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VIDEO URODYNAMIC FINDINGS IN MEN WITH THE CENTRAL CORD SYNDROME

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ABSTRACT

Purpose: The central cord syndrome reportedly has a favorable prognosis and rehabilitation outcome. However, to our knowledge the status of the lower urinary tract in patients with the central cord syndrome is unclear. We report on 22 men with the central cord syndrome who were evaluated by video urodynamics.

Materials and Methods: From 1986 to the present we identified 22 men with a mean age of 51 years who had the central cord syndrome and were included in the Houston Veterans Affairs spinal cord registry. All patients underwent video urodynamic evaluation a mean of 34.5 months after injury.

Results: Video urodynamic testing for vesicourethral dysfunction was normal in 3 patients, while it showed bladder outlet obstruction secondary to benign prostatic hyperplasia in 2, detrusor areflexia in 4, external detrusor-sphincter dyssynergia in 11, detrusor hyperreflexia with a synergistic external urethral sphincter in 1 and detrusor hypocontractility in 1. Urinary tract infection recurred in 3 patients with external detrusor-sphincter dyssynergia and urolithiasis developed in 2.

Conclusions: Urodynamic testing revealed a high incidence of external detrusor-sphincter dyssynergia in men with the central cord syndrome. Due to the potential for upper tract deterioration all patients with the central cord syndrome should undergo baseline urodynamic studies. Those at high risk for upper tract deterioration with external detrusor-sphincter dyssynergia or a loss of compliance should be treated more aggressively with clean intermittent catheterization and anticholinergic medication when possible.

KEY WORDS: urinary tract, spinal cord injuries, central cord syndrome, urodynamics

The traumatic central cord syndrome is caused by an incomplete cervical spinal cord injury. Initially described by Schneider et al, it is characterized by incomplete quadriplegia with disproportionately worse impairment of the upper than the lower extremities.[1] In addition to this pattern of muscular weakness, other findings include neurogenic bladder dysfunction and various degrees of sensory deficits below the level of injury.[2] Previous reports indicate that the traumatic central cord syndrome involves between 9% and 16% of all spinal cord injuries.[3,4] The syndrome is more common in elderly individuals, typically after hyperextension injury when there is preexisting cervical spondolysis.[5] The predominant injury is axonal disruption in the white matter of the lateral column, which is best explained by bulging of the ligamentum flava into an already narrowed cervical spinal canal, causing direct compression of the cervical spinal cord.[6]

The central cord syndrome reportedly has a good prognosis and favorable rehabilitation outcome.[2,5] However, to our knowledge the status of the lower urinary tract in patients with this condition is much less clear. Only 2 studies in the literature specifically addressed the urinary tract in the central cord syndrome and each had a favorable urological outcome.[7,8] Urodynamic evaluation was done in all patients in only 1 series using carbon dioxide cystometry with simultaneous electromyography.

Video urodynamics, which involves simultaneous fluoroscopy, are more accurate for studying the lower urinary tract, especially when neurogenic bladder dysfunction is present. We evaluated our patients with the central cord syndrome by video urodynamics and report the incidence of vesicourethral dysfunction and the clinical outcome.

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MATERIALS AND METHODS

We retrospectively reviewed the records of all 442 patients diagnosed with the central cord syndrome who were admitted to the Houston Veterans Affairs Hospital between 1986 and the present, and entered into the spinal cord registry. Of these 442 cases 208 (49%) involved cervical cord level injury, including 22 (11%) diagnosed as the central cord syndrome. All 22 patients underwent video urodynamic evaluation. Mean time from injury to the most recent study was 34.5 months (range 1 to 94). All studies were performed after spinal shock resolved according to parameters defined by the International Continence Society. Filling cystometry was done with room temperature sterile contrast medium instilled at 20 or 50 cc per minute, followed by voiding pressure flow study. Intravesical and urethral pressure was measured with a 7Fr triple lumen catheter and abdominal pressure was determined using a rectal catheter. Simultaneous video images were obtained by C-arm fluoroscopy.

The urodynamic parameters evaluated were bladder capacity, detrusor compliance, detrusor leak point pressure, uninhibited detrusor contractions, synergistic or dyssyner-

gistic voiding, flow rate, magnitude of detrusor contraction during voiding and post-void residual urine volume. Vesicoureteral reflex was also noted. Detrusor compliance was considered normal, decreased and poor at greater than 20, 10 to 20 and less than 10 ml./cm. water, respectively. Bladders were characterized based on urodynamic pattern, including normal, obstruction with synergistic voiding with detrusor pressure 60 cm. water or greater and maximum flow less than 12 ml. per second, hypocontractility, areflexia, detrusor hyperreflexia and external detrusor-sphincter dyssynergia. We also noted other urological problems, such as recurrent urinary tract infections and those requiring hospitalization, renal and bladder calculi, and bladder management method.

