KEY WORDS: home care services; nutrition therapy; Parkinson’s disease; rehabilitation; sarcopenic dysphagia

INTRODUCTION

Parkinson’s disease (PD) is one of the most common neurodegenerative diseases worldwide, with a reported global prevalence of approximately 1% of the population aged 65 years or older. Patients with PD may be at higher risk of becoming malnourished than others of the same age because of dyskinesia, rigidity, and dysphagia, as well as medication side effects.1–3 More than 80% of PD patients develop dysphagia during the course of the disease. Moreover, dysphagia leads to malnutrition and aspiration pneumonia (AP), both of which are major causes of death in PD patients.4 Sarcopenic dysphagia is caused by a decline in muscle mass and total body strength, including the swallowing muscles.5 The key to treating sarcopenic dysphagia is combined therapy with rehabilitation and aggressive nutrition management...
management.\(^5\) Although some studies have reported the effects of combined therapy with rehabilitation and aggressive nutrition management in hospitalized patients,\(^5\) to our knowledge, no studies based in a home medical care setting have yet been published. Previous studies have reported that sarcopenia is mainly associated with the progression of parkinsonian syndromes, and PD may accelerate the progression of age-related sarcopenia.\(^2,\)\(^8\) There may also be common early pathways in both PD and sarcopenia, suggesting the existence of “extended neurodegenerative overlap syndrome”.\(^3\) However, it is unclear whether sarcopenic dysphagia is a complication of PD. In addition, whether combined therapy with rehabilitation and aggressive nutrition support in a home medical care setting is effective for sarcopenic dysphagia in PD patients remains to be elucidated. We herein describe a patient with PD who developed sarcopenic dysphagia and showed improvement through home-based combined therapy with rehabilitation and aggressive nutrition support.

This study was approved by the Ethics Committee of the Japan Primary Care Association (Approval Number and date: 2017–001), and the patient provided written informed consent for publication of this case report.

**CASE**

**Patient Profile**

A 72-year-old man presented with muscular rigidity, akinesia, and visual hallucinations that had worsened over the previous 7 months. He had been diagnosed with PD 17 years earlier, and at presentation was at Hoehn and Yahr stage 4. The patient had no other medical history or relevant family history. He had been taking medicine orally: 125 mg levodopa, benserazide hydrochloride three times/day, 25 mg zonisamide and 4 mg ropinirole once/day, and 4 mg trihexyphenidyl hydrochloride twice/day. Because the patient could no longer walk due to aggravation of his PD symptoms, he was hospitalized for medication adjustment. On admission, the patient was unable to eat anything because of visual hallucinations and the exacerbation of tremors of the upper limbs and lower jaw. Parenteral infusion was therefore started to supply water and electrolyte correction. Although the PD symptoms, such as muscular rigidity and lower jaw tremors, did not significantly improve, oral intake was resumed 5 days after admission, and the patient developed aspiration pneumonia (AP). Oral intake was therefore stopped and feeding via a nasal gastric tube was started. Dysphagia rehabilitation continued after the development of AP, and the patient attempted to resume oral ingestion several times. Despite these attempts, however, oral intake was finally stopped on day 31 because of repeated severe aspiration. The patient was advised to undergo percutaneous endoscopic gastrostomy, but he refused. By day 62 of hospitalization, the patient had lost weight (−33.8%, BMI: 15.3 kg/m\(^2\)), and the Barthel Index score had decreased from 90 to 30 points.

For further PD treatment and dysphagia rehabilitation, the patient was transferred to another hospital. After the transfer, he exhibited improvement of his PD symptoms, such as muscular rigidity and tremors of the upper limbs and lower jaw, after switching from oral to transdermal administration of 9 mg rotigotine, and changing the dose of levodopa, benserazide hydrochloride to 250 mg three times/day and zonisamide to 50 mg once/day, and starting on 2.5 mg selegiline hydrochloride once/day. The patient’s body weight (BW) remained 39.0 kg, but his PD symptoms improved after this medication adjustment. The Barthel Index score had increased from 30 to 45 points by day 96 after transfer to the second hospital and the patient was able to walk around his bed and to the toilet in front of the hospital room independently. Tube feeding was discontinued, but the patient’s dysphagia remained. At this point, because he had already been in hospital for more than 150 days, he expressed a strong wish to go home at the earliest opportunity, but also to continue dysphagia rehabilitation. Therefore, it was decided that he would be discharged on day 159 and continue dysphagia rehabilitation at home. By the day of discharge, a home-based speech-language pathologist (SLP) was given the patient’s information by a hospital SLP. This information included the detailed clinical course and medical problems such as low BW, malnutrition, and declining swallowing function. At discharge, the patient’s BW was 39.0 kg (−33.8%/4 months, BMI: 15.3 kg/m\(^2\)), the Barthel Index score was 45, and the Functional Oral Intake Scale (FOIS) level was 4, indicating total oral diet of a single consistency.\(^9\) The patient’s modified diet was level 4 for foods and level 1 for drinks, according to the International Dysphagia Diet Standardisation Initiative (IDDSI) framework.\(^10\)

