Urinary Retention without Tetraparesis as a Sequel to Spontaneous Spinal Epidural Hematoma

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Abstract

A 55-year-old man suddenly developed neck pain, tetraplegia and decreased sensation below the neck. He was diagnosed with SSEH. Surgical removal of the hematoma, and laminoplasty were performed. At 2 months after the onset of the disease, the patient regained the ability to walk. However, at 5 months after the onset of the disease, the patient remained in a state of urinary retention even though his neurological findings were normal, except for mildly brisk reflexes in the lower extremities and decreased superficial sensation below the level of T4 including the perineal area. A urodynamic study showed normal bladder sensation, despite an acontractile detrusor and an unrelaxing external sphincter upon voiding. It is postulated that the descending micturition pathways (just inside the pyramidal tracts) were selectively affected, while the ascending micturition pathways (the dorsal columns) were preserved in the present case.

Key words: urinary retention, spinal epidural hematoma, urodynamic study

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Introduction

Spontaneous spinal epidural hematoma (SSEH) is a rare condition that causes severe paresis and pain (1). The most appropriate treatment for SSEH is surgery, with rapid decompression of the spinal cord being the most effective (1). However, following decompression surgery, some clinical symptoms may persist. We recently examined a patient who suffered from urinary retention without tetraparesis after SSEH.

Case Report

A healthy 55-year-old man suddenly developed severe neck pain, followed by tetraparesis. He had no history of previous illnesses such as hypertension or coagulopathy, nor did he have straining effort just prior to the onset of neck pain. Upon admission to the Orthopedic Surgery Department in our hospital, the patient had tetraplegia and decreased sensation below the neck, and an indwelling urinary catheter was inserted. Magnetic resonance imaging (MRI) of the cervical spine showed a C2-7 SEH anteriorly but without vascular malformation on T2-weighted images (Fig. 1). Surgical removal of the hematoma together with laminoplasty gradually ameliorated the patient’s tetraplegia. At 2 months after the onset of the disease, the patient regained the ability to walk, however, after the urethral catheter was removed, he was unable to urinate at all and was taught to perform clean intermittent self-catheterization (CISC) 4 times a day. At 5 months after the onset of disease, the patient was still in a state of urinary retention and was referred to our urodynamic laboratory. Upon referral, the patient had normal muscle power in the four extremities, while his deep tendon reflexes in the lower extremities were slightly brisk bilaterally, without extensor plantar reflexes. Superficial sensation...
after the urodynamic assessment, the patient’s difficulties included voiding problems, specifically a small bladder capacity and a low-compliance detrusor. During the voiding phase, the patient had a post-void residual volume of 600 ml (200 < normal < 600) during the storage phase, 120 ml (100 < normal < 300), and at bladder capacity, i.e., the first sensation of the bladder filling, i.e., a volume of 60 ml (< normal < 30). He had constipation that needed daily laxative.

Determinants of pin prick was moderately decreased bilaterally below the level of T4. Position and crude touch sensations in the extremities, including the perineal area, were preserved. He had constipation that needed daily laxative. He had no perspiratory dysfunction or postural syncope. A urodynamic study was performed to measure post-void residual volume and medium-fill (50 ml/min) water cystometry. Spinal electromyography (EMG) was carried out simultaneously using a concentric needle electrode in the external anal sphincter muscles. Normal volumes were observed at the first sensation of the bladder filling, i.e., a volume of 120 ml (100 < normal < 300), and at bladder capacity, i.e., a volume of 600 ml (200 < normal < 600) during the storage phase. There was no evidence of detrusor overactivity or low-compliance detrusor. During the voiding phase, the patient showed an acontractile detrusor without urinary flow, and the sphincter EMG remained unchanged while he tried to start voiding; this was followed by considerable straining during the voiding phase. The patient had a post-void residual volume of 600 ml (normal < 30). He was started on 20 mg/day pyridostigmine, a cholinesterase inhibitor, and 60 mg/day urapidil, an alpha-adrenergic antagonist. Six months after the urodynamic assessment, the patient’s difficulty in urination had ameliorated only slightly.

Pure urinary retention rarely appears in compressive disorders; however, a remarkable feature of the present patient was that there was dissociation between his recovery from tetraplegia and urinary retention after an acute transverse lesion; that is, while his tetraplegia disappeared completely, his urinary retention remained to the extent that needed CISC over a period of 9 months. To the best of our knowledge, there have been no previous reports on urodynamic studies in SSEH. However, the results of the present study (acontractile detrusor and unrelaxing sphincter on voiding) are consistent with those found in patients in the acute spinal shock phase, which could last for more than 6 months after the initial insult on the spinal cord (2). In the case of a patient with anterior spinal artery (ASA) infarction who initially presented with urinary retention, a postmortem pathological examination suggested that the patient’s urinary retention might have been due to the central cord syndrome, e.g., an initial ischemic event in the watershed area of the ASA, which involved the lateral columns that include the pyramidal tract and the adjacent long tract pathways (3). Similarly, the central cord syndrome due to cord compression with/without ischemia occurs in extramedullary disorders including SSEH (4). In addition, in a myelitis case with pure urinary retention (5), an MRI scan revealed localized inflammatory lesions that involved the lateral medullary reticulospinal tract just inside the pyramidal tract, e.g., the descending pathway to the bladder (6). Although no intramedullary lesions were visualized on MRI, it is possible that in the present patient the same area might have been selectively involved by the compressive/ischemic event by SSEH, leading to the presentation of urinary retention as an outstanding symptom. These reported cases, including ours, may indicate the vulnerability of the micturition descending fibers within the spinal cord. Nevertheless, a urodynamic study showed that bladder sensation was preserved in our patient. Since the major ascending pathways from the bladder were thought to travel in the dorsal columns (7), these pathways seemed to be preserved in the present case.

We have reported here the case of a man with urinary retention without tetraparesis as a sequel to spontaneous spinal epidural hematoma. The present case may indicate vulnerability of the micturition descending fibers within the spinal cord.

References

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