Urinary Retention Associated with Stroke

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Abstract: Patients often exhibit urinary retention following a stroke. Various neuropathological and animal studies have implicated the medulla oblongata, pons, limbic system, frontal lobe as areas responsible for micturition control, although the exact area responsible for urinary retention after stroke is not clear. The purpose of this study was to identify the stroke area responsible for urinary retention by localizing the areas where strokes occur. We assessed 110 patients with cerebral infarction and 27 patients with cerebral hemorrhage (78 men, 59 women; mean age, 73.0 years) who had been admitted to our hospital between October, 2012 and September, 2013. We used computed tomography (CT) and magnetic resonance imaging (MRI) to investigate the stroke location, and evaluated whether post-stroke urinary retention occurred. Twelve (8.8%) of the 137 patients (7 men, 5 women; mean age, 78.8 years) exhibited urinary retention after a stroke. Stroke occurred in the right/left dominant hemisphere in 7 patients; non-dominant hemisphere in 1; cerebellum in 3; and brainstem in 1. Strokes in the dominant hemisphere were associated with urinary retention ($P = 0.0314$), particularly in the area of the insula ($P < 0.01$). We concluded that stroke affecting the insula of the dominant hemisphere tends to cause urinary retention.

Keywords: urinary retention, insula, stroke, detrusor disorder after stroke, fMRI.

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Background

Patients often exhibit urinary retention following a stroke. It has been reported that aphasia, cognitive impairment, poor functional status, and diabetes mellitus are associated with urinary retention after stroke [1], but the exact area responsible is not clear. Several studies have identified areas responsible for micturition control by using functional brain imaging techniques, such as functional MRI (fMRI) and positron-emission tomography (PET). A study by Fukuyama and colleagues reported bilateral activation in the supplementary motor areas, pons and midbrain during micturition [2]. Blok and colleagues found significant activation in the right pontine micturition center (PMC), periaqueductal grey (PAG), hypothalamus, and right inferior frontal gyrus during micturition, compared to that during rest with an empty bladder [3]. However, it is not clear from these brain imaging studies which of these areas, when damaged, is responsible for urinary disorders. To clarify this issue, we retrospectively identified stroke areas associated with impaired voiding function.
Methods

We enrolled 137 patients (78 men, 59 women; mean age 73 years) who had been admitted to our hospital because of cerebral infarction and intra-cerebral hemorrhage between October, 2012 and November, 2013. Of these, 110 patients (80.3%) were diagnosed with cerebral infarction and 27 patients (19.7%) with intra-cerebral hemorrhage.

We investigated both voiding control and brain lesions caused by infarction or hemorrhage. The stroke areas were determined by computed tomography (CT) and magnetic resonance imaging (MRI). Voiding function was assessed in all patients within 8 h of admission. If voiding had not been confirmed by 8 h after admission, the residual urine volume was determined by urethral catheterization. A patient was considered to have urinary retention if the residual urine volume was more than 200 ml on 2 consecutive occasions. For patients who were admitted with indwelling catheters, these were removed and their residual urine volume was determined 2 days after catheter removal, which was within 2 weeks of admission. Those patients with obvious prostatic enlargement or diabetic neuropathy or a history of voiding dysfunction were excluded from the study. Age, sex, dominant hand, and stroke location were recorded in the patients exhibiting urinary retention.

The patients’ data was handled carefully and preserved anonymously, in consideration of medical ethics. The data was assembled between October, 2012 and September, 2013 and was investigated retrospectively.

Results

Of the 137 patients (78 men, 59 women; mean age: 73 years) who participated in the study, 12 (8.8%; 7 men, 5 women; mean age: 78.8 years) demonstrated urinary retention, as diagnosed by an urologist. All of these patients were right-handed, except for 1 (case 4), who was left-handed.

Regarding the site of stroke, 45 patients presented with right hemisphere infarcts: 17 had infarcts in the basal ganglia and the internal capsule supplied by the lenticulostriate arteries; 2 had infarcts in the deep white matter; and 26 had infarcts in the cortical and subcortical regions. Thirty-seven patients presented with left hemisphere infarcts: the number of patients with infarcts in the above-mentioned regions was 17, 2 and 18, respectively. Four patients had bilateral scattered infarcts, 9 had cerebellar infarcts, 13 had brainstem infarcts, and 2 had brainstem and cerebellar infarcts (Table 1).

Seven patients had a cerebral hemorrhage in the right basal ganglia, 5 in the left basal ganglia, 10 in the left subcortical region, 3 in the cerebellum and 2 in the brainstem (Table 1).

