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Pelvic Floor Dysfunction and Poliomyelitis: A Case Report with Neurophysiological Evidence

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Abstract

Background: Normal pelvic visceral function depends on the complex interactions of intact somatic and autonomic nervous systems. Living with polio increases the risk of having bladder dysfunction. Urinary problems in polio infection may occur because the pelvic floor and bladder detrusor muscles have been paralyzed by the poliovirus. Neurophysiological tests should include electromyography (EMG), nerve conduction studies and the evaluation of the sacral reflexes in order to investigate the integrity of the somatic innervation of the pelvic floor muscles and urinary and anal sphincters.

Case Report: This is a case of a 53-year-old South-American man who had a history of poliomyelitis which affected his lower right limb when he was 2-years old. The patient was unable to empty his bladder completely. The aim of this paper is to describe the correlation between pelvic floor dysfunction, bladder symptoms and neurophysiological tests because this concept has not been investigated adequately in literature.

Conclusion: Neurophysiological diagnostic procedures adopted in our study can discern the degree of central and peripheral nervous system damage and confirmed that pelvic floor and detrusor muscles have been paralyzed by the poliovirus, in order to perform a personalized rehabilitation treatment.

Keywords: Pelvic floor; Electrophysiologic evaluation; Poliomyelitis; Neurophysiology

Introduction

A number of diseases or injuries of the central or peripheral nervous system can result in alterations in pelvic visceral function. The response to poliovirus infection is highly variable

and has been categorized on the basis of the severity of clinical presentation. Urinary problems in polio infection include retention, incomplete emptying of the bladder, incontinence, nicturia, and hesitancy. They may occur because the pelvic floor and bladder detrusor muscles have been paralyzed by the poliovirus [1].

A weak detrusor muscle may cause incomplete voiding, leaving residual urine-behind, consequently voiding becomes more frequent and overflow incontinence may occur. The autonomic sympathetic and parasympathetic nervous system that control body functions other than striated muscles, may be imbalanced (sympathetic preponderance) and give rise to difficulties in initiation of the voiding process.

An autonomic control of the filling and emptying bladder phases is exercised by nerve centers located predominantly in the spinal cord; the most important of which is the sacral medullary center. The Pontine micturition center regulates the activity of medullary centers with an inhibitory activity. Diseases or injuries of the nervous system in adults can damage the perfect synergism between spinal and supraspinal centers that control normal micturition. In order to understand the level and the extension of neurologic damage, a complete neurophysiological study of the pelvic floor is needed.

Case Presentation

In this study, we have evaluated a patient admitted to the Spinal Unit of the Scientific Institute of Pavia of ICS Maugeri, in the period between January 2014 and February 2014; he was subsequently re-evaluated in July 2017 to complete the diagnostic assessment of pelvic floor and urinary dysfunctions.

The patient was a 53-year-old South-American man, who had a history of poliomyelitis which affected his lower right limb when he was 2 years old, while his upper limb had not been affected. There was no medical history of diabetes mellitus, thyroid disease or alcohol dependence.

He presented post-polio residual deformities of the affected limb, consisting in a flexion-abduction contracture of hip and

flexion contracture of knee. A subtalar arthrodesis had been performed when the patient was 23 to correct the valgus deformity of right foot.

Physical examination revealed atrophy of his right lower limb muscles. There were fasciculations in the quadriceps muscle. The muscle tone was generally reduced. Further examination revealed weakness from myotome L3. Deep tendon reflexes were missing in the right lower limb. Sensation to light touch, temperature and pain was intact. Bladder function was impaired: although voiding was attempted by manual compression of the bladder, patient was unable to empty his bladder completely causing significant post voiding residual volumes; self-catheterization, performed four times a day, showed also significant post voiding residual volumes. Due to his urinary dysfunction, patient suffered recurrent urinary tract infection, which increased episodes of involuntary leakage of urine.

Magnetic resonance imaging (MRI) of the dorsal spine did not reveal any neurological compression of the cord or lumbar and sacral roots. Urodynamic testing revealed detrusor underactivity, or underactive bladder (UAB). The renal scintigraphic investigation showed a glomerular filtration function within the limits of normality.

Needle Electromyography (EMG) of right transverse perineal muscle, innervated by the pudendal nerve, evidenced chronic denervation signs, without denervation activity at rest. Motor Evoked Potentials, (MEPs) of the sacral motor roots were bilaterally delayed, with the right latency significantly longer (8 ms) than left latency (5 ms). Somatosensory Evoked Potentials, (SEPs) resulting from stimulation of the dorsal nerve of the penis evidenced delayed latency of cortical response (P1). Peripheral conduction (ankle-popliteal fossa) and cortical response (P40) resulting from electrical stimulation of the tibial nerve at the ankle resulted normal. Interestingly, these findings are not different from those we found in lower limb muscles (i.e., gastrocnemius medialis).

