CASE REPORT

Unusual case of acute urinary retention in a young female

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SUMMARY

We report an unusual case of a 17-year-old young female presenting to the emergency department with varicella infection, acute urinary retention (AUR) and no other neurological deficits. An MRI of the spine confirmed the diagnosis of acute transverse myelitis. Positive serum IgG antibodies against varicella zoster virus (VZV) suggested a parainfectious aetiology. The patient eventually developed weakness and a sensory level from the third thoracic dermatome on day 2 of hospitalisation. Awareness that AUR can precede other neurological deficits in VZV transverse myelitis will prevent misdiagnosis and allow for the prompt treatment of this debilitating illness.

BACKGROUND

Varicella zoster virus (VZV)-related transverse myelitis is rare with a frequency of 0.3%.¹ Patients with this condition usually present with sensory or motor deficits. We describe a case of transverse myelitis in a young female with varicella infection presenting to the emergency department with only acute urinary retention (AUR) and no neurological deficits. Awareness that AUR can precede other neurological deficits in VZV transverse myelitis will prevent misdiagnosis and allow for the prompt treatment of this debilitating illness.

CASE PRESENTATION

A 17-year-old young female presented to our emergency department with a 1-day history of suprapubic pain and inability to void her bladder. Three days prior, she had seen a general practitioner and was diagnosed with primary VZV infection and had started oral acyclovir. She denied ingestion of any other medications such as opioids, antihistamines and anticholinergics that could have precipitated the AUR.

Physical examination showed that she had normal vital parameters and was afebrile. Vesicular rashes were noted over the face, neck and trunk. No vesicles were observed on the external genitalia. Both upper limb and lower limb tone, power and reflexes were normal. There was no sensory level detected. Digital rectal examination revealed normal anal tone, perianal sensation and no impacted stools. The bladder was full and distended. A 12-French Foley catheter was inserted and 1200 mL of urine was drained, after which her suprapubic pain resolved. Urinalysis was normal and urine pregnancy test was negative. Following a failed trial of

catheter removal, the patient was admitted to the neurology department on the suspicion of AUR from VZV transverse myelitis.

On day 2 of admission, the patient started to show signs of lower limb weakness distally and a sensory level was noted from the third thoracic dermatome. The inability to void persisted. The sensory level progressed to the bilateral seventh cervical dermatomes on day 3. In the subsequent days, her gait was noted to be broad-based and unsteady.

INVESTIGATIONS

Full blood count, C reactive protein and procalcitonin were normal. VZV IgG was positive. HIV was negative. Cerebrospinal fluid (CSF) analysis showed the following: red blood cells 4 (<1 cell/ μ L), nucleated cells 13 (0–5 cells/ μ L), neutrophils 0%, lymphocytes 91%, monocytes 9%, protein 1.37 (0.10–0.40) and glucose 2.8 mmol/L (2.5–5.5). CSF Gram stain and culture were negative, as was the tetraplex CSF PCR for VZV, herpes simplex virus, cytomegalovirus and toxoplasma gondii.

MRI of the thoracic and lumbar spine showed patchy T2-weighted image changes in the third to eighth thoracic spinal cord level (figure 1), with focal areas of asymmetric hyperintensity affecting the central grey matter of the spinal cord at the levels of the fifth (figure 2) and seventh thoracic spinal cord. There was equivocal mild contrast enhancement in the fifth to seventh thoracic spinal cord level.

DIFFERENTIAL DIAGNOSIS

The aetiology of AUR can be either obstructive or functional.^{2,3} In a young female, obstructive causes include pelvic masses, faecal impaction, pregnancy, imperforate hymen or urogenital abnormalities resulting in the formation of hydrocolpos,⁴ all of which our patient did not have. Functional causes of AUR include inflammatory, pharmacological, infectious or neurological (eg, transverse myelitis, cauda equina) aetiologies.

TREATMENT

The patient was diagnosed with acute transverse myelitis secondary to VZV, and intravenous acyclovir was administered for 14 days. Steroids were not administered as her neurological deficits plateaued and gradually improved.

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Figure 1 Sagittal view of the T2-weighted MRI scan of the thoracic cord showing mild patchy changes from the level of the third to eight thoracic cord.

OUTCOME AND FOLLOW-UP

The acute retention of urine resolved and the bladder catheter was removed on day 8 of admission. She was eventually discharged on completing her 14-day course of intravenous acyclovir with normal gait and resolved neurological deficits.

