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Swallowing in Parkinson's disease: How is it affected?

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ABSTRACT

Objectives: The aim of this study was to evaluate the swallowing in patients with Parkinson's disease (PD) using comprehensive and multimodal methods.

Patients and methods: The present study was conducted on 120 patients and 60 controls between January 2017 and January 2018. All participants' demographic data were recorded, and the swallowing of the subjects was evaluated by using several methods, including clinic, electrophysiologic, endoscopic and ultrasonographic procedure.

First, the swallowing functions of the patient and control groups were compared. Subsequently, the patients were divided into two groups as patients with (n = 63) or without (n = 57) dysphagia symptoms according to their clinical evaluation. Finally, the data of these three groups were compared.

Results: In comparison with healthy subjects, the swallowing evaluated by all diagnostic methods were affected in patients with PD. This effect was greater in patients with clinically symptomatic dysphagia. No difference was found between patients without dysphagia symptoms and healthy controls based on clinic, endoscopic and some electrophysiologic methods. Interestingly, thickness of all oral phase muscles in healthy controls were significantly higher than both dysphagic and non-dysphagic PD patients according to ultrasonography.

Conclusion: Although it is widely known that dysphagia symptoms in patients with PD usually occur in advanced stages and cause serious problems for patients, the present study establishes that swallowing functions may have been affected in early stage patients without dysphagia symptoms. Assessment of swallowing functions is important and should be also assessed in patients without dysphagia symptoms. Moreover, ultrasonographic method may be used in the diagnosis and follow-up of patients with PD.

1. Introduction

Parkinson's disease (PD) is a common neurodegenerative disorder and affects approximately 1% of the population over 60 years of age [1]. Patients with PD had a shorter survival time [2]. In patients with PD, the most common cause of pneumonia and bronchitis, which is important causes of mortality and morbidity, is the swallowing difficulty or disfunction unrecognized or undetected at the time [3,4].

More than 80% of patients with PD develop swallowing dysfunction, known as dysphagia, during the course of their disease. Swallowing dysfunction reduces quality of life and leads to insufficient medication intake, malnutrition, dehydration, and aspiration with subsequent pneumonia, which is a major cause of death in patients with PD [5]. A recent meta-analysis showed that the pooled prevalence of oropharyngeal dysphagia based on subjective outcomes in PD patients is 35% and increases to 82% by taking objective measures of swallowing dysfunction into account [6].

Dysphagia in PD occurs slowly and progresses in an insidious manner. Classical thought is that the incidence of dysphagia increases as the disease progresses [7]. It affects all three phases of swallowing [7].

Recent studies evaluated dysphagia in PD patients using videofluoroscopy (VF) and fiberoptic endoscopic examination of swallowing (FEES) as well as nonspesific clinical screen tests [8–10]. Videofluoroscopy (VF) is widely considered as gold standard, particularly for the analysis of the physiology of swallowing and the detection of aspiration in the pharyngeal phase. On the other hand, it has some disadvantages including the cost, qualitative nature rather than quantitative, and the raised risk of radiation exposure in repeated applications. Although FEES is cheaper than VF, it cannot be used to evaluate the oral phase. In addition, some methods such as electroneuromyography (EMG) and ultrasonography (US) have been used in a small number of studies [11–13]. In these studies, while EMG was used to evaluate the swallowing function (oral and pharyngeal phase) of patients by dysphagia

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limit and swallowing time interval techniques, US was used to evaluate the pharyngeal phase muscles as quantitative and real-time tracking.

As can be seen from these studies, dysphagia in PD was primarily considered as a pharyngeal phase disorder. Contrarily, some studies in recent years have reported that oral phase disorders-such as difficulty in keeping food or fluid in the mouth-are more prevalent in PD than pharyngeal phase disorders. Moreover this condition may be a predictor for severe dysphagia [14,15]. Dysphagia due to oral phase disorders, may cause pneumonia, malnutrition, dehydration and even death due to silent or apparent aspirations similar to pharyngeal phase disorder. That is why the oral phase is as important as pharyngeal phase in patients with PD.

Despite the reported importance of the oral phase, there is no study to evaluate the oral phase in detail and how it is affected in PD. Moreover, other recent studies regarding swallowing dysfunction have reported a period of latent-subclinical dysphagia before clinical dysphagia occurs [8,16–18]. These studies have shown that the prevalence of dysphagia, if are assessed by sensitive objective methods, is seen up to 80% in PD patients. However, there is no adequate research defining the details of this period in literature.

