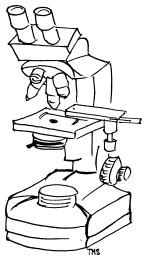

The Creative Clinican

Acute Herpetic Infections Resulting In Acute Urinary Retention In Young Women

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Case#1: A forty three year old woman presented to the emergency room in acute urinary retention. Several days previously, she noted reduction in her urinary stream and progressive difficulty voiding. She had intact sensation of bladder filling but could not voluntarily void. She denied fevers, chills, nausea, vomiting, dysuria, hematuria, urinary incontinence and constipation. She had no neurological complaints. A foley catheter was placed in the emergency department and a residual of approximately one liter was evacuated. Urine culture was sent and a lumbar MRI performed. Urine culture did not grow any bacteria, and her MRI was normal. She was referred to urology for further evaluation. The pertinent finding on physical examination was a pink, vesicular rash which was hypersensitive along the right sacral dermatomes. She noted that this rash had appeared 6 days prior to presentation and was causing an abnormal sensation in the right buttock and right lateral hip. The rash did not extend across the midline. A history of childhood chicken pox was confirmed, and she was diagnosed with acute urinary retention secondary to neurogenic bladder. The clinical diagnosis was sacral herpes zoster virus. She was taught clean intermittent catheterization and started on Famciclovir after consultation with both her neurologist and dermatologist. The patient performed clean intermittent catheterization for approximately three days and then voided spontaneously. Repeat post void residual one week later was 0cc and she had complete resolution of all signs and symptoms of herpes zoster.

Case#2: A thirty one year old woman presented to the emergency department with twenty four hours of urinary retention. Two days previously, she had developed urinary frequency and difficulty voiding. She denied fevers, chills, nausea, vomiting, dysuria, or hematuria. She did have mild suprapubic discomfort. Sensation to void remained intact but she was unable to voluntarily void. A foley catheter was placed and a volume of 500cc was drained from her bladder. Urine analysis was negative and she denied history of recurrent **urinary tract infections (UTIs)**. On questioning, she noted new painful vesicles on the right buttock region close to the anus. She also revealed that she had unprotected anal intercourse with a new partner approximately ten to twelve days prior to the appearance of the vesicles. A diagnosis of acute herpes simplex virus was made and the patient was educated on clean intermittent catheterization and started on oral Famciclovir therapy. Testing for other sexually transmitted diseases was performed, and she was referred to an infectious disease specialist. The vesicles resolved several days after initiation of therapy and she voided spontaneously after three weeks of clean intermittent catheterization.

Acute urinary retention is a common urologic problem. The vast majority of patients are men with **benign prostatic hyperplasia** (**BPH**); the evaluation and treatment algorithms for BPH are well described.[1] In women, the evaluation and treatment of urinary retention are more elusive. A recent review article outlined a myriad of potential etiologies for acute urinary retention in women including: multiple sclerosis, spina bifida occulta, tethered cord, viral sacromyeloradiculitis, lumbar disc protrusion, cauda equine syndrome, primary bladder neck obstruction, pseduomyotonia, reflex sympathetic dystrophy, and psychogenic urinary retention.[2] Many of these etiologies are rare and studies pertaining to these pathologic mechanisms of urinary retention are limited. In this article, we would like to detail anogenital **herpes simplex virus** (**HSV**) and sacral **herpes zoster virus** (**HZV**) as potential causes of acute urinary retention in women.