DISCUSSION

Complications involving the peripheral and central nervous system occur in 0.1%–0.75% of those with VZV

infection.^{1 5} These include encephalitis, meningoencephalitis, aseptic meningitis, Guillain-Barre syndrome, optic neuritis, peripheral neuropathy, facial paralysis, cerebellar ataxia, transverse myelitis, ventriculitis and delayed contralateral haemiparesis.^{1 6} These are rare and even more so in immunocompetent individuals, such as our patient.^{1 5–7}

Transverse myelitis is reported to occur at a rate of 0.3% during or after VZV infection.¹ It has a sudden onset shortly after the appearance of a rash over a specific dermatome associated with paraparesis, sensory loss at the corresponding level as well as a dysfunctional urinary sphincter.^{1 5} However, the diagnosis of VZV transverse myelitis can be challenging. Typically, the detection of VZV antibodies and VZV DNA in CSF is confirmatory, yet studies have shown severe neurological signs even in patients with negative results.⁷ MRI is more sensitive in these circumstances and is likely to show T2-weighted hyperintensity of the spinal cord.^{1 6} In our patient, the discrepancy between the identified thoracic cord level of involvement on MRI done on day 2 and the eventual development of the lower cervical neurological deficits on day 3 are due to the ongoing evolution of the disease.

Most case studies described patients with VZV transverse myelitis presenting initially with sensory and motor deficits. Therefore, it was interesting to note that our patient's initial presentation was solely AUR with no sensory or motor deficits. One study by Hiraga *et al*⁸ looked into the incidence of AUR as a sole presenting complaint for acute myelitis and showed that only 3 out of 32 patients presented with AUR as the sole presenting complaint. Out of these three patients, two were mistakenly referred to urology department first prior to the development of other neurological symptoms. Other studies by Berger *et al* and Pradhan *et al* reveal a prevalence of 1 in 8 and 3 in 12 patients, respectively, who presented with only AUR and no neurological deficits.^{9 10}

The management of VZV transverse myelitis is intravenous antivirals, such as acyclovir, for a period of 10–14 days, titrating according to the patient's symptoms.^{6 11} There is only class IV evidence supporting the use of steroids in the treatment of acute transverse myelitis.¹² Case studies have reported reduced disease activity and faster neurological recovery with the combination of both intravenous acyclovir and high-dose intravenous methylprednisolone, 1 g daily for 3–7 days.^{1 5 11} Unfortunately, no predictable markers for disease progression are available as yet to determine the efficacy of these drugs.⁶

Factors that have shown to influence the prognosis of patients with VZV transverse myelitis include patients' initial presenting features, the rate of progression of their symptoms as well as their immune status.^{13 14} However, it has been noted that urinary symptoms often do not correlate with the resolution of other motor and sensory neurological deficits.

Multiple studies also look specifically into the micturition disturbances in acute transverse myelitis.^{15 16} The majority of patients present with AUR secondary to detrusor hyporeflexia associated with either a sensory level and/or weakness. On follow-up, it was noted that patients with detrusor hyporeflexia eventually developed detrusor hyper-reflexia and/or detrusor sphincter dyssynergia that persist beyond resolution of other neurological deficits. The initial detrusor hyporeflexia can be attributed to spinal shock in the acute phase of spinal cord inflammation. This eventually gives way to bladder spasticity due to anterior horn

Figure 2 Axial view of the T2-weighted MRI scan at the level of the fifth thoracic cord showing areas of asymmetric hyperintensity affecting the central grey matter of the spinal cord (white arrow).

cell excitability and supranuclear pelvic and pudendal nerve dysfunction. Persistent detrusor hyporeflexia was observed in patients with lumbosacral involvement. The persistence of urinary symptoms emphasises possible long-term morbidity despite good prognosis of transverse myelitis. This includes long-term urinary incontinence requiring intermittent catheterisation.

Learning points

- ▶ In the absence of the usual obstructive and functional causes, a high index of suspicion for acute transverse myelitis is required when investigating acute urinary retention (AUR) in patients with varicella zoster virus (VZV) infection.
- ▶ AUR may be the only presenting sign in VZV transverse myelitis.
- ▶ The treatment of VZV transverse myelitis is 10–14 days of antivirals. High-dose corticosteroids are a class IV treatment for this condition.

Contributors All authors contributed to the article equally. KWH conceptualised and designed the content. BZHC, SR and GV conducted research, acquired data and analysed the information acquired. The article was written by BZHC, SR and GV, and was finalised by KWH.

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