Twelve patients showed urinary retention caused by neurogenic bladder dysfunction; of these, 7 had infarcts and 5 had hemorrhage. Table 2 shows the stroke areas in detail. In total, 8 patients exhibited stroke in the cerebral hemisphere, of which 6 were localized in the left side. One patient (case 4), who had right frontal infarcts, developed sensory aphasia, which led us to believe that the stroke had occurred on his dominant side. Therefore, 7 of the 8 patients with urinary retention had strokes in their dominant hemisphere.

Those with dominant side strokes were significantly more likely to develop urinary retention, compared to the patients with stroke on the non-dominant side ($P = 0.0314$, Table 3). Upon evaluating the area associated with urinary retention, the insula was included in the stroke area in 6 of the 7 patients with dominant-side stroke. Figure 1 shows the stroke area of the cases in which the insula was involved.

We will describe 2 representative cases: Case 4 and Case 7. Case 4 was an 82-year-old left-handed man who had right cerebral infarction that caused sensory aphasia and urinary retention. Figure 2 shows the initial MRI diffusion image, in which the stroke areas were the insula, the middle frontal gyrus (MFG) and the inferior frontal gyrus (IFG). His urinary retention improved in a few weeks. Case 7 was a 74-year-old right-handed man who had left cerebral infarction. Figure 3 shows the initial MRI diffusion image. The stroke areas were the insula and the IFG. His urinary retention also improved in a few weeks.

Next we evaluated whether left insular damage was more likely to cause urinary retention compared to other stroke areas, including the thalamus, cerebellum, and brainstem. One of the 9 patients with a right thala-
mus stroke and 3 of the 12 patients with a cerebellar lesion exhibited urinary retention. Urinary retention was also found in 1 patient out of the 15 with brainstem stroke.

Strokes of the left insula occurred in 9 of the 137 patients. We evaluated the relationship of all of these areas (left insula 9, thalamus 9, cerebellum 12, brainstem 15 and total 45) to urinary retention by $2 \times 2$ chi-square analysis. Statistical analysis showed that among these areas, the left insula was significantly associated with urinary retention ($P = 0.000983$, Table 3).

### Table 1. Localization of cerebral infarcts in the 137 patients

<table>
<thead>
<tr>
<th>Localization</th>
<th>n</th>
<th>right</th>
<th>left</th>
<th>bilateral</th>
</tr>
</thead>
<tbody>
<tr>
<td>Basal ganglia, internal capsule</td>
<td>17</td>
<td>17</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Deep white matter</td>
<td>2</td>
<td>2</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cortical and subcortical</td>
<td>26</td>
<td>18</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>45</td>
<td>37</td>
<td>4</td>
<td></td>
</tr>
</tbody>
</table>

Localization of cerebellar and brainstem infarcts

- Cerebellum: 9
- Brainstem: 13
- Brainstem and cerebellum: 2
- Total: 24

Localization of cerebral hemorrhage in patients

- Basal ganglia, thalamus: 7, 5
- Subcortical: 0, 10
- Cerebellum: 3
- Brainstem: 2

![Fig. 1. Areas of stroke in the 7 cases with urinary retention and a stroke.](image1)


![Fig. 2. MRI diffusion image of case 4, showing insula, middle frontal gyrus (MFG), and inferior frontal gyrus (IFG) stroke.](image2)

![Fig. 3. MRI diffusion image of case 7, showing that the stroke areas are insula and IFG stroke.](image3)
Urinary disturbance following acute stroke has been reported previously. Some reports have suggested detrusor disorder after stroke as the primary cause of this phenomenon [4], while others have claimed that stroke-induced cognitive impairment and aphasia cause the urinary disturbance, as patients with cognitive impairment and/or aphasia are more likely to have difficulties indicating or communicating their voiding needs and challenges [1, 5]. However, the scientific mechanism of urinary dysfunction after stroke is not well understood. In general, it is thought that urinary function is controlled by the sacral spinal cord, and micturition is