**Assessment and Diagnosis on the First Day after Discharge**

The patient’s PD symptoms were controlled, although orthostatic hypotension and wearing-off were observed. He scored three points on the Mini Nutritional Assessment-Short Form, indicating malnutrition. The patient’s swallowing-related muscle strength declined, with the range of laryngeal elevation reduced to about 70%. His speed of laryngeal eleva-
vation was also reduced. Because the patient refused to have his swallowing function evaluated by videofluoroscopy or videoendoscopy, we applied a Modified Water Swallowing Test (MWST),11,12 a Food Test (FT),11,12 a Dysphagia Severity Scale (DSS) evaluation,12 cervical auscultation, visual inspection, and palpation of the laryngeal bulge and hyoid bone. The MWST and FT scores were three points, and the DSS score was four points, indicating occasional aspiration. The patient underwent regular dental checkups, did not have any oral cavity or dental problems, and did not need dentures. The evaluation of body composition with a bioelectrical impedance analyzer (InBody Japan Inc.; InBody 270) was not possible because the patient could not maintain the upright posture during measurement. Sarcopenia was confirmed by a calf circumference (CC) of 23.8 cm,13 handgrip strength of 22 kg, and gait speed of 0.5 m/s, according to the diagnostic criteria of the Asian Working Group for Sarcopenia.14

Because the PD symptoms were only minimally responsible for the patient’s dysphagia, its main cause was considered to be sarcopenia. However, we could not measure the tongue pressure because of the unavailability of a measuring device in the home-based intervention setting. Therefore, the patient was diagnosed with possible sarcopenic dysphagia in accordance with the diagnostic criteria and diagnostic algorithm for sarcopenic dysphagia.4,15

**Intervention**

Under the guidance of the SLP, home-based rehabilitation was conducted starting from the first day after discharge. This rehabilitation was performed independently by the patient or with the assistance of family members and comprised of the following three programs: neck muscle training, oral motor training, and daily activity training of about three metabolic equivalents (METs) per day for at least 1 h in total. The SLP visited once a week to re-evaluate swallowing function and activities of daily living (ADL), as well as to adjust the amount of training and the level of diet modification, depending on the patient’s swallowing function. Because a dietician could not intervene in this case, the SLP, who was certified as a clinician of the nutrition support team and capable of nutrition management, also conducted nutrition education for the patient and his family.

Initially, the patient’s energy requirement was calculated to be 1397.5 kcal/day, based on 25 kcal/kg ideal BW (IBW)/day.16 Energy expenditure during exercise, excluding energy consumption at rest, was calculated to be 78 kcal/day (2 MET*1 h*39 kg BW).17,18 Energy storage was calculated to be 642 kcal/day to achieve a weight gain of 11 kg over a period of 5 months, to bring the patient’s BW to over 50 kg (90% of IBW) and to eliminate malnutrition. The initial month was a warm-up period to increase nutritional intake with a modified standard meal and a snack, e.g., ice cream, a cream puff, or sweet red-bean soup. He did not take any fortified food or oral nutritional supplements, and ingested a balanced diet of carbohydrates, lipids, and protein three times a day. We adjusted lunchtime by about 30 min to 1 h depending on the PD symptoms, which often worsened around lunchtime.

The SLP set a target total intake of 2117 kcal/day by adding together the daily energy requirement, energy expenditure, and energy storage. The estimated nutritional intake was assessed by a food record. The patient’s intake was started at 1200 kcal/day orally, the same amount of energy as hospital meals, and was gradually increased. The absence of symptoms of refeeding syndrome, such as consciousness disturbance, convulsions, arrhythmia, vomiting, and diarrhea was carefully confirmed while increasing the amount of nutritional intake. We also increased awareness of health problems caused by overnutrition and started to evaluate the patient’s body composition with a bioelectrical impedance analyzer when he achieved 75% of IBW and became able to maintain the upright posture during measurement. The patient was able to consume the target amount of energy on day 35 after discharge. Subsequently, the patient’s activity levels increased significantly, and he could work on his garden at home for several hours a day. Once BW exceeded 42 kg (75% of IBW), the SLP recalculated the energy consumption during exercise to 660 kcal/day in up to four METs over a duration of 5 h and increased the patient’s maximum energy intake to 2800 kcal/day. When BW exceeded 90% of IBW, the SLP reset the energy intake to the standard value. Since the beginning of the intervention, to prevent AP, the SLP consistently had advised the patient to take a break from exercise without eating when he had symptoms such as the wearing-off phenomenon or orthostatic hypotension.

