Frequency and Characteristics of Taste Impairment in Patients with Parkinson’s Disease: Results of a Clinical Interview

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Abstract

Objective  Patients with Parkinson’s disease (PD) frequently complain of a diminished sense of smell. Less frequently, they may complain of taste impairment. In the present study, we investigated the symptoms, frequency, and severity of taste impairment as well as smell impairment in PD patients and compared the results with those of age- and sex-matched healthy controls.

Patients and Methods  We interviewed 285 PD patients (120 men, 165 women) without dementia or nasal problems. Control subjects comprised 61 (20 men, 41 women) healthy spouses of the PD patients. Alteration of smell and taste sense lasting more than 3 months was defined as abnormal.

Results  One hundred and sixteen patients with PD complained of smell impairment and 26 complained of taste impairment. Only 5 controls complained of smell impairment, and no control subjects reported taste impairment. Taste impairment was more marked in patients with smell impairment. Impaired taste included diminished taste perception in 21 patients, altered sense of taste in 4 patients and burning mouth in 1 patient.

Conclusion  Taste as well as smell perception is impaired in patients with PD. The frequency of smell and taste impairments tended to increase with disease progression.

Key words: smell, taste, olfactory, ageusia, dysgeusia, burning mouth

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Introduction

Smell impairment is reportedly present in 74-97% of patients with Parkinson’s disease (PD) (1-3). Odor threshold, discrimination and identification have been used to evaluate smell impairment (3, 4). The determined frequencies of smell impairment vary among reports based on the method of evaluation (i.e., psychophysical “Sniffin’ Sticks” testing vs. electrophysiologic testing). Taste impairment has also been reported in patients with PD, but less frequently and with contradictory results (5-7). Abnormal taste includes reduced, sensitive and altered sensation and taste perception. Taste impairment may be caused not only by an altered threshold of taste and sensory pathway but also by various mental and physical disorders, including depression, taste bud or mucosal lesions, gum disease, dry mouth, gastrointestinal diseases, zinc deficiency, and medication (8, 9). Therefore, the symptoms of taste impairment may vary depending on the cause. Subnormal taste often induces appetite loss, which results in malnutrition and impairs the quality of life. In the present study, we aimed to clarify the frequency and characteristics of taste impairment in patients with PD. For this purpose, we interviewed PD patients about taste abnormalities as well as smell abnormalities and compared the results with those of the patients’ healthy spouses as controls.

Patients and Methods

Patients  We interviewed 285 consecutive PD patients (120 men, 165 women) without dementia defined by DSM-IV, asthma,
rhinitis, COPD, history of middle ear surgery, nasal surgery, severe head trauma, nor Bell’s palsy which may affect both smell and taste and who had not experienced a drug change in the past 3 months. Control subjects comprised 61 (20 men, 41 women) healthy spouses of the patients. Diagnosis of PD was made by two movement disorder specialists (K.K. & T.I.) according to the United Kingdom Parkinson’s Disease Society Brain Bank Diagnostic Criteria (10).

**Evaluation of abnormal smell and taste**

Alteration of smell and taste sense lasting for at least 3 months was classified as abnormal. The questions about problems with smell or taste were presented in a similar manner to those asked in the National Health Interview Survey conducted in the United States (8), but translated into Japanese. If subjects answered that they had smell problems, the impairment was evaluated as follows: 0, normal; 1, slight hyposmia; 2, moderate to severe hyposmia; 3, anosmia. Taste impairment was evaluated as: 0, normal; 1, slight hypogeusia (a diminished taste perception) or dysgeusia (an altered sense of taste or the unexplained presence of tastes such as bitter, salty, sour or sweet, or pain); 2, moderate to severe hypogeusia or dysgeusia; 3, aguesia. Subjects who had nasal inflammation or those undergoing chemotherapy were excluded from the study.

Levodopa equivalent dose (LED) for dopamine agonists was calculated as follows: pergolide ×100, cabergoline ×50, pramipexole ×50, ropinirole ×12.5 and bromocriptine ×10.

This study was approved by the Ethics Committee of Okayama Kyokuto Hospital. Informed consent was obtained from each subject prior to the study.

**Statistical analysis**

Differences in age, disease duration and dose of LED between two groups were evaluated using Student’s t-test. Differences in sex and ratio of smell and taste impairment were evaluated using a χ² test. The ratio of patients with taste impairment between PD patients with and without smell impairment was also analyzed using a χ² test. Differences in Hoehn and Yahr stage and Mini-Mental State Examination (MMSE) scores between PD patients with and without taste impairment were analyzed using the Mann-Whitney U-test.