Due to the above reasons, we have tried to answer the following questions in this study:

1 How can the effects on the functional and anatomic nature of the swallowing in PD patients be evaluated in these phases with comprehensive and multimodal methods?
2 Is there any difference in swallowing between subclinical and clinical periods of dysphagia in PD patients?

In order to find accurate answers to these questions, we aimed to assess the swallowing function in PD patients in both clinical and subclinical period of dysphagia compared to healthy subjects by using multimodal anatomic and physiologic methods.

2. Materials and methods

This study was conducted on 180 subjects in our inpatient rehabilitation clinic with 120 PD patients, and 60 healthy volunteers with age- and sex-matched patient relatives or caregivers between January 2017 and January 2018. Patients were hospitalized in the rehabilitation clinic and evaluation parameters were applied in the clinic.

We included subjects aged between 50–75 years who were diagnosed with idiopathic PD by the same neurology specialist according to the criteria of the UK PD Association Brain Bank [19], had modified Hoehn and Yahr (H&Y) stage ≤ 3 and disease duration of at least 3 years, had not undergone drug changes for the past three months, and had not previously been treated for swallowing problems.

Exclusion criteria for all subjects were causes of swallowing disorder including history of malignancy, severe malocclusion, severe dental loss, temporomandibular joint disorders, facial, servical, orthodontic and thoracic surgery and/or trauma, metabolic and endocrine diseases, other progressive central and peripheral neurologic disorders, respiratory distress, smoking and alcoholism. Additionally, exclusion criteria for FEES and EMG methods were the presence of contagious or infectious disease, risk for bleeding and decompensated heart disease and known gastroesophageal reflux disease or presence of its symptoms, and motor and mental disability (Mini-Mental State Examination score of < 24) and bedridden.

Subjects were informed about the study and their written consents were obtained at the beginning of the study. The approval of the Ethical Board of the hospital was obtained, and the study was conducted in accordance with the principles of the Helsinki Declaration.

2.1. Demographic and disease characteristics

Demographic characteristics such as age, gender and education status were recorded for all subjects.Clinical characteristics including disease duration, stage of disease (modified H&Y) [20] and Unified Parkinson's disease rating scale (UPDRS) part II (activities of daily living), III score (motor symptoms), and total scores for impairment and disability were recorded only for the patient groups [21].

2.2. Evaluation parameters

All subjects were assessed for clinical symptom presence and severity by Eating Assessment Tool (Eat-10), for pharyngeal phase disorders by FEES and oropharyngeal phase disorders by EMG (swallowing time intervals and dysphagia limit), as well as oral and pharyngeal phase disorders by ultrasonographic evaluations (resting and contracting thickness of masseter, temporalis, orbicularis oris (OO) and resting of geniohyoid and anterior digastric (AD) muscles).

2.2.1. 10-item eating assessment tool (Eat-10) [22]

Dysphagia symptom severity were evaluated by Eat-10 tool which is a self-administered and symptom-specific outcome instrument for dysphagia. It consists of 10 questions and each question scores from 0 (no problem) to 4 (severe problem). If Eat-10 score is 3 or higher, it considered as " presence of dysphagia".

2.2.2. Flexible fiberoptic endoscopic evaluation of swallowing (FEES)

Endoscopic evaluation of patients was performed in sitting position by a non-ducted fiberoptic nasopharyngoscope of 3.4 mm diameter, a light source, camera, monitor, and DVD recorder (KarlStorz GmbH & Co KG, Tuttlingen, Germany). Local anesthetics were not used. To determine residue, aspiration or penetration up to 90 milliliters of water, yoghurt and a piece of biscuit were used as liquid, semisolid and solid foods. The findings were recorded as video images and examined to score the dysphagia levels of our patients with Dzeiwas [23] endoscopic evaluation protocol between 1-6. Score 1 was considered as "normal swallowing function" while scores 2–6 were considered as "dysphagia".

2.2.3. Swallowing electrophysiology

A dysphagia limit (DL) and swallowing time intervals were determined by the same specialist using Medelec Synergy (Oxford, UK) 10 channels EMG device, as noted in the previous studies. Electroneuromyography (EMG) records were taken from submental electrodes and a laryngeal sensor. Patients were subsequently provided with 1, 3, 5, 10, 15 and 20 ml water and they were asked to swallow with a single command. According to DL, patients were separated as "dysphagic" (with existence of any repetition and/or indications of aspiration during the swallowing within 8 s in any of each type of swallow) and "normal" (who drink 20 ml of water in one gulp). Also, as mentioned in previous studies [24], "0-2″ interval is the triggering time of swallowing reflex, the 'A-0′ interval indicates the duration of swallowing reflex, and the "A-C" interval is recorded as total oropharyngeal swallowing time.

