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Unilateral temporary functional stasis in the upper urinary tract caused by "a filled bladder" on Tc-99m DTPA diuresis renography: a teaching case

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A 2-year-old girl with recurrent urinary tract infection having slight left pelvicaliceal dilatation on her renal ultrasound underwent a Tc-99m DTPA diuresis renography. During the excretion phase, a prominent and persisting left pelvicaliceal stasis was noticed even after the diuretic injection. However, it disappeared simultaneously with an uncontrolled micturition. This patient is presented to show the effect of filled bladder on the physiological drainage of urine. Since urine flow in the urinary system is more complicated than simple drainage, a thorough understanding of the physiological basis for diuresis renography and the pitfalls of the technique is required for its appropriate use in the management of patients suspected of urinary tract obstruction.

Key words: bladder, diuresis renography, Tc-99m DTPA, urine flow

INTRODUCTION

URINE FORMATION BEGINS in the renal corpuscle, where blood passing through the glomerulus is filtered. After renal tubular processing of glomerular ultrafiltrate, the urine that is going to be excreted from the uretra reaches the renal pelvicaliceal system. The journey of urine from pelvis to bladder is triggered by the collection of urine in the renal pelvis. Both mechanic- and chemoreceptors are present in the renal pelvis[1] and mechanoreceptors in the renal pelvis are sensitive to changes in urine outflow pressure.[2,3] When renal pelvic pressure reaches a predetermined threshold level, an electromechanical wave inducted by pelvic pacemaker cells takes a pelvic urine bolus to the ureter.[4] Excitation waves spread from the renal pelvis to the ureter, determining the propulsion of urine through intermittent phasic-type contractility. Considering the ureter as a syncytium, the propagation of impulses occurs as a purely myogenic process (via electrotonic spread) at points of intimate contact between ureter muscle cells or "gap junctions."[5–7] The spreading of the pacemaker electrical activity through the smooth muscle of the renal pelvis and its coupling with contraction both involve the activation of voltage-dependent L-type calcium channels. These channels also are recruited obligatorily for propagation of ureteral peristalsis and electromechanical coupling in the ureter smooth muscle.[5,8] Waves of peristaltic contraction usually travel along the ureter from the renal pelvis to the bladder at a roughly constant speed but at variable intervals.[9]

The ureter inserts into the trigone and the distal and of the ureter courses through the intramural portion of the bladder wall at an oblique angle. A healthy intramural tunnel, within the bladder wall, functions as a flap-valve mechanism for the intramural ureter and prevents urinary reflux. When an electromechanical wave reaches the urinary bladder, it causes a vesical pressure drop. This seems to create a negative pressure in the urinary bladder that might assist in the drainage of urine from the ureter through the opened ureterovesical junction.[10]

The bladder functions as a temporary reservoir for urine and possesses features that enable urine to enter, be stored, and later be released for evacuation from the body. Urination is usually stimulated by the distention of the bladder as it fills with urine. When the walls of the bladder contract, nerve receptors are stimulated, and the urination reflex is triggered. The urination reflex causes the internal urethral sphincter to open and the external urethral sphincter to relax. This relaxation allows the bladder to empty.

Since the journey of urine, starting from the renal pelvis

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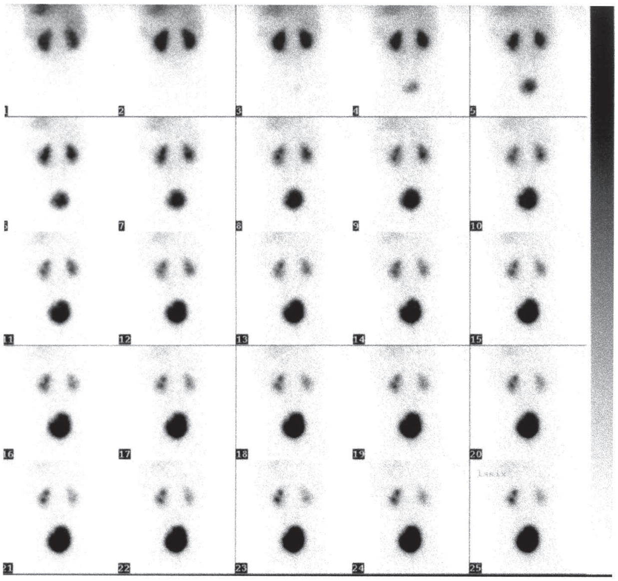