Table 2. Clinical characteristics in the patients with urinary retention

<table>
<thead>
<tr>
<th>Case</th>
<th>age</th>
<th>sex</th>
<th>stroke side</th>
<th>stroke</th>
<th>dominant or non-dominant</th>
<th>area of stroke</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>85</td>
<td>M</td>
<td>left</td>
<td>cerebral infarction</td>
<td>dominant</td>
<td>insula, STG, MTG, ITG</td>
</tr>
<tr>
<td>2</td>
<td>73</td>
<td>〃</td>
<td>〃</td>
<td>cerebral hemorrhage</td>
<td>〃</td>
<td>insula, Putamen</td>
</tr>
<tr>
<td>3</td>
<td>79</td>
<td>F</td>
<td>〃</td>
<td>cerebral hemorrhage</td>
<td>〃</td>
<td>STG, MTG, ITG, insula, PosG, SMG, AG</td>
</tr>
<tr>
<td>4</td>
<td>82</td>
<td>M</td>
<td>right</td>
<td>cerebral infarction</td>
<td>〃</td>
<td>insula, MFG, IFG (this patient has aphasia)</td>
</tr>
<tr>
<td>5</td>
<td>89</td>
<td>F</td>
<td>left</td>
<td>cerebral hemorrhage</td>
<td>〃</td>
<td>SMG, AG, SPL, IPL</td>
</tr>
<tr>
<td>6</td>
<td>47</td>
<td>M</td>
<td>〃</td>
<td>cerebral hemorrhage</td>
<td>〃</td>
<td>insula, SFG, MFG, IFG, PreG, PosG</td>
</tr>
<tr>
<td>7</td>
<td>74</td>
<td>〃</td>
<td>〃</td>
<td>cerebral infarction</td>
<td>〃</td>
<td>insula, IFG</td>
</tr>
<tr>
<td>8</td>
<td>86</td>
<td>F</td>
<td>right</td>
<td>cerebral infarction</td>
<td>non-dominant</td>
<td>thalamus</td>
</tr>
<tr>
<td>9</td>
<td>79</td>
<td>M</td>
<td>bilateral</td>
<td>cerebellar infarction</td>
<td>〃</td>
<td>bilateral lobule, cerebellar peduncle, culmen, lt tonsil</td>
</tr>
<tr>
<td>10</td>
<td>90</td>
<td>F</td>
<td>right</td>
<td>cerebral infarction</td>
<td>〃</td>
<td>right cerebellum lobule</td>
</tr>
<tr>
<td>11</td>
<td>73</td>
<td>M</td>
<td>〃</td>
<td>cerebral infarction</td>
<td>〃</td>
<td>right tonsil, cerebellum lobule</td>
</tr>
<tr>
<td>12</td>
<td>88</td>
<td>F</td>
<td>bilateral</td>
<td>brainstem hemorrhage</td>
<td>〃</td>
<td>pons, midbrain</td>
</tr>
</tbody>
</table>


Table 3. Stroke localization in the patients with or without urinary retention

<table>
<thead>
<tr>
<th>Site of lesion</th>
<th>with urinary retention</th>
<th>no urinary retention</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stroke of dominant side</td>
<td>7</td>
<td>46</td>
<td>$P = 0.0314, \chi^2 = 4.629$</td>
</tr>
<tr>
<td>Non-dominant side</td>
<td>1</td>
<td>50</td>
<td></td>
</tr>
<tr>
<td>Left insula</td>
<td>6</td>
<td>3</td>
<td>$P = 0.00983, \chi^2 = 10.85$</td>
</tr>
<tr>
<td>Thalamus, cerebellum, brainstem</td>
<td>5</td>
<td>31</td>
<td></td>
</tr>
<tr>
<td>Right thalamus</td>
<td>1</td>
<td>8</td>
<td>$P = 0.298, \chi^2 = 1.082$</td>
</tr>
<tr>
<td>Left insula, cerebellum, brainstem</td>
<td>10</td>
<td>26</td>
<td></td>
</tr>
<tr>
<td>Cerebellum</td>
<td>3</td>
<td>9</td>
<td>$P = 0.958, \chi^2 = 0.00273$</td>
</tr>
<tr>
<td>Left insula, thalamus, brainstem</td>
<td>8</td>
<td>25</td>
<td></td>
</tr>
<tr>
<td>Brainstem</td>
<td>1</td>
<td>14</td>
<td>$P = 0.0497, \chi^2 = 3.850$</td>
</tr>
<tr>
<td>Left insula, thalamus, cerebellum</td>
<td>10</td>
<td>20</td>
<td></td>
</tr>
</tbody>
</table>

Discussion

Urinary disturbance following acute stroke has been reported previously. Some reports have suggested detrusor disorder after stroke as the primary cause of this phenomenon [4], while others have claimed that stroke-induced cognitive impairment and aphasia cause the urinary disturbance, as patients with cognitive impairment and/or aphasia are more likely to have difficulties indicating or communicating their voiding needs and challenges [1, 5]. However, the scientific mechanism of urinary dysfunction after stroke is not well understood. In general, it is thought that urinary function is controlled by the sacral spinal cord, and micturition is
controlled by the PMC, which is inhibited by the frontal lobe, internal capsule, and basal ganglia; however, this remains unconfirmed.