HSV 1 and 2 and HZV are common yet under diagnosed infections worldwide.[3] In the United States in 2003, there were approximately 200,000 initial visits to doctors' offices for these viruses.[1] The transmission of the virus by close contact of skin or mucous membranes results in the high prevalence of HSV. Furthermore, the virus increases the risk of susceptibility to HIV by 3-fold. Once transmitted, the herpes virus initiates cytolytic replication in epithelial cells at the site of entry. Histologically, this is manifested as intranuclear inclusions and fused cells that form multinucleated giant cells. On physical examination this translates to fluid-filled blisters that contain cellular debris, inflammatory cells, and progeny virions.[4] This process, often referred to as the primary infection, may often have a variable constellation of clinical symptoms ranging from mild to severe. The symptomology ranges from painful oral or genital ulcers (98%), tender local lymphadenopathy (80%) to constitutional symptoms such as fever or headache (67%). Dysuria was found in the majority of patients with sacral HSV (63%). In women experiencing their first episode of primary genital herpes, 82% had HSV isolated on urethral cultures.[5] [6]

Chickenpox and herpes zoster represent the two clinical manifestations of varicella zoster virus infection.[7] Primary infection with VZV is transmitted through airborne droplets to the nasopharyngeal lymphoid tissue. This results in a host of immunologic changes responsible for the initial infection. Preferential infection of CD4 T-cells and the subsequent down regu-

lation of MHC class I expression have been described as possible etiologies for the virus's ability to evade the immune system and enhance transport to cutaneous epithelium. Clinical manifestations, in an immunocompetent host, include fever, malaise, and pharyngitis after a 2 to 3 week viral incubation period. This is often followed by a generalized vesicular rash within 24 hours. The rash persists for approximately four days after which there is crusting of the vesicles ending the infective period. Atypical presentations may be seen in immunocompromised individuals, including absence or unusual distribution of the rash, prolongation of the prodromal period, or other associated illnesses.[8]

HZV represents a reactivation of the latent herpes varicella zoster virus. After initial infection with VZV the virons exhibit latency within the dorsal root ganglions.[9] The virons possess several mechanisms to evade the immune system.[10] Clinically, patients develop a rash of erythematous papules distributed over one dermatome, which may be preceded by a burning pain over the same distribution. These papules coalesce and form bullea.[11] A well-known sequela of this disease is post-herpetic neuralgia. Elliott reported that peripheral motor neuropathy, also known as segmental motor paresis occurs in 3% of those with herpes zoster and may reflect either pain or sensory abnormalities in the affected dermatome.[11] Involvement of the sacral dermatomes can result in acute urinary retention.

The mechanism of acute urinary retention has been debated in the setting of sacral herpetic lesions. Severe dysuria associated with HZV infection, coined herpetic cystitis, has been implicated as a possible source of urinary retention. Alternatively, in the setting of HSV and HZV infections, the viruses are harbored in the dorsal root ganglia and sacral nerve roots. This results in detrusor areflexia likely induced by a sacral myeloradiculitis.[12] This theory was originally coined as Elsberg Syndrome.[13] Physical examination of a patient with sacral myeloradiculitis often reveals a saddle anesthesia, poor perineal muscle reflex, decreased sphincter tone, lower extremity weakness and/or parasthesias. The neurologic manifestations are typically preceded by the vesicular rash although rarely the reverse is also observed. Associated bowel dysfunction may be present in up to 50% of affected patients.[2] The majority of patients are not subjected to urodynamic evaluation because most of these patients will void spontaneously. However, when a cystometrogram is performed the usual diagnosis is a hypotonic or areflexic detrusor muscle. The cited bladder dysfunction almost always resolves spontaneously within 12 weeks of onset.[14, 15]

Treatments for herpes viruses have been well established. Anti-viral therapy has been shown to decrease the severity and duration of the initial infection. Famciclovir or Valacyclovir is given two or three times daily for seven to ten days for initial infection. Both agents can also be used in shorter duration (three to five days) for recurrent infection upon detection of the first clinical sign. Further, there is utility in using either medication as a daily maintenance therapy to achieve suppression of future outbreaks.[5, 16]

The use of **clean intermittent catheterization** (**CIC**) is the mainstay of management of acute urinary retention. This allows the bladder to be emptied in a timed fashion, to avoid stretch injury to the detrusor muscle and avoid potential development of upper tract damage. Further, this allows the bladder to cycle and allows for initiation of spontaneous voiding between catheterizations. If the patient does not begin to void spontaneously within 12 weeks of presentation or the residual urinary volumes remain elevated (>250cc), urodynamics should be performed.

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