2.2.4. Ultrasonographic (US) evaluation

All measurements were performed in supine with the head in a neutral position without using a pillow. Real-time imaging of cross-sectional thickness (for masseter, temporalis and OO muscles as bilaterally and genioglossus muscles), and cross-sectional areas (CSAs) (for right and left geniohyoid and bilateral anterior digastric muscles) were performed by ultrasound device (GE Logiq P5, General electric, Korea) and 7–12 MHz linear array transducer, by PMR specialist. The transducer was placed on the line and special care was taken to avoid excessive pressure. For the contracted state (except genioglossus), patients were instructed to hold for 3–4 s until the image was registered on the monitor. This scanning was repeated 5 min later to reduce the measurement errors.

2.2.4.1. Genioglossus muscle. The transducer was placed along the

midline of the long axis of tongue as well as submentally [12].

2.2.4.2. Masseter muscle. A line was drawn joining the lateral commissure of the mouth with the intertragic notch of the ear, crossing the masseter muscle. The angle of the probe was adjusted to produce the strongest echo from the mandibular ramus. The measurement site was at the thickest part of the masseter, close to the level of the occlusal plane, approximately in the middle of the mediolateral distance of the ramus which was achieved when the scan plane was perpendicular to its surface. Imaging and measurements were performed in two cases; as the teeth are occluded gently with the muscles were in a relaxed position and during maximal clenching with the masseter muscles in contracted state [25].

2.2.4.3. Temporalis muscle. The transducer was oriented in front of the anterior border of the hairline both in relaxed (in slight interocclusal contact) and contracted stages (clenching the teeth in the maximum intercuspal position) [25].

2.2.4.4. Orbicularis oris (OO) muscle. The transducer was placed near the angle of the mouth with keeping lips in the normal position for relaxed state and with a slight smile for contracted state [12].

2.2.4.5. Geniohyoid and anterior digastric (AD) muscles. Subjects were told to keep their tongues in their mouths as the most comfortable position. The distance between the inferior border of the mandible bone and the hyoid was measured and the skin was marked at one-third behind the lower border of the mandible. The transducer was placed in coronal plane to measure the CSAs of the muscles [12].

2.3. Study protocol

Clinic, EMG, FEES, and ultrasonographic evaluations were performed on the same day after taking the drug and during patients' "on" open period, respectively.

Subjects were evaluated by a physical medicine and rehabilitation (PMR) specialist in terms of Eat-10 scale. EMG and US evaluations were performed by another blinded PMR specialist as well as endoscopic evaluation was done by a blinded otolaryngologist.

2.4. Comparisons

Healthy subjects and patients were compared in term of all parameters. Afterwards, all patients were divided as "non-dysphagic" or "dysphagic" according to the Eat-10 screen test and all subjects were divided into three groups as; Group 1 (n = 63, patients with dysphagia), Group 2 (n = 57, patients without dysphagia) and Group 3 (n = 60, healthy volunteers). Three groups were compared among themselves in terms of evaluation methods.

2.5. Statistical analysis

Statistical Package for the Social Sciences (SPSS 22.0 for Windows) software package was used in the analysis of the data. The continuous variables were evaluated using the Kolmogorov-Smirnov test to determine whether or not they exhibited normal distribution. In descriptive statistics, the data were expressed as mean (standard deviation) for continuous variables, and as frequencies and percentages (%) for nominal variables using the chi-square test. Statistically significant differences between the groups in terms of normal undistorted continuous variables were analyzed with the Mann-Whitney *U* test and among the groups with Kruskal Wallis and Anova test with Tukey-LSD for post-hoc analysis. On the other hand, the significance of difference for nominal variables was analyzed using Fisher exact test. Values of $p < 0.05$ were considered statistically significant.

Table 1
Demographic characteristics of the patient groups and controls.

