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Male Lower Urinary Tract Symptoms

Voiding Dysfunction Induced by Tetanus: A Case Report

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ABSTRACT

A 34-year-old man presented with sudden voiding dysfunction and lower limb paraplegia. As a central nervous system disorder was suspected, he was referred to the neurology department. Under the diagnosis of neurosarcoidosis, steroid pulse therapy was initiated. To ensure the effect of this therapy, the patient was referred back for urodynamic testing. Urodynamic testing indicated that the urethral sphincter was not relaxed and could not void. Due to the sudden appearance of repeated and refractory opisthotonus, tetanus was strongly suspected. After administration of antibiotics and tetanus immune globulin, those symptoms disappeared.

Introduction

Tetanus is rare in developed countries; despite widespread immunization with tetanus toxin (TT)-containing vaccines, its incidence in Japan is 60–120 cases/year.[1] The initial symptoms of tetanus include trismus, masseter muscle spasm, nuchal rigidity, and dysphagia.[1] Voiding dysfunction might suddenly develop due to neurogenic, myogenic, or iatrogenic factors, bladder outlet obstruction (BOO), or aging. When young patients with unidentified voiding dysfunction are examined, a neurogenic bladder due to a central nervous system (CNS) disorder is often considered in the differential diagnosis.[2] To the best of our knowledge, we report the first case of voiding dysfunction manifested as an initial symptom of tetanus.

Case presentation

A 34-year-old man was referred to our department due to sudden voiding dysfunction. Before referral, he experienced urinary retention twice. He also complained of difficulty walking 10 days after appearance of voiding dysfunction. He had a history of being scratched by a wild cat before 1 month before his symptoms started. Physical examination indicated lower limb paraplegia. As a CNS abnormality was suspected, he was admitted to the neurology department for further examination. Magnetic resonance imaging (MRI) of the brain and spinal cord were normal. Due to the likelihood of neurosarcoidosis, steroid pulse therapy was initiated. As he could not void without catheter during steroid therapy, he was referred back to our department for a urodynamic study (UDS) (Fig. 1). The UDS comprised a filling cystometry and pressure-flow study (PFS). The PFS indicated that no contraction of the detrusor and no relaxation of the urethral sphincter after the permission to void. At that time, it was suspected that this voiding dysfunction might be caused by a neurogenic disorder such as neurosarcoidosis. However, due to a lack of inflammatory findings on a cerebrospinal fluid examination, a past history of scratches by a wild cat, and the sudden appearance of repeated and refractory opisthotonus (that could only be resolved by midazolam), tetanus was strongly suspected. After administration of antibiotics and human tetanus immune globulin for approximately 1 week, the refractory opisthotonus resolved, and he was re-referred to our department for a re-evaluation of his voiding function. A UDS and free uroflowmetry were performed (Fig. 2). UDS showed no contraction of the detrusor, but the relaxation of urethral sphincter on the electromyography. Furthermore, he could void without catheter during the uroflowmetry. Uroflowmetry showed that the maximum flow rate, average flow rate, voided volume, and an ultrasound measurement of the post-void residual urine volume were recorded at 28.3 mL/s, 18.6 mL/s, 430 mL, and 0 mL, respectively. Finally, the patient could void without assistance and was discharged.

Discussion

The majority of young physicians in Japan rarely encounter tetanus cases. Since the initial symptoms of tetanus include