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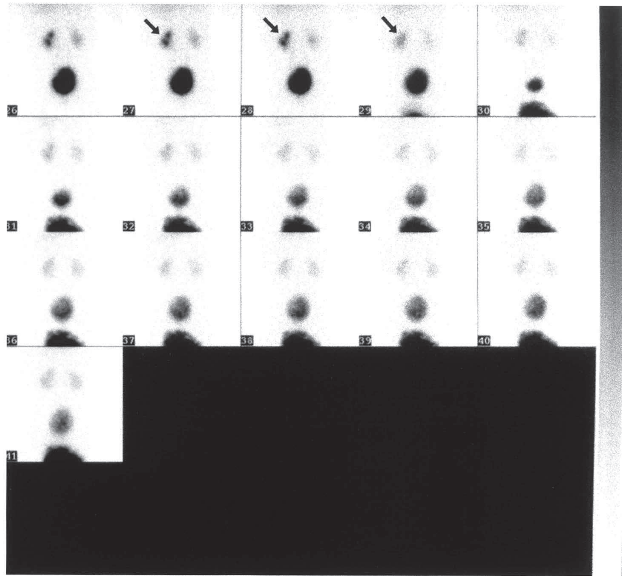

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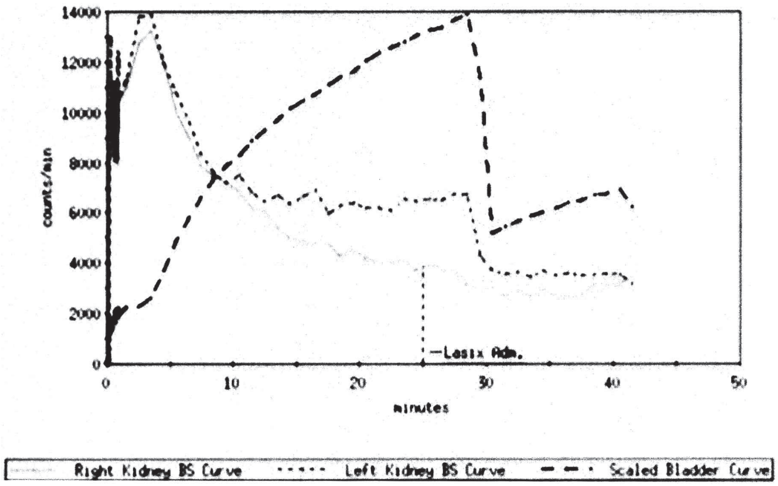

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Fig. 1 Prominent left pelvicaliceal stasis appeared at the end of the concentration phase (a). Images acquired during the excretion phase showed that the persistent left pelvicaliceal stasis (*arrows*) not responding to IV diuretic administration was cleared simultaneously with emptying the bladder (b). The renogram curve of the left kidney showed a plateau pattern in the excretion phase of the study that was inconsistent with vesicoureteral reflux. Simultaneous count decrease in region of interests of the bladder and left kidney was noticed on the renogram curve (c).

to its expulsion to the exterior at the urethral orifice, seems to be under the control of various physiologic mechanisms, the effect of filled bladder should be considered in the evaluation of excretion functions of the kidneys on dynamic renal perfusion scan, especially in patients with obstructive pattern on their scans.

CASE REPORT

A 2-year-old girl with recurrent urinary tract infection having a minimal left pelvicaliceal dilatation on her renal ultrasound was referred to our Department of Nuclear Medicine for a Tc-99m DTPA diuresis renography. A conventional diuresis renography, with an IV injection of furosemide (1 mg/kg) 25 minutes after the administration of 1.5 mCi (5.55 MBq) Tc-99m DTPA was performed to assess the split renal functions and upper tract urine drainage dynamics. Oral hydration was given as water and juice beginning 1.5 hour before the study. When the patient was in the supine position, posterior images of the kidneys were acquired. Rapid sequential digital flow images at 2 sec/frame for 1 min and functional images at 60 sec/frame for 40 min were recorded in a 64 × 64 matrix following the intravenous injection. Acquisition of images was performed with a large field of view gamma camera peaked with a 20% window at 140 keV, fitted with a low-energy, general purpose collimator. A hardware zoom for acquisition was 1.33. Perfusion and concentration (Fig. 1a) function of both kidneys were normal. During the excretion phase, a prominent and persisting left pelvicaliceal stasis was noticed even after the diuretic injection. However, it disappeared simultaneously with an uncontrolled micturition (Fig. 1b, c). The renogram curve of the left kidney was not consistent with the vesicoureteral reflux, because there was a plateau pattern indicating urinary stasis (Fig. 1c). The decrease in intravesical pressure after the emptying of the bladder led to free urinary flow. This patient is presented to show the effect of a full bladder on the physiologic drainage of urine.