	Group 1 (n = 63) mean (SD)/n (%)	Group 2 (n = 57) mean (SD)/n (%)	Group 3 (n = 60) mean (SD)/n (%)	p
Age (years)	63.32 (8.67)	62.81 (11.91)	62.97 (7.79)	0.109
Gender	42 (66.7)	35 (61.4)	47 (78.3)	0.093
Male	21 (33.3)	22 (35.6)	13 (21.6)	
Female				
Educational status	3 (4.8)	0	3 (5)	0.234
Illiterate	7 (11.1)	8 (14.0)	6 (10)	
Under 5-year	35 (55.6)	32 (56.1)	39 (65)	
5-year	9 (14.3)	7 (12.3)	9 (15)	
8-year	5(7.9)	7 (12.3)	3 (5)	
11-year	4(6.3)	3 (5.3)	0	
More than 11 years				

SD: Standard deviation.

3. Results

The mean age of patients and control groups were 63.3 (SD 8.67) and 62.9 (SD 7.79), respectively. The majority of individuals were males in both groups (64.2% and 78.3%, respectively). The mean disease duration of patients was 9.27 (SD 5.13) years. The mean of modified H&Y score was 2.03 (SD 0.12), part II score of UPDRS was 15.93 (SD 10.08), part III score of UPDRS was 14.56 (SD 9.14), and total UPDRS score was 37.12 (SD 11.74).

Patients were divided into two groups according to the Eat-10 scores as group 1 (with dysphagia symptoms, n = 63) and group 2 (without dysphagia symptoms, n = 57). Patient and healthy (group 3) groups' demographic characteristics were similar, also three groups were similar, too (Table 1).

Comparison of clinical characteristics of patients are presented in Table 2. The mean disease stages of the patients was between mild to moderate.

Between patients (n = 120) and healthy groups (n = 60) in terms of all parameters results were found significantly different ($p < 0.05$).

Comparison of evaluation methods according to the three groups are demonstrated in Tables 3 and 4.

In post-hoc analysis; presence of dysphagia was significantly higher in dysphagic patients group (group 1) compared to both non-dysphagic (Groups 2) and healthy group (group 3) according to clinic, endoscopic and electrophysiological DL evaluation (for all, p = 0.001). There was no difference between Group 2 and 3 for these evaluation parameters (p = 0.918, p = 0.518, p = 0.692, respectively).

Three swallowing intervals including the triggering time of swallowing reflex (group 2 p = 0.001, group 3 p = 0.001), duration of swallowing reflex (group 2 p = 0.003, group 3 p = 0.001) and total oropharyngeal swallowing time (group 2 p = 0.003, group 3 p = 0.001) were longer in Group 1 than in the other groups. Also, there

Table 2
Clinical characteristics of the patients.

	Group 1 n = 63 mean (SD)	Group 2 n = 57 mean (SD)	p
Disease duration (years)	11.74 (6.48)	8.56 (3.81)	0.069
Modified H&Y (0–5)	2.08 (0.15)	2.04 (0.13)	0.541
UPDRS	16.12 (9.02)	15.48 (10.11)	0.521
Part II (0–52)	15.08 (10.72)	14.21 (8.11)	0.214
Part III (0–72)	37.31 (24.96)	35.74 (18.72)	0.516
Total score (0–199)			
Eat-10 (0–40)	9.76 (1.13)	1.07 (1.21)	**0.001**

SD: Standard deviation, H&Y: Hoehn and Yahr, UPDRS: Unified Parkinson's disease rating score, Eat-10: 10-item Eating Assessment Tool.

Table 3
Comparison of clinic, FEES and EMG results according to the groups.

	Group 1 n = 63 mean (SD), n (%)	Group 2 n = 57 mean (SD), n (%)	Group 3 n = 60 mean (SD), n (%)	P
Eat-10 score	0	57 (100)	60 (100)	0.001
Normal	63 (100)	0	0	
Dysphagia				
FEES	2 (3.2)	57 (100)	60 (100)	0.001
Normal swallowing	61 (96.8)	0	0	
Dysphagia				
Dysphagia Limit	0	56 (98.2)	60 (100)	0.001
Normal swallowing	63 (100)	1 (1.8)	0	
Dysphagia				
Swallowing intervals	956.15	525.20	451.38	0.003
(msn)	(185.56)	(176.23)	(176.02)	0.001
A-0 interval	419.37	285.31	178.14	0.001
0-2 interval	(217.20)	(196.51)	(154.72)	
A-C interval	1475.48	828.84	640.46	
	(386.59)	(224.34)	(220.71)	

SD: Standard deviation, FEES: Flexible Fiberoptic Endoscopic Evaluation of Swallowing, EMG: electroneuromyography.

