Case Report

Spontaneous expulsion of large vesicle calculi in a woman with paraparesis

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Introduction: Urolithiasis is a common but preventable complication of Spinal Cord Disorders (SCD).

Case report: We report a 25-year-old woman with paraparesis who spontaneously passed two large calculi perurethra without pain and developed urethral scarring. Detrusor hyperreflexia, absence of sensations and lack of sphincter tone could have contributed to painless expulsion of the large calculi in this patient.

Conclusion: Dysuria, a prominent symptom of urolithiasis may not be present in subjects with SCD. Awareness about urolithiasis among health professionals involved in the care of SCD patients is necessary for prevention and early intervention.

Keywords: paraplegia; urinary complications; vesical calculus; spontaneous expulsion

Introduction

The incidence of urolithiasis among spinal cord injury patients varies from 8% to 29%.^{1,2} The calculi are either passed as gravel or require surgical intervention. This communication reports the spontaneous expulsion of two large vesical calculi per urethra by a patient with residual paraplegia following transverse myelitis.

Case report

A 25-year-old woman developed paraplegia and retention of urine following acute transverse myelitis. Initially she was put on a continuous indwelling Foley's catheter for 3 months after which she could void by Crede's maneuver. Two years later she noted frequent interruption of the urinary stream with change of posture. Subsequently she passed two stones measuring 2.5 × 2 cms and 4 × 2.5 cms in size (Figure 1). Both stones were smooth surfaced, hard, and chalky white to yellow in colour. She did not have any pain or dysuria. She denied any past history of neurological or urological problems. She had spastic paraparesis (MRC grade 3–4) with impaired spinothalamic and kinesthetic sensations below the level of 10th thoracic dermatome. The stretch reflexes were exaggerated in lower limbs and the plantar response was

extensor bilaterally. Her upper limbs were normal. She had hesitancy of micturition and incomplete evacuation of bladder.

Her haemoglobin, blood cell count, blood urea, glucose and serum and creatinine, calcium, phosphorous, alkaline phosphatase, total proteins and albumin were normal. Urine microscopy and culture were negative. Ultrasound examination of the abdomen and pelvis revealed mild dilation of the calyceal system. The urinary bladder was normal. No calculi were noted in the urinary tract. The external urethra showed a patulous sphincter and scarring with a larger than normal external urethral orifice. Cystometry revealed a hyperreflexic detrusor with synergistic sphincters. X-ray of the spine and myelography were normal. She was treated with antispastic drugs and motor retraining with a supportive orthosis for locomotion. Bladder symptoms were managed with amitriptyline 75 mg daily, Kegel's pelvic floor exercises, and scheduled voiding. Biochemical analysis of the stone was not done for the want of facility.

Discussion

Patients with SCD are at high risk for urolithiasis.^{1–3} One of the most commonly reported clinical symptoms of bladder calculi is dysuria. The perurethral passage of calculi is very painful and only small sized calculi get expelled via this route spontaneously. This woman

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Figure 1 Large calculi passed painlessly

passed two very large vesical calculi without pain. Absence of pain, paralysis of the sphincters making the urethra patulous and detrusor hyperreflexia causing high intravesical pressures due to a spinal cord lesion might have facilitated the spontaneous expulsion of large calculi. The passage of large calculi probably injured the urethra and caused scarring at the external urethral orifice.

Risk factors for urolithiasis among patients with SCD include complete transection of the cord, lesions at or above the 4th thoracic spinal cord segment, upper motor neurone type of bladder, indwelling catheters, urinary tract infection with urease producing bacteria, and presence of residual urine.¹⁻³ Patients with indwelling catheters have a sixfold risk of stone formation in 2 years compared to those with normal voiding.² However this view has been challenged recently.³ Our patient was not on continuous indwelling catheter for about 2 years. The most

common cause of vesical calculi is infection by urease producing bacteria.³ Residual urine predisposes to infection and thus stone formation. Hence, clean intermittent catheterisation is essential for prevention of urolithiasis in patients with neurogenic bladder.

Subjects with complete cord lesions above the 4th thoracic spinal cord segment have a higher incidence of urolithiasis. This patient had an incomplete lesion at the 10th thoracic segment of spinal cord. Patients with SCD have a rapid and intense bone resorption from the paralysed limbs leading to hypercalcemia, hypercalciuria and increased hydroxyproline excretion predisposing to urolithiasis.⁴ Hypercalciuria due to poor mobility may have contributed to the development of vesical calculi in this patient.

Conclusion

The aim of this report is to highlight the problem of urolithiasis in patients with SCD. The clinical symptoms of bladder calculi may not be present in these patients with consequent delay in diagnosis. The lack of sensation, detrusor hyperreflexia and absence of sphincter tone may cause painless expulsion of large stones and damage to the urethra.

References

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