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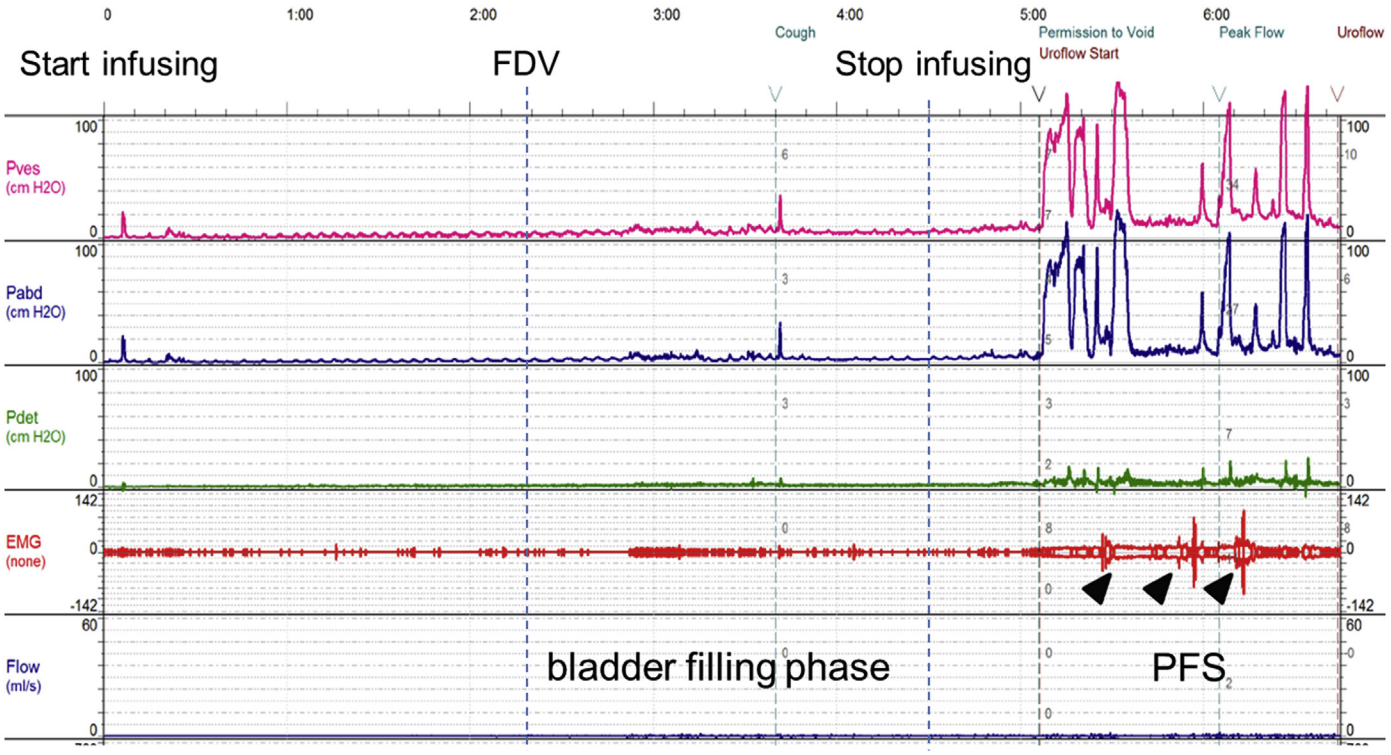

Figure 1. Urodynamic study before tetanus therapy. During the bladder filling phase, the volume at first sensation and the maximum capacity were 126 mL and 225 mL, respectively. Detrusor overactivity was not observed during this phase. Although the patient was permitted to void, he was unable to do so. The detrusor contractility was not confirmed and the urethral sphincter was not relaxed on electromyography during the pressure-flow study (arrowheads). Pves, vesical pressure (bladder); Pabd, pressure of the abdomen (rectal); Pdet, pressure of the detrusor = Pves − Pabd; EMG, electromyography of the urethral sphincter; Flow, flow of urine.

trismus, dysphagia, nuchal rigidity, and facial spasm,[1] patients typically present to the otolaryngology, internal medicine, orthopedics, dentistry, and emergency departments. Our case was unusual, as the initial symptom of tetanus was voiding dysfunction. To the best of our knowledge, no such case has been reported previously.

TT—a product of *Clostridium tetani*—is taken up by the nerve endings of lower motor neurons, which control volitional muscles.[3] It is retrogradely transported in the axons of lower motor neurons and reaches the CNS. TT is then transported across synapses and taken up by the nerve endings of inhibitory γ-aminobutyric acid–ergic and/or glycinergic neurons that regulate the activity of lower motor neurons. Based on this mechanism, urethral sphincter spasms are thought to be caused by a sacral spinal cord abnormality induced by TT. This hypothesis has been supported by sphincter electromyography data showing that relaxation of the urethral sphincter was not confirmed before treatment, but it was confirmed after treatment. Furthermore, it might be supported by the fact that urinary retention is occasionally observed as tetanus advances.[1] In our case, an abnormal volume at first sensation and maximum capacity values were observed on cystometry. It was unclear how tetanus affected the patient's bladder's sensory function. However, as the volume at first sensation and maximum capacity were within the normal range before the tetanus treatment, the effect of the injury to the bladder sensation was likely minimal.

Although detrusor contractility could not be assessed accurately in the PFS, cystometric and free uroflowmetry data implied that the urethral sphincter function, bladder sensation, and detrusor contractility were impaired by tetanus in the present case. The physical examination also revealed lower limb paraplegia, difficulty walking, and paresthesia. However, MRI of the spinal cord and brain did not reveal any abnormal findings. Considering the neurogenic, physical, and MRI findings and the tetanus diagnosis, voiding dysfunction was suspected to be caused by not only urethral sphincter, but also detrusor muscle dysfunction induced by a motor neuron system abnormality of the thoracic or sacral spinal cord as a result of the TT.

Voiding dysfunction can be caused by BOO, weak detrusor contractility, and detrusor sphincter dyssynergia, which can be associated with neurogenic, myogenic, or iatrogenic disorders, aging, or drugs.[4] Prostatic hyperplasia commonly induces voiding dysfunction in older men.[5] Since our patient was young and had a normal prostate volume (10 mL), benign prostate hyperplasia was unlikely. Considering the free uroflowmetry results after treatment, BOO could also be excluded. On the other hand, voiding dysfunction has been reported as an initial symptom of CNS disease.[1] Hence, when patients (particularly, young patients) with unidentified voiding dysfunction are examined, CNS disease should always be considered in the differential diagnosis. This is important, as some CNS diseases, including inflammatory nervous diseases, spinal cord injury, and tetanus, require emergency care.