**Results**

Mean age ± SD for patients with PD was 72.4 ± 9.0 years and that for controls 70.9 ± 8.6 years. There was no significant difference in age between the two groups (t=1.192, p=0.2343). Of the 285 PD patients, 122 (42.8%) complained of either smell or taste impairment; 116 (39.3%) patients with PD complained of smell impairment and 26 (9.1%) complained of taste impairment. In the control group, only 5 subjects (8.2%) complained of smell impairment and none (0%) complained of taste impairment (Table 1, Fig. 1). Statistical analysis revealed that the frequencies of smell and taste impairments were significantly higher in patients with PD than in controls (smell impairment: χ²=21.717, p<0.0001; taste impairment: χ²=6.017, p=0.0142) (Fig. 1). Of the 122 PD patients who complained of smell impairment, 27 (9.5%) had anosmia, 51 (17.9%) had moderate smell impairment and 31 (11.9%) had mild smell impairment. No patient complained parosmia. Taste impairment was reported by 10 (8.3%) of 120 male patients and 16 (9.7%) of 165 female patients. There was no statistical difference in the taste impairment ratio between males and females (χ²=0.156, p=0.6930). The ratio was significantly higher in female patients compared to control subjects (χ²=4.311, p=0.0379), but this was not the case for male patients (χ²=1.795, p=0.1803) when compared with controls. Im-

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**Table 1. Demographic Characteristics of Parkinson’s Disease Patients with and without Taste Impairment and Controls**

<table>
<thead>
<tr>
<th></th>
<th>Patients</th>
<th>n</th>
<th>F/M</th>
<th>Age ±SD (years)</th>
<th>Disease duration (years)</th>
<th>H&amp;Y stage</th>
<th>MMSE</th>
<th>LED (mg)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Controls</strong></td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>61</td>
<td>41/20</td>
<td>70.9 ±8.6</td>
<td></td>
<td></td>
<td></td>
<td>396</td>
</tr>
<tr>
<td>Taste impairment</td>
<td>(-)</td>
<td>259</td>
<td>159/110</td>
<td>72.0 ±9.1</td>
<td>5.8 ±5.0</td>
<td>2.5 ±0.9</td>
<td>25.5</td>
<td>395</td>
</tr>
<tr>
<td>PD</td>
<td></td>
<td>26</td>
<td>16/10</td>
<td>72.8 ±7.4</td>
<td>5.4 ±3.7</td>
<td>2.7 ±1.1</td>
<td>24.9</td>
<td>429</td>
</tr>
</tbody>
</table>

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**Figure 1.** Percentages of smell and taste impairments in Parkinson’s disease patients and controls. *p<0.05 when compared with controls.
Table 2. Types of Taste Disturbance Observed in Patients with Parkinson’s Disease

<table>
<thead>
<tr>
<th>Taste disturbance</th>
<th>Number of patients (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ageusia</td>
<td>3</td>
</tr>
<tr>
<td>Hypogeusia</td>
<td>18</td>
</tr>
<tr>
<td>Dysgeusia</td>
<td></td>
</tr>
<tr>
<td>Bitter taste</td>
<td>1</td>
</tr>
<tr>
<td>Metallic taste</td>
<td>1</td>
</tr>
<tr>
<td>Enhanced hot perception</td>
<td>1</td>
</tr>
<tr>
<td>Painful and red areas on tongue</td>
<td>1</td>
</tr>
<tr>
<td>Burning mouth</td>
<td>1</td>
</tr>
</tbody>
</table>

Figure 2. Smell and taste impairments and disease severity in Parkinson’s disease patients. Percentages of smell (bottom) and taste (top) impairments are shown by Parkinson’s disease patients with Hoehn & Yahr stages 1 and 2, 3, and 4 and 5. *<0.05 when compared with Parkinson’s disease patients with Hoehn & Yahr stages 1 and 2.

Discussion

We determined the frequencies of smell and taste impairments by a clinical interview of PD patients and age- and sex-matched healthy controls and evaluated their symptoms. The results indicate that smell and taste impairments were found in 39.3% and 9.1%, respectively, of PD patients, and 8.2% and 0%, respectively, of controls. In our study, smell and taste impairments are evaluated by subjective report by patients and may not be as sensitive as those obtained by a quantitative evaluation such as the odor stick identification test for the Japanese (OSIT-J) and filter paper taste strip tests (TSTs). Therefore, there is a limitation to evaluate the degree of smell and taste impairments. The present findings, however, still indicate that the frequencies of smell and taste impairments are significantly higher in patients with PD when compared with controls, and that smell impairment occurs more frequently than taste impairment in PD patients. Using a self-completed questionnaire, Chaudhuri et al (12) reported that 26.0% of PD patients, with a mean age of 68.1 years, disease duration of 6.4 years and mean Hoehn and Yahr stage of 2.6, reported either smell or taste impairment. In the control group, only 7.3% of subjects reported smell and taste impairment. The results from their control subjects (7.3%) are almost the same as the results from the control subjects in the present study (8.1%). The frequency of smell or taste impairments in PD patients in our study was 42.8%, which is 16.8% higher than the results of Chaudhuri et al (26%). The older mean age of 72.4 years in our patients as well as the methods of data collection (self-completed questionnaire vs. interview) may have contributed to these differences, although the mean disease duration of 5.8 years in Parkinson's disease was significantly higher in PD patients at Hoehn and Yahr stage 3 (χ2=9.602, p=0.0019) and without taste impairment (χ2=3.840, p=0.050) when compared with controls (Fig. 4).
but not in male patients when compared with controls. This was evaluated by use of TSTs and electrogustometry. Kim et al (6) and Shah et al (6) and completed questionnaires. Our patients was shorter and the mean Hoehn and Yahr stage of 2.4 was milder than those in the report of Chaudhuri et al. They reported much lower values than results from other previous studies using psychophysical or electrophysiological testing of 74-97% (1-3) for smell impairment and 27% (6) for taste impairment. This large difference is the result of the higher sensitivity of these tests in detecting smell or taste impairment as compared to interviews or self-completed questionnaires.