Table 4
Comparison of results of theUS evaluation according to the groups.

	Group 1 n = 63 mean (SD)	Group 2 n = 57 mean (SD)	Group 3 n = 60 mean (SD)	P
Genioglossus (relaxed) (cm)	2.53 (1.58)	2.69 (1.76)	4.47 (2.09)	0.001
Masseter (relaxed) (cm)	0.67 (0.31)	0.84 (0.22)	1.23 (0.11)	0.001
Right	0.65 (0.27)	0.78 (0.23)	1.16 (0.14)	0.001
Left				
Masseter (contracted) (cm)	0.74 (0.27)	1.16 (0.18)	1.59 (1.21)	0.001
Right	0.73 (0.28)	1.14 (0.31)	1.57 (0.27)	0.001
Left				
Temporalis (relaxed) (cm)	0.37 (0.09)	0.41 (0.03)	0.59 (0.11)	0.021
Right	0.36 (0.11)	0.39 (0.07)	0.58 (0.04)	0.024
Left				
Temporalis (contracted)	0.42 (0.08)	0.44 (0.05)	0.69 (0.14)	0.012
(cm)	0.41 (0.06)	0.43 (0.08)	0.68 (0.11)	0.012
Right				
Left				
Orbicularis oris (relaxed)	0.38 (0.11)	0.39 (0.09)	0.58 (0.12)	0.024
(cm)	0.37 (0.12)	0.39 (0.12)	0.57 (0.08)	0.023
Right				
Left				
Orbicularis oris	0.40 (0.17)	0.42 (0.15)	0.68 (0.14)	0.015
(contracted) (cm)	0.39 (0.18)	0.41 (0.09)	0.67 (0.17)	0.011
Right				
Left				
Anterior digastric (relaxed)	0.63 (0.21)	0.92 (0.19)	0.95 (0.15)	0.026
(cm^2)	0.62 (0.18)	0.91 (1.21)	0.94 (0.12)	0.028
Right				
Left				
Geniohyoid (relaxed) (cm^2)	1.18 (0.68)	1.59 (0.31)	1.71 (1.24)	0.010

SD: Standard deviation; US: ultrasonography.

was significantly difference between Group 2 and 3 in terms of the triggering time of swallowing reflex ($p = 0.018$) and total oropharyngeal swallowing time ($p = 0.027$).

Ultrasonographic measurement thickness in oral phase muscles in Group 3 demonstrated thicker than both Group 1 and 2 for the genioglossus, masseter, temporalis and OO muscles in both relaxed and contracted state ($p < 0.05$).

Ultrasonographic measurement results were similar regarding the thicknesses in relaxed state of genioglossus, masseter, temporalis, and OO muscles as in contracted state of temporalis and OO muscles in Group 1 and 2 ($p > 0.05$). In group 1, the thickness in contracted state of masseter muscle was less than in group 2 (right/left, $p = 0.002$/ $p = 0.005$).

While the results of pharyngeal phase muscles in relaxed states of right/left AD and geniohyoid muscles were similar for Group 2 and 3 ($p > 0.05$), Group 1 measurements were lower than the results of Group 2 ($p = 0.028$, $p = 0.029$, $p = 0.013$, respectively) and Group 3 ($p = 0.009$, $p = 0.008$, $p = 0.002$, respectively).

4. Discussion

Parkinson disease (PD) is a disorder that is characterized by dysfunction of movement variables such as motor control, coordination and speed. The cardinal symptoms of PD such as akinesia/bradikinesia and rigidity can effect simultaneous and sequential movements of swallowing [26]. This condition affects swallowing function and disrupts the swallowing physiology that causes the prolongation of durations of laringeal elevation, pharyngeal phase and total swallowing, delays the triggering of swalowing reflex and eventually leads to aspiration [13,16]. The chronic denervation and reinnervation, developed secondary to general neurodegeneration in over 3 years disease, which leads to atrophy of swallowing muscles in pharyngeal phase of PD patients with dysphagia [27]. These structural changes can cause aspiration alike swallowing function disorder, too. Therefore, structural features of swallowing muscles such as muscle atrophy is important as well as the swallowing function and physiology.

In our study we performed with EMG and FEES methods for swallowing function and US for structural evaluation. We found that both the swallowing function and structural condition of pharyngeal phase in PD patients with dysphagia symptoms was disrupted compared to healthy subjects and patients without dysphagia symptoms.