In conclusion, we described a case of a male patient with tetanus whose UDS showed impaired urethral sphincter function, bladder sensation, and detrusor contractility, indicating abnormal motor neuron system function in the thoracic and sacral spinal cord. Hence, voiding dysfunction due to tetanus might be considered in the differential diagnosis of patients with a neurogenic bladder of unidentified causes and as an initial symptom of tetanus.

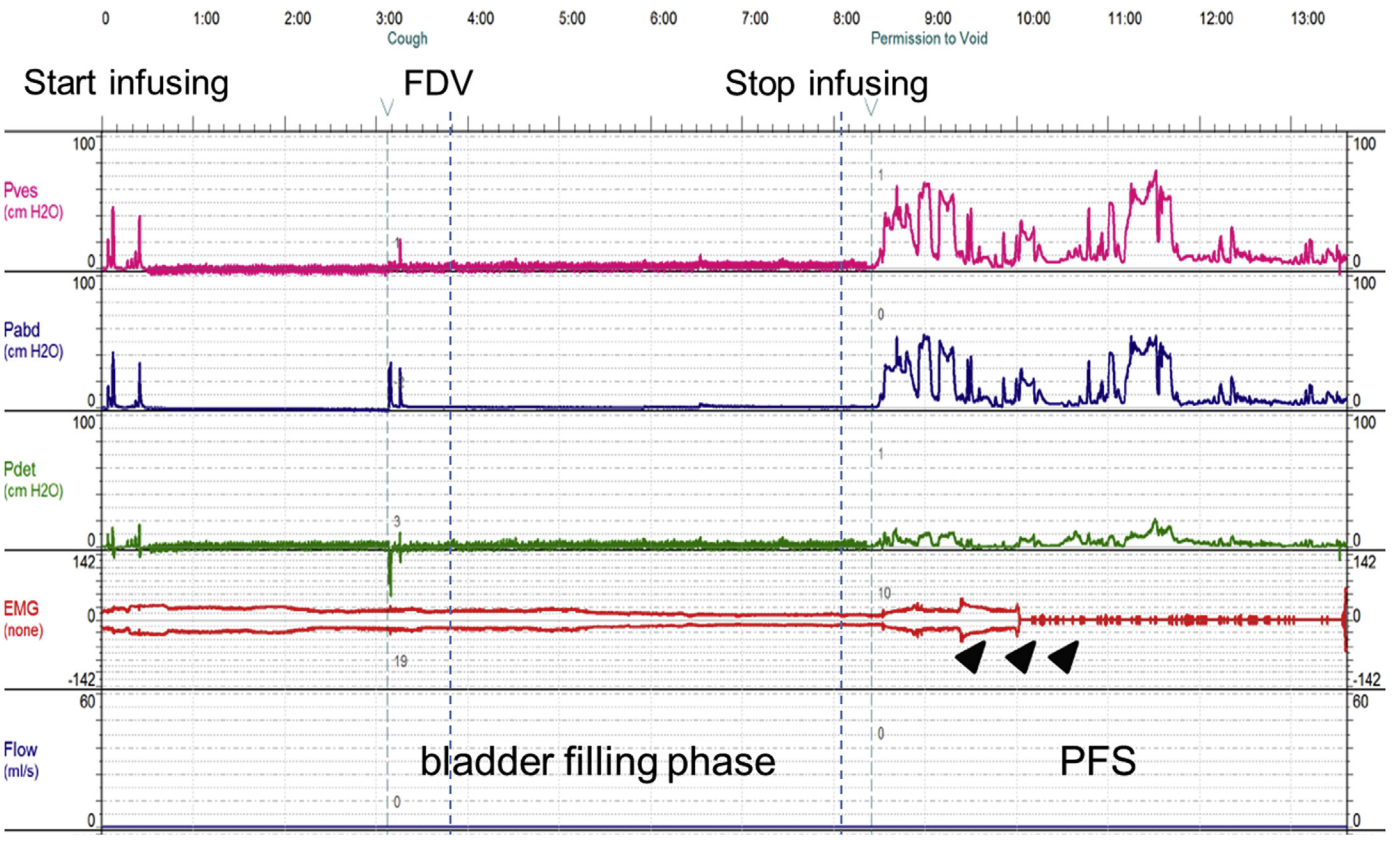

Figure 2. Urodynamic study after tetanus therapy. During the bladder filling phase, the volume at first sensation and maximum capacity were 172 mL and 393 mL, respectively. Detrusor overactivity was not observed during this phase. Although the patient was permitted to void, he was unable to void. The detrusor contractility was not confirmed, but the urethral sphincter was relaxed on electromyography during the pressure-flow study (arrowheads). Pves, vesical pressure (bladder); Pabd, pressure of the abdomen (rectal); Pdet, pressure of the detrusor = Pves − Pabd; EMG, electromyography of the urethral sphincter; Flow, flow of urine.

Conflicts of interest

None declared.

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