Discussion

This case report describes the results of electrophysiological evaluations of the afferent and efferent nerve pathways to the pelvic floor. The tests allow an evaluation of perineal somatic functions through the study of the pudendal nerve and its terminal branches.

In our study, needle electromyography (EMG) of right transverse perineal muscle showed clear chronic neurogenic changes in motor unit action potentials. EMG performed in the left transverse perineal muscle showed only mild chronic neurogenic changes. These findings are typical of patients diagnosed with chronic poliomyelitis.

The EMG examination of right transverse perineal muscle evidenced a reduced recruitment of motor units with prevalence of bi- and triphasic motor unit potentials; this finding allowed us to confirm the chronic phase of the disease excluding a post-polio recrudescence, which is characterized by positive sharp waves or fibrillation potentials. Interestingly,

we also studied the Motor Evoked Potentials (MEPs) in transverse perineal muscles by magnetic stimulation of the sacral motor roots to investigate the efferent motor path [2,3]. According to normal values used in our Unit of Clinical Neurophysiology, the sacral MEPs were bilaterally delayed. All these neurophysiological findings confirm a damage of motor path by poliovirus and are compatible with neurogenic weakness of pelvic floor muscles.

Motor Evoked Potential testing was performed to investigate the efferent motor path. The stimulus is able to activate the pyramidal paths and sacral roots and is used to appreciate the changes of the total conduction time. This is determined by the sum of the central conduction time (cortico-medullary) and the peripheral conduction time (time of depolarization of the motor roots from the spinal canal to the sphincter). The magnetic stimulation, that has the characteristic of being painless and of being able to activate the cortical pyramidal pathway and sacral roots, was attempted, in accordance with recent works [2,3] to appreciate changes in central conduction time and peripheral conduction time.

In addition to the motor pathways, sensory conduction was investigated by SEPs. Recording of the somatosensory evoked potentials for electrical stimulation of the dorsal nerve of the penis (or clitoris) [4,5] and for anal stimulation [6] completes the ascending evaluation of the pathway to the primary somatosensory cortex and allows to define pathologies involving the peripheral nerve, the medulla, the dorsal columns or the supraspinal central pathways [7]. In our patient, peripheral and central sensory conduction was fully normal ruling out any involvement of sensory pathways to the observed changes in pelvic floor function.

Proper function of the lower urinary tract depends on the integrity of the central and peripheral nervous pathways on multiple levels, and the complexity of this system leaves it susceptible to even minor lesions. Neurophysiological studies of the pelvic floor provide useful information about the extension of damage to the peripheral and central nervous system in subjects with poliomyelitis. Although there are many electrophysiological studies of the definition of normative values of the central and peripheral pathways involved in the continence function, the specific notions about electrophysiological investigations on groups of patients suffering from urinary and fecal continence disorders are still very few [8,9].

Our case report describes the association between pelvic floor, bladder symptoms and neurophysiological tests and suggests applying the above neurophysiological tests to patients with poliomyelitis and bladder dysfunction.

The level of spinal cord damage can discriminate the evolution and type of bladder dysfunctions that must be quantified with specific investigations. Polio survivors may have a weak pelvic floor leading to stress incontinence or an imbalance in the autonomic nervous system giving rise to urge incontinence or difficulties in initiation of voiding.

The electrophysiological study of the pelvic floor is useful in functional analysis of the disorders of sphincteric function [10], since it represents the required integration to morphological and functional assessment in sacral area dysfunction. By means of application of neurophysiological tests identifying site, type and degree of neurogenic lesion, the diagnosis of neurogenic alteration is allowed [7].

Unfortunately, such a study is not routinely applied in clinical neurophysiology laboratories [7] since testing performed in the pelvis is more complicated than elsewhere in the body due to the unique difficulty to get access to anatomical structures of the pelvis, as well as to the particular morphology of the pelvic floor and sphincter muscles.

Conclusion

In our study, urinary disorders of the patient were investigated through neurophysiological tests in order to underline a direct effect of primary polio infection on pelvic floor and detrusor muscles. EMG and neurophysiological findings were typical of patients diagnosed with chronic poliomyelitis and were compatible with a neurogenic weakness of pelvic floor muscles.

We propose that any patient with poliomyelitis and pelvic floor dysfunction should undergo pelvic floor needle EMG, motor conduction velocity study of terminal branches of the pudendal nerve, MEPs of transverse perineal muscles for efferent pathway from the spinal cord, SEPs from pudendal nerve which explores afferent pathway to spinal and supraspinal centers.

Our findings reveal that pelvic floor and bladder detrusor muscles have been paralyzed due to a direct effect of the poliovirus. A complete and individual rehabilitation project of polio survivors should also include the evaluation and treatment of pelvic floor dysfunction.

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