Dysphagia symptoms have been shown to occur following degeneration of the brainstem in PD patients [28]. Therefore, results in PD patients with dysphagia are consistent with those reported in the literature. The interesting part of this study is our other results. In this study it was found that PD patients (with and without dysphagia) were significantly different regarding both structure and function in oral phase of swallowing compared to healthy subjects.

Although swallowing is traditionally assessed in three phases; oral, pharyngeal, and esophageal, these phases are closely related and should be considered as a whole. Swallowing abnormalities in the oral phase in PD include inappropriate swallowing plan, defect of sensory information from the cranial nerves, mastication insufficiency, impaired bolus formation and drooling disorders [16,29]. Oral phase abnormalities are an expected result in response to the impaired coordination of the affected pharyngeal phase muscles. However, this does not explain the oral phase disorder in patients without dysphagia symptoms by clinical test and with normal swallowing by the FEES method. The result suggests that the oral phase is affected in the subclinical period, unlike the literature.

Swallowing process begins with a pre-oral phase even before the bolus touches the lips. The brain is stimulated by smell and visual stimuli and the mouth is opened by muscle activation [28]. Oral taste sense, receptors, lip, tongue and chewing muscles and the bolus are formed voluntary under the control of cerebral cortex [28]. Uncoordinated and slowed-dyskinetic movements of oral muscles can result in lack of transition of the muscles from voluntary motions to sequental and reflex motions of pharyngeal phase, alike the deficits of other striated muscles in PD patients. But the pharyngeal phase function in these patients, may be evaluate normal due to the possible compensation mechanisms such as reduction of dysphagia limit and prolongation of oral phase duration. There are some electrophysiological studies showing that the dysphagia limit is low even though there is no serious dysphagia detected by the FEES method [13,16]. Although the dysphagia limit was normal in our patients without dysphagia symptoms, the duration from the “swallow” command to the triggering time of the swallowing reflex was long. These results show that swallowing

function in PD patients without symptoms and findings is also affected.

However, these results do not explain whether there is a permanent effect (structural changes) on swallowing in PD patients without symptom or finding. Therefore, as a topic not studied in the literature before, we measured the thickness and CSA of oral muscles in relaxed and contracted states. We found that the oral muscle thicknesses in patients without symptoms and findings were thinner than those of healthy ones.

Another interesting result is that, unlike the OO and temporalis muscles between patients with and without dysphagia, we found a decrease in the masseter muscle thickness only during contracted state. We think that this result depends on the muscles we choose. The perioral muscles such as OO have only a stable activity at the onset of the oral phase and provide bolus control by keeping food in the mouth [30]. Because of the anatomical position of the temporalis muscle, only a sling of the muscle is inserted to the coronoid process and is less effective than the masseter during mastication [31]. Therefore, these muscles do not constantly activate and contract during swallowing function. On the contrary, the masseter muscle has a regular activity pattern and produces maximum contraction when the mouth is fully open during the masticatory cycle and then ceases its activity when the mouth is almost closed [31]. The masseter muscle helps hyoid elevation and starts to contract at the beginning of the oral phase, assists pharyngeal phase muscles that are mainly active in hyolaryngeal elevation, and so its activity continues until the end of the swallowing. Studies in PD patients with dysphagia have reported that limiting the excursion of the mandible contributes to an increase in the time of oral preparation or chewing, which occurs as the first dysphagia symptom [32]. We have noted that masseter muscle contraction is reduced in patients without dysphagia compared to healthy subjects as well as that this reduction is different in PD patients with and without dysphagia. These results have shown that although the masseter is damaged early, this weakness can be compensated for the swallowing function without symptoms until the damage becomes apparent.

As a limitation of this study, we did not aim to evaluate the movements of the tongue muscles, which is known to have significant effects during swallowing function. We believe that the studies that evaluate the concept of swallowing with real-time US will gain clarity to our results.

5. Conclusion

Swallowing function in the oral phase may be impaired even in PD patients without dysphagia symptoms. However, due to compensatory mechanisms, it may not be noticed by patients. Assessment of swallowing is important and should be assessed in PD patients without dysphagia symptoms. Ultrasonographic (US) evaluation can be used as a simple and non-invasive method in the diagnosis and following the clinical course of patients with PD.

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Conflict of interest

All author declares no conflict of interest.

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