RESULTS

We identified 22 men 21 to 72 years old (mean age 51) at injury who were diagnosed with the central cord syndrome and included in the spinal cord injury registry. Injury level was C6 in 5 cases, C5 in 6, C4 in 9, C3 in 1 and C2 in 1 (table 1). At evaluation renal ultrasound in 19 patients (86%) was normal in 18 (95%), while renal scarring in 1 was presumably associated with a history of recurrent febrile urinary tract infection. Hydronephrosis was not observed in any case, and renal and bladder calculi were present in 1 each. Recurrent urinary tract infections or those requiring hospitalization were noted in 3 patients.

Bladder management involved spontaneous voiding in 9 men, reflexive voiding by triggering and/or tapping in 1, intermittent self-catheterization in 8, a chronic indwelling Foley catheter in 3 and self-catheterization of an Indiana pouch in 1. Of the 9 patients on oxybutynin 6 performed self-catheterization, 1 voided spontaneously, 1 had an Indiana pouch and 1 had a chronic Foley catheter.

Urodynamic testing for vesicourethral dysfunction was normal in 3 men, while it revealed bladder outlet obstruction secondary to benign prostatic hyperplasia (BPH) in 2, detrusor areflexia in 4, detrusor hyperreflexia with a synergistic external sphincter in 1, external detrusor-sphincter dyssynergia in 11 and detrusor hypocontractility in 1 (table 2). There was urodynamic evidence of bladder outlet obstruction in 3 of the 7 patients with a synergistic voiding pattern, including 1 with detrusor hyperreflexia and 2 with an otherwise normal voiding pattern.

Followup urodynamics were performed in 8 patients a mean of 41 months (range 11 to 96) after the initial study. Initially urodynamic evaluation showed external detrusor-sphincter dyssynergia in 4 cases, areflexia in 2, detrusor hyperreflexia with synergistic obstruction or BPH in 1 and bladder outlet obstruction secondary to BPH in 1. Of the 8 patients 5 were on anticholinergic therapy, including 2 of the 4 with external detrusor-sphincter dyssynergia. Bladder management involved clean intermittent catheterization in 4 men, including 2 with external detrusor-sphincter dyssynergia, 2 with an indwelling Foley catheter and 2 with spontaneous voiding.

A followup study at 32 months showed that areflexia changed to hypocontractility in 1 case. Another patient with external detrusor-sphincter dyssynergia who was on clean intermittent catheterization and anticholinergics, and who had a history of recurrent urinary tract infections and bladder calculi had decreased bladder compliance on 15-month followup urodynamics. All other urodynamic patterns remained similar.

DISCUSSION

The central cord syndrome is an incomplete traumatic cervical cord injury that was initially described in 1954 by Schneider et al[1] The syndrome is characterized by disproportionately greater motor dysfunction of the upper versus the lower extremities, bladder dysfunction and variable sensory loss below the level of the lesion. Central cord lesions are reportedly common in elderly patients with cervical spondylosis after hyperextension injury.[5] Often injury results from a fall on level ground, which causes an apparently minor injury with no obvious bony spine abnormality.[9] Roth et al described a bimodal incidence of the central cord syndrome with injury in the younger group most often the result of major trauma and cervical fracture, while in the elderly group injury was usually associated with a minor fall and cervical spondylosis.[2] The mean age of our cohort was 51 years (range 21 to 72) and the majority of patients were between 40 and 60 years old. Of our patients 91% had spinal cord injury between the C4 and C6 levels (table 1).

The mechanism of the central cord syndrome is thought to be compressive forces on the spine in the anteroposterior plane. In 1951 the myelography autopsy studies of Taylor demonstrated that during hyperextension the posterior ligamentum flavum buckles inward to compress the spinal cord, aided on the anterior side by osteophytes or bulging intervertebral disks (see figure).[10] Mechanisms reported by Morse include severe cervical compression fracture, fracture and dislocation, vertebrobasilar insufficiency and congenital nar-

TABLE 1. *Patient characteristics*

Pt. No.—Age at Injury	Injury Level	American Spinal Injury Association Score	Mos. From Injury to Urodynamics	Urological Morbidity
1—70	C4	C	2	No
2—39	C6	D	3	No
3—48	C4	D	41	No
4—65	C4	D	3	No
5—53	C4	C	2	No
6—65	C5	B	58	No
7—72	C6	D	2	No
8—62	C4	D	90	No
9—56	C6	D	59	Urinary tract infections
10—40	C4	C	2	No
11—50	C4	D	32	No
12—66	C3		1	No
13—45	C4	C	12	No
14—54	C4	D	72	Renal stone
15—	C5	D		No
16—31	C5		94	No
17—21	C5		52	No
18—53	C2		40	Urinary tract infections
19—46	C6	D	3	No
20—33	C5	D	82	No
21—53	C6	D	46	Bladder transitional cell Ca
22—45	C5	D	29	Urinary tract infections + bladder stone