PET and fMRI studies have shown that various cortical areas are activated during micturition and the storage phase. In a study of 17 male volunteers, Blok et al. reported that PET showed activity in the PAG, hypothalamus and in the right PMC during micturition [3]. Nour et al. have also reported that micturition is associated with increased activity in the pons, IFG, hypothalamus, PAG, and several other cortical areas [6]. On the other hand, during the storage phase, descending cortical control occurs, which allows the individual to postpone emptying the bladder until an appropriate juncture [7]. Increasing bladder volumes have been correlated with increased activity in the PAG, midline pons, mid-cingulate cortex, and bilateral frontal cortex [8]. Matsuura et al. have shown broadly similar cortical activation during bladder distension [9]. It has been suggested that these findings reflect the cerebral suppression of the desire to void.

Kuhtz-Buschbeck et al. also found insular activation during urinary storage, in addition to posterior parietal and prefrontal cortex activity, which were interpreted as important in deciding the timing of voiding [10]. Blok et al. and Nour et al. have shown that the storage of urine affects activity in the anterior cingulate and insula. Therefore, it is possible that two cerebral mechanisms control the storage phase, namely inhibition and activation of voiding.

In our study, strokes in the dominant hemisphere and dominant insula were strongly associated with urinary retention. The insular cortex was shown to be activated in the storage phase in PET and fMRI studies, indicating that this area is related to the transition between the storage phase and the onset of micturition. The finding by Kuhtz-Buschbeck et al. that the dominant insula connects to many other brain regions during attempted micturition supports this hypothesis. The insula has a visceral sensory function, and has been labeled as the limbic sensory cortex, with activation in response to visceral stimulation [11, 15]. Griffiths et al. have shown that neural circuits are associated with control of the lower urinary tract, and the circuit that includes the insula is associated with perception of bladder filling, which connects to the voiding reflex [15]. Hence we thought that disturbance of the insula due to stroke caused the loss of control of micturition.

However, three of our patients with stroke that included the left insula did not have urinary retention (Table 3). These were left frontal hemorrhage, left frontal cerebral infarction and left putamen hemorrhage. We did not find any relationship between left insula stroke and urinary retention. Table 2 shows that multiple lesions together with the insula contributed to urinary retention, but the areas were not consistent. Urinary control is highly complicated, and we think the insula plays an important role in voiding, but that disturbance of the insula does not always cause urinary retention.

None of the patients in our study had urinary retention after infarcts in the basal ganglia and internal capsule supplied by the lenticulostral arteries. It has been reported previously that damage to the internal capsule results in hyperreflexia [4], and that detrusor hyperreflexia causes urinary incontinence, rather than urinary retention [5]. Other investigated stroke areas (thalamus, cerebellum, and pons) have been shown to be activated during the micturition phase in PET and fMRI studies [2, 3, 6, 7], and there have been some reported cases of strokes in these areas causing urinary retention [12, 13]. Descending pathways of the PMC, including the medulla, have also been associated with voiding function [14], but in our study these areas were not associated with urinary retention. The role of these areas is probably minor in the vast network of urinary control, and damage to these regions may be a risk factor for urinary retention, which may result when patients with strokes in these areas have prostatic hyperatrophy or infections that are further risk factors.

Urinary control is highly complicated, and several risk factors feature in it. Urinary retention following a stroke tends to improve, as we have observed in the current cases, who got better in a few weeks, and may also help explain the cause of urinary retention that occurs after stroke.

Conflicts of Interest

The authors declare that they have no conflict of interest.
References

脳卒中による障害部位と脳卒中後尿閉の検討

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要旨：脳卒中後の患者は尿閉を伴うことがしばしば認められる。さまざまな神経病理学的研究や動物実験より, 延髄, 橋, 大脳辺縁系, 前頭葉が排尿コントロールを行うと言われているが, 正確な領域は特定されていない。今回脳卒中で発症し尿閉を生じた症例について脳の障害部位を検討し,どの領域が尿閉に関係するかを調査した。2012年10月〜2013年9月の間, 当科へ脳卒中で入院した症例 (脳梗塞 110例, 脳出血 27例, 男性 78人, 女性 59人, 平均年齢 73歳) を対象とし, 脳の障害部位を頭部MRI, CT検査にて検討し, 尿閉の有無を調査した。脳卒中後に尿閉を発症した症例は137例中12例 (8.8%) (脳梗塞 7例, 脳出血 5例, 平均年齢 78.8歳) であり, 12例中7例が優位半球の障害であった。1例が非優位半球であり, 小脳が1例, 脳幹が1例の結果であった。優位半球に脳卒中を発症した症例は, 有意に尿閉と関連があり (P = 0.0314). 特に島の障害は有意に尿閉を発症した結果であった (P < 0.01). 優位半球の島を含めた領域の脳卒中の場合, 脳卒中後尿閉を起こしやすいと考えられた。

キーワード：尿閉, 島, 脳卒中, 脳卒中後排尿障害, fMRI.