Detrusor Hyperreflexia in Stroke

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Abstract: In this study detrusor hyperreflexia was investigated by urodynamic study during the acute phase of stroke in patients who became incontinent after a cerebrovascular accident. Urodynamic studies reveal physiopathological findings of incontinence while the acute period of cerebrovascular accident do not cover neurogenic bladder features. In our study it was observed that most of the patients (60%) had normal bladder functions and detrusor hyperreflexia was a rare findings. This result is consistent with the other studies in literature.

Key Words: Detrusor hyperreflexia, stroke.

Introduction

Voiding disorders happen very frequently during the acute phase of a stroke. Although incontinent patients who have lesions above the brain stem are expected to have involuntray bladder contractions, urodynamic studies generally do not support this assumption. Clinical studies indicate that detrusor hyperreflexia is a rare findings in these patients and most of the patients have normal bladder functions (1). It is also suggested that incontinence happened after a cerebrovascular accident is a reversible condition and must be due to an unbalance between cortical and pontomesencephalic centers. The objective of this study is to observe the cystometric changes in the acute period of a stroke.

Material and Methods

The study was done between May 1995 and December 1995. 15 patients who became incontinent after a cerebrovascular accident were included into the study. None of the patients were comatose and their Glasgow coma scales were more than ten points. All the patients underwent a urological evaluation including firstly a descriptive history and physical examination. It was known from the history of the patients that none of them had voiding problems before the cerebrovascular event. Urodynamic study was done to all of patients in the first 5 days of the stroke. Cystometric studies were performed by using a multichannel urodynamic system (Griffon Urodynamics V.2.04v, England.). 37° centigrade isotonic sodium chloride solution used for instillation at the filling speed of 20 ml/min.. The presence of detrusor contractions with amplitude of 15 cm H₂O or over the filling phase of cystometry was accepted as detrusor hyperreflexia. Progressive stroke cases were excluded from the study in order to establish a correlation with a stable clinical state and prognosis of urinary incontinence. The pathological type of the cerebrovascular lesion (thrombosis or hemorrhage) was not a special condition for the study. All of the patients with the cortical and subcortical lesions were incontinent. The examination was repeated one week later regardless of the patients’ status of incontinence.

Results

Of the 15 patients with stroke 8 cases were male, and the others were female. The patients’ mean age were 61.7±5.9. Patients did not have any risk factor other than high blood pressure and hyperlipidemia. All of the patients had stroke for the first time in their life. Cerebrovascular lesion was hemorrhage in 2 (13%) patients and the other 13 (87%) patients had infarct in one of the territory of middle (80%), anterior (2%) or posterior (1%) cerebral arteries. Symptoms, CT, and cystometric findings were summarised in table 1.

Discussion

Urinary incontinence is a very frequent complaint
after a stroke. However, this symptom recovers in a short
time period. Urodynamic studies reveal physiopathological
findings of incontinence while the acute period of
cerebrovascular accident does not cover neurogenic bladder
features (1, 2, 3). In another study, it has been also
shown that cystometry findings might be normal in spinal
cord diseases (4). In this study, normal bladder functions
were observed in 60% of the patients. This result is
consistent with the other studies in literature.

The innervation of the bladder is very complex.
Parasympathetic and sympathetic nerves are in a
sensitive balance which is controlled by the higher cortical
and subcortical centers. The parasympathetic center is
located in the sacral and sympathetic center is in thoracic
segments of the spinal cord. The detrusor muscle is
innervated with parasympathetic nerves while the internal
sphincter is innervated with sympathetic nerves. The
external sphincter is composed of striated muscle fibers
and is innervated segments via the pudendal nerves. The
micturition center in the pontomesencephalic tegmentum of
the periaqueductal region receives afferent impulses
from anteromedial parts of the frontal cortex, limbic
regions, amygdaloid nuclei, thalamus, hypothalamus and
cerebellum (5, 6). In this study cerebrovascular lesions
were located above the brain stem and micturition center
was spared so it can be hypothesized that there were
temporary unbalances between the cortex and the
micturition center. Dysfunction of higher cortical
functions of the patients like speaking, reading etc. did
not improve though incontinence got well in a few weeks.
It can easily be asserted that incontinence does not
depend on the cortical localization of the lesion. Gelber
performed urodynamic studies in 51 patients who were
diagnosed cerebrovascular accident and 19 were
incontinent and observed that most of the patients had
large lesions and were aphasic (1). In another study,
Arena obtained similar results (7). Most of the
urodynamic investigations have inconsistent results with
the clinical situation of the patient in the acute phase of
cerebrovascular accident but healthy cortical structures
induce the balance again in a short time period. We
concluded that micturition center’s effects over the sacral
segments decline temporarily. However, this theory needs
evidence by postmortem studies.

<table>
<thead>
<tr>
<th>Age</th>
<th>Lesion in CT</th>
<th>Neurological Findings</th>
<th>Cystometry</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Left Temporoparietal infarct</td>
<td>Sensorial dysphasia, Right hemiparesis</td>
<td>NM*</td>
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<tr>
<td>2</td>
<td>Left Temporoparietal infarct</td>
<td>Global dysphasia, Right hemiparesis</td>
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</tr>
<tr>
<td>3</td>
<td>Left Frontotemporo-parietal infarct</td>
<td>Global aphasia Right hemiplegia</td>
<td>DH**</td>
</tr>
<tr>
<td>4</td>
<td>Right Thalamic hemorrhage</td>
<td>Left hemiplegia</td>
<td>DH</td>
</tr>
<tr>
<td>5</td>
<td>Left Frontal infarct</td>
<td>Right Hemiparesis Motor aphasia</td>
<td>NM</td>
</tr>
<tr>
<td>6</td>
<td>Right Frontotemporal-parietal infarct</td>
<td>Right Hemiplegia Global aphasia</td>
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<td>7</td>
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<tr>
<td>8</td>
<td>Left Temporal Hemorrhage</td>
<td>Confusion</td>
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<td>Left Hemihypoesthesia Confusion</td>
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<td>Impaired Left sided Cerebellar tests</td>
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<td>Left Hemiparesis, Motor dysphasia</td>
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<td>15</td>
<td>Left Temporoparietal infarct</td>
<td>Right Hemianopia Sensorial aphasia</td>
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</table>

*NM: Normoactive bladder **Detrusor Dyperreflexia
References