According to our results and the results from previous reports, abnormal sense of taste in PD patients occurs less frequently than smell impairment. Sense of taste has been reported to be one reason we did not observe a significant difference between patients and controls in men. Shah et al (6) and Sienkiewicz-Jarosz et al (5) used electrogustometry and reported contradictory findings; the former group reported impairment of taste threshold in 27% of PD patients, while the latter reported lowered taste thresholds in PD patients. Such inconsistencies may be derived from the fact that electrogustometry may be sensitive for detecting taste thresholds but not sensitive enough to evaluate altered taste quality. Our results based on interviews make it impossible to evaluate subclinical changes in thresholds of smell and taste sense but can evaluate the patient reports of altered quality of smell and taste.

Taste impairment includes reduced, altered or sensitized sensation in taste perception. The most frequent symptom of taste impairment was ageusia or hypogeusia, a diminished taste perception, followed by altered and sensitized taste perception. Burning mouth was noted in one patient (Table 2). Background conditions could cause taste impairment and may be responsible for such different symptoms. More variation of symptoms in taste impairment than smell impairment may indicate a wider variety of factors related to altered taste in patients with PD.

In order to identify the risk factors for the development of taste impairment, we compared the clinical characteristics of PD patients with and without taste impairments. There were no differences in age, sex, disease duration, disease severity, cognitive impairment as indicated by MMSE score, or amount of dopamine replacement therapy determined by LED between the two groups. PD patients at Hoehn and Yahr stage 3, however, showed more frequent taste impairment than those at Hoehn and Yahr stages 1 and 2, although PD patients at Hoehn and Yahr stages 4 and 5 showed no significant increase. The small number of PD patients involved in this group may explain the results. Kim et al (7) denied the correlation with age, duration or severity of the disease, cognitive function or olfactory function in patients with PD. The majority of reports indicate that smell impairment does not correlate with disease severity and duration (1, 3, 13, 14). However, other authors report a correlation between severity of motor symptoms and olfactory dysfunction (15). In the present study, PD patients at Hoehn and Yahr stage 3 and those at stages 4 and 5 showed a higher frequency of olfactory dysfunction than those at stages 1 and 2. Our results suggest there may be some correlation between disease severity and smell impairment.

PD patients with smell impairment showed a higher frequency (25%) of taste impairment when compared with patients without smell impairment (9%). Because both impairments were tended to occur more frequently at advanced disease stages, common pathological changes may be considered for both impairments. Olfactory impairment in PD patients may be caused primarily by Lewy body related degeneration in the olfactory tubercle and/or limbic structures connected to the olfactory tubercle (16-19). The gustatory afferent nerves end in the nucleus of the solitary tract of the medulla from which the information runs to the thalamus and then to the cortex. Analogous to the olfactory pathway,
taste information also connects to the amygdala and hippocampus (20). Results from functional MR imaging indicate that cortical areas including the insula and orbitofrontal cortex also are involved in the processing of gustatory stimuli (21, 22). PD-related neurodegenerative changes responsible for taste impairment may progress in parallel with those for smell impairment. However, common pathological changes may not be the single cause of taste impairment, because PD patients without smell impairment still complain of taste impairment; 11 (6.4%) of 183 PD patients without smell impairment in the present study also complained of taste impairment. It can also be possible that the altered sense of smell itself produced taste impairment secondarily in some patients. Taste sense may be altered not only by central nervous system degeneration, but also by a variety of mental and physical conditions such as depression, reduced saliva secretion, poor oral hygiene, gastrointestinal diseases, zinc deficiency, medication and smoking (8, 9). These factors also may explain, in part, the causes of taste impairment complaints, especially in PD patients without smell impairments. For example, development of autonomic failure, some symptoms of which may occur in relation to olfactory dysfunction (23), may cause reduced saliva secretion, which in turn results in poor oral hygiene followed by taste impairment. It is also possible that neurodegenerative changes for taste impairment may have progressed concomitantly with subclinical neurodegenerative change for smell impairment.

Because taste impairment often results in reduced appetite and subsequent malnutrition, we should pay careful attention to reports of altered sense of taste in patients with PD.

Conclusion

Smell and taste impairments were found in 116 (39.3%) and 26 (9.1%) of 285 PD patients, respectively. These frequencies were significantly higher when compared with reports from age-matched controls (8% and 0%). The frequencies tended to increase with disease progress. The frequency of taste impairment was significantly higher in PD patients with smell impairment (14.3%) compared to those without smell impairment (6.4%). The pathology responsible for smell impairment may be related to taste impairment to some extent, but this may not explain the dysfunction in all patients.

The authors state that they have no Conflict of Interest (COI).

References


