skin lesions.³ Varicella-zoster causes 2 distinct clinical syndromes: chickenpox/varicella, the initial or primary infection of the host; and shingles/zoster, corresponding to reactivation of latent infection.²

In healthy children, chickenpox usually has no prodrome, is self-limiting, and is characterised by a distinct pruritic exanthem (macules progressing to papules and virus-rich vesicles before crusting), malaise and low-grade fever. Treatment is symptomatic.

Once the self-limiting initial infection is contained by the immune system, the virus establishes itself within the spinal cord ganglia (dorsal root/sensory ganglia being the most common site) and becomes latent. Transport to the ganglia is thought to be via retrograde axonal transport (from the skin) and via haematogenous spread.²

Re-activation of dormant varicella-zoster virus occurs when cell-mediated immunity is weakened, allowing viral replication within the infected ganglia. Viral spread along the nerves associated with the affected ganglia causes symptoms associated with nerve dysfunction.³ Re-activation within sensory ganglia results in a painful dermatomal zoster rash whereas signs of re-activation within the autonomic sacral ganglia include urinary retention, zoster cystitis, or anorectal dysfunction.^{2,4} We found 1 report in the English literature of urinary retention complicating primary varicella-zoster infection.¹ Their case differed in having a large primary varicella-zoster vesicle on the glans penis obstructing the urethral meatus.¹ Incision and removal of the vesicle did not resolve the retention, necessitating suprapubic catheterisation and dilatation of a meatal stricture, with

gradual regaining of full micturition control. The authors speculated that the retention was due to combined neurogenic effects of the primary varicella-zoster infection on the sacral ganglia (i.e. the virus establishing itself within the sacral ganglia before becoming latent) and obstruction caused by the vesicle and stricture.¹

Having excluded other pathology, we believe that our patient's presentation was congruent with the hypothesis that, towards the end of primary varicella-zoster infection, virus infecting a specific ganglion (prior to becoming latent) may cause transient neurological effects. As this was the patient's first varicella-zoster infection, we believe that, should infection be re-activated (by old age or immune suppression), he may again develop urinary dysfunction and constipation.

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Responding to the evidence for improved treatment for cryptococcal meningitis in resource-limited settings

To the Editor: The World Health Organization (WHO) issued the first evidence-based treatment guidelines for cryptococcal meningitis in December 2011.¹ Although its incidence has decreased with increased access to antiretroviral therapy, cryptococcal meningitis remains a major cause of death in people with HIV/AIDS, with over 500 000 deaths every year in sub-Saharan Africa. It is a leading cause of death in the Médecins sans Frontières (MSF) HIV/AIDS programmes in Africa.^{2,3}

The preferred treatment in the WHO guidelines combines amphotericin B injectable with oral solid formulations of either flucytosine or fluconazole. The liposomal injectable form of amphotericin B is also indicated as an alternative to conventional amphotericin B because it is associated with fewer side-effects. However, it is acknowledged that this option is currently too expensive for routine use in most countries.¹

Access to fluconazole was a major concern a decade ago, with 100-fold price differences reported in developing countries with the similar gross domestic product.⁴ In South Africa, the drug became an early symbol of the struggle to improve access to affordable treatment for people living with HIV/AIDS. In early 2000 the Treatment Action Campaign imported generic versions of fluconazole from Thailand, in defiance of patent laws at the time. Fluconazole is generally available today, with quality-assured generics costing as little as US\$0.07 per 200 mg capsule.⁵ However, access to the preferred drugs, amphotericin B and flucytosine, is a challenge, as highlighted by a rapid survey of MSF HIV/AIDS programmes in 9 countries. Amphotericin B was available in only 4 countries, and cost between US\$70 and \$170 per patient for a 2-week induction treatment. Data on the registration status, availability and price of flucytosine and