DISCUSSION

There is an interaction between the lower and upper urinary tract and the effects of bladder filling on upper tract urodynamics were demonstrated by Jones et al.[11] Renal pelvic pressures were altered by bladder filling in some of their cases. They reported that it can be explained by the "open-tube" mode of ureteric transport in dilated upper tracts that permits proximal pressure transmission, but a similar effect may occur when a normal ureter intervenes between a dilated renal pelvis and a normal bladder under conditions of marked diuresis.[11] Similarly, Hvarness et al. reported that acute bladder distention caused a decrease in the glomerular filtration or "pooling of urine" in the upper urinary tract in healthy humans, although the mechanism was unknown.[12] It has also been said that noncompliant bladder (usually because it is full of urine) may generate sufficient back-pressure in a normal patent urinary system.[13] The effect of filled bladder on diuresis renography is to progressively impede drainage as the bladder fills and increase the risk of a false-positive result.[14] Asking the patient to void towards the end of the drainage phase of the study or patient catheterization to let free drainage can solve the problem. Posture is also a significant factor, because drainage is assisted by hydrostatic pressure in the erect posture. Since the temporary functional stasis of the upper urinary tract takes place under physiologic conditions without manipulation, our case is a teaching one and we would like to discuss the underlying mechanisms.

The urinary bladder functions as a temporary reservoir for urine. The bladder is innervated by afferent branches of the visceral nervous system, which transmit sensations of pain to the spinal cord and brain by branches of both the parasympathetic and sympathetic divisions of the autonomic or peripheral nervous system. The urination reflex is triggered by the distention of the bladder as it fills it urine. When the ureter inserts into the trigone, the distal end of the ureter courses through the intramural portion of the bladder wall at an oblique angle. As the bladder fills with urine and the bladder wall distends and thins, the intramural portion of the ureter also stretches, thins out, and becomes compressed against the detrusor backing. The process allows a continual antegrade flow of urine from the ureter into the bladder but prevents retrograde transmission of urine from the bladder back up to the kidney; thus, a healthy intramural tunnel, within the bladder wall, functions as a flap-valve mechanism for the intramural ureter and prevents urinary reflux. Therefore the renal pelvis is hydrodynamically isolated from the bladder under normal conditions. Since vesicoureteral reflux and ureteral dilatation were not detected in our case, back-pressure does not seem to be a plausible cause of temporary functional stasis in the upper urinary tract.

When intravesical pressure reaches the predetermined threshold level that causes the bladder to loose its compliance, pelvic pacemaker cells seem to be inhibited temporarily to prevent the urine flow. Some reflex mechanisms may be responsible for this temporary functional stasis in the upper urinary tract. The main, and possibly only, function of the pyeloureteral complex is to ensure the unidirectional transport of urine from the kidney to urinary bladder. The myogenic properties of the pyeloureteral smooth muscle, as reviewed in the introduction part of this case report, seem largely sufficient to account for normal ureteral peristalsis. On the other hand, the mammalian ureter is innervated mainly by unmyelinated fibers that originate from the renal, ovarian/spermatic, and sympathetic plexuses, but the existence and distribution of ureteric ganglia are controversial topics.[5] There is clear evidence that the mammalian renal pelvis and ureter receive innervations from several sources and that diverse transmitters play a role in the pyeloureteral tract, but with the exception of the role exerted by sensory neuropeptides and possibly NO, the overall evidence indicates that several transmitters which are clearly important for local regulation of motility in mammals.[5] On the other hand, the capsaicin-sensitive sensory innervation of the pyeloureteral complex can activate several local and reflex responses, which affect the production of urine and modify ureteral peristalsis. Although the significance of some of these reflexes is not fully understood, it is evident that noxious stimuli or "energies of activation" that are potentially noxious or tissue damaging are the most effective activators of both pyeloureteral reflexes and pain.[5] In addition, certain reflex responses can be activated by mechanical stimuli and/or changes in the chemical composition of the urine and may be important for regulating urine production, water, and Na balance.[1] Some other possible reflex mechanisms have already been proposed.[10,15,16] Although, the relationships existing between the activation of different populations of afferent nerves in the renal pelvis and the activation of specific reflexes is still a matter of investigation, the hypothesis of reflex inhibition of pelvic pacemaker cells by filled bladder via the neurogenic pathway may be a reasonable explanation in our case.

It should be remembered that each pyeloureteral tract works independently. Although, a reflex relationship is postulated to exist between the two renal pelves,[17] the urine collection and drainage in each pelvicaliceal system are not synchronized. For that reason, diuresis renography may show both unilateral and bilateral temporary functional stasis in the upper urinary tract in the presence of filled bladder.

In conclusion, since urine flow in the urinary system is more complicated than simple drainage, a thorough understanding of the physiological basis for diuresis renography and the pitfalls of the technique are required for its appropriate use in the management of patients suspected of having urinary tract obstruction.[18]

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