Meningomyelocele (MMC) is a congenital malformation of the spine, spinal cord, and overlying meninges that is known to affect 1 in 500–1 in 2000 live-born infants [1]. It is the most common open neural tube defect characterized by failure of the neural tube to close in the lumbosacral region during embryonic development (4th week post-fertilization), leading to the herniation of the meninges and the spinal cord through a vertebral defect [2]. The neural tube fusion starts at the level of the hindbrain (medulla and pons) and progresses rostrally and caudally. Incomplete fusion caudally leads to the formation of MMC around day 26 of gestation [3]. Prenatal triple screening for an elevated alpha-fetoprotein level during the first trimester is diagnostic in 85% of cases. Ultrasound is also very effective in screening for neural tube defects. The cause of myelomeningocele is unknown, but a deficiency in folic acid is believed to have a role in the pathogenesis of these neural tube defects [1].

The neurologic lesion produced by the MMC is variable and depends on what neural elements have everted with the meningocele sac. The bony vertebral level often provides little or no clue of the exact neurologic level or lesion produced. The height of the bony level may differ from the highest extent of the neurologic lesion for one to three vertebrae in either direction [4]. The neurologic lesion produced by this condition influences the lower urinary tract (LUT) function in a variety of ways and cannot be predicted just by looking at the spinal abnormality or the neurologic function of the lower extremities. We report a case of a 54-year-old male with lumbosacral MMC who was asymptomatic till 6 months back and was not treated for MMC. He presented with urinary voiding complaints following a fall and sustaining trauma. The patient underwent excision of MMC with detethering of the cord. Following surgery, the patient continued to have symptoms of the inability to pass urine.

**ABSTRACT**

Meningomyelocele (MMC) is a congenital malformation of the spine, spinal cord, and overlying meninges. The neurologic deficit produced by the MMC can be variable and depends on what neural elements, if any, have everted with the meningocele sac. The neurologic lesion produced by this condition influences the lower urinary tract function in a variety of ways. We report the case of a 54-year-old male with lumbosacral MMC who was asymptomatic till 6 months back and was not treated for MMC. He presented with urinary voiding complaints following a fall and sustaining trauma. The patient underwent excision of MMC with detethering of the cord. Following surgery, the patient continued to have symptoms of the inability to pass urine.

**Key words:** Lumbosacral, Meningomyelocele, Spinal cord, Urinary voiding

**CASE REPORT**

A 54-year-old male presented to the urological services of our hospital with complaints of acute retention of urine following a fall and sustaining an injury to the back for 6 months. The patient was born with lumbosacral MMC, who was asymptomatic till about 6 months ago and presented with urinary voiding complaints following a fall and sustaining an injury to the back.

On examination, pulse rate was 86/min and blood pressure was 130/80 mmHg. His per abdomen examination shows distended lower abdomen and bladder was palpable up to umbilicus. External genitalia examination shows no abnormality. Per rectal examination shows laxity of the anal tone. Neurological examination revealed 5/5 power in both right and left upper and lower limbs. Deep tendon reflexes were 3+ in the right upper and lower limbs and 3+ in the left upper limb and 2+ in the left lower limb. Plantar reflex was decreased bilaterally. Bulge with a scar over lumbosacral spine noted. Sensory loss was noted over S1 dermatome.
On investigations, the serum creatinine was 1.3 mg% and hemoglobin was 15.8 g%. The patient gave a history of fall from a height and developed severe lumbosacral region pain and loss of bowel bladder control. The patient was admitted to a neurosurgical center elsewhere. The discharge summary of that center stated that the examination of the back had revealed a soft non-pedunculated swelling of 4 × 3 cm with a broad base (Fig. 1a). Muscle power was 4/5 at the right hip, knee, and ankle. A magnetic resonance imaging spine revealed C4-C5 focal central disc protrusion (Fig. 1b) and C5-C6 central and paracentral disc bulge without significant nerve root compression.

The patient underwent excision of MMC with detethering of the cord at that hospital. Post-operative period was uneventful. In the post-operative period, the patient failed three catheter-free trials. The patient also had bowel complaints of constipation. Histopathological examination of the excised tissue showed no evidence of neoplastic pathology.

On examination, abdominal ultrasonography revealed bilateral normal appearing kidneys with a thickened bladder wall. Cystometry revealed a bladder capacity of 320 mL, maximum filling pressure of 51.7 cm H$_2$O, detrusor pressure of 42.1 cm H$_2$O at peak flow and peak flow of 2.6 mL/s and a post-void residue of 215 ml (Fig. 2). Cystoscopy was normal. The patient was put on clean intermittent catheterization and nighttime chemoprophylaxis. The patient is in regular follow-up for the past 4 months and he is doing well, and his serum creatinine was 1.02 mg/dL and his urine routine and microscopic examination were within normal limits on the last follow-up visit.

**DISCUSSION**

The primary goal of the management of neuropathic dysfunction of the LUT is the preservation of renal function. Secondary goals of management include urinary and fecal continence, prevention of urinary tract infection (UTI), and facilitation of sexual function and fertility. Preservation of renal function is achieved by maintaining low bladder pressures and active management of vesicoureteric reflux and the prevention of UTI [5].

Our case is an interesting case that the patient in spite of not having been treated in childhood was asymptomatic till 6 months back. The episode of trauma led to bowel and bladder symptoms. Clean intermittent catheterization is the preferred method of bladder management for patients with neurogenic bladder dysfunction with partial or complete urinary retention. Regular bladder emptying reduces intravesical bladder pressure and improves blood circulation in the bladder wall, making the bladder mucous membrane more resistant to infectious bacteria [6]. Intermittent catheterization improves self-care and independence and reduces barriers to sexual intimacy compared to the use of an indwelling catheter. If the patient with neurogenic bladder dysfunction has some ability to perform voluntary voiding, then voiding attempts are attempted every 3 h while awake including just before intermittent catheterization attempts. If the voiding attempts enable the patient to empty the bladder sufficiently so that residual urine volume in the bladder is under 100 cc consistently, then the catheterization can be discontinued. Residual urine volumes under 100 cc are associated with a reduced risk of the development of bacterial cystitis [7,8].
CONCLUSION

Management of neurogenic bladder requires patient education and interventions such as timed voiding, medications, intermittent catheterization, indwelling urinary catheter, and bladder and/or urethral surgical procedures. Individualized patient education regarding the management of their neurogenic bladder through specialized nursing is important for achieving successful neurogenic bladder management.

REFERENCES


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