**Outcome**

By day 100 after discharge, the patient had gained 10.5 kg of weight, and by day 121 he had gained 12.1 kg, an increase of 31%. The patient’s percent body fat was 8.2%. He was able to eat all foods except hard food (MWST: 4–5, FT: 4–5, DSS: 6, FOIS: 6, IDDSI framework: level 6 for foods, level 0 for drinks) on day 100. By day 121 after discharge, his swallowing function returned to normal (MWST: 5, FT: 5, DSS: 6, FOIS: 7, IDDSI framework: level 7 for foods) (Table 1). On the first day after discharge, it took about 40–60 min to
finish a meal; however, this time had shortened to 30–40 min on day 42 after discharge. On day 121, the patient showed an increase in BMI (20.1 kg/m$^2$), muscle mass (SMI: 7.4 kg/m$^2$, CC: 32 cm), muscle strength (handgrip strength: 34 kg), physical function (gait speed: 1 m/s), ADL (Barthel Index score: 90), and Mini Nutritional Assessment-Short Form (12), indicating recovery from sarcopenia.

**DISCUSSION**

The clinical course of this patient indicated two important issues. First, sarcopenic dysphagia may be a complication in patients with PD. Second, home-based combined therapy with rehabilitation and aggressive nutrition management may be effective for PD patients with sarcopenic dysphagia.

We considered that the patient likely had possible sarcopenic dysphagia on the first day after discharge. However, before hospitalization, the patient was almost independent in terms of ADL, and his nutritional status was normal. However, inactivity resulting from his PD symptoms, prescribed rest for about 1 week after hospitalization, and malnutrition, led to sarcopenia during hospitalization. Furthermore, the patient developed pneumonia during hospitalization and his condition quickly deteriorated. In the second hospital, his PD symptoms were controlled. Although dysphagia remained, BW was maintained, and ADL improved. The patient’s drug-controlled PD symptoms remained unchanged after discharge. However, the sarcopenia treatment that he underwent at home alleviated not only his sarcopenia but also his dysphagia. A recent study has revealed a relationship between low numbers of motor neurons and sarcopenia and has suggested the presence of neurodegenerative components leading to sarcopenia. Consequently, we suspected that the main cause of our patient’s dysphagia was sarcopenia of the swallowing muscles, not PD. Because sarcopenic dysphagia occurs in conjunction with sarcopenia, sarcopenic dysphagia might be a complication of PD.

For sarcopenia and sarcopenic dysphagia in patients with PD, combined therapy with rehabilitation and aggressive nutrition management in a home medical care setting may be effective. PD patients tend to suffer from malnutrition, which is a risk factor of sarcopenia, because of an increase in muscle activity resulting from PD symptoms (e.g., muscle rigidity and tremors). For PD patients able to handle oral

**Table 1. Time course of the patient’s state at home**

| Days after discharge | 0 | 42 | 58 | 100 | 121 |
|----------------------|---|----|----|-----|-----|
| FOIS                 | 4 | 4  | 5  | 6   | 7   |
| IDDSI framework level for foods | 4 | 4  | 5  | 6   | 7   |
| IDDSI framework level for drinks | 1 | 1  | 1–0| 0   | 0   |
| MWST                 | 3 | 4  | 4–5| 5   |     |
| FT                   | 3 | 4  | 4–5| 5   |     |
| DSS                  | 4 | 5  | 6  | 6   |     |
| BW, kg               | 39.0| 41.8|44.0|49.5|51.1|
| %IBW, %              | 69.8| 74.8|78.7|88.6|91.4|
| BMI, kg/m$^2$        | 15.3| 16.4|17.3|19.4|20.1|
| SMI, kg/m$^2$        |     |     |6.3 |     |7.4 |
| BFP, %               |     |     |7.1 |     |8.2 |
| BI                   | 45  | 65  |    |     |90  |
| HS, kg               | 22.0| 27.0|    |     |34.0|
| GS, m/s              | 0.5 |     |    |     |1.0 |
| AC, cm (L/R)         |     |21/–|21.5/20.0|25.3/24.4|25.5/24.6|
| CC, cm (L/R)         | 23.8/–|24.8/–|25.0/26.3|30.0/30.0|32.0/30.8|
| MNA-SF               | 3   | 3   | 8  | 10  | 12  |
| Energy intake, kcal/day | 1200|1800–2800|2800|2400|1600–1800|

Before hospitalization, BW: 59 kg, height: 159.4 cm. AC, arm circumference; BFP, body fat percentage; BI, Barthel Index; BMI, body mass index; BW, body weight; CC, calf circumference; DSS, Dysphagia Severity Scale; FOIS, Functional Oral Intake Scale; FT, Food Test; GS, gait speed; HS, handgrip strength; IBW, ideal body weight; IDDSI framework, dysphagia diet framework of International Dysphagia Diet Standardisation Initiative; L/R left/right; MNA-SF, Mini Nutritional Assessment Short Form; MWST, Modified Water Swallowing Test; SMI, skeletal muscle mass index.
intake, home-based combined therapy with rehabilitation and aggressive nutrition management has advantages over hospital treatment. First, they can adjust to their on–off state rather than eating at a fixed time, as they would in a hospital. Second, they can eat as many times as they want, and it is easy to adjust the number of intakes/contents on a daily basis. Third, they can choose familiar or favorite meals and snacks. In the present case, we shifted the start time of the patient’s lunch by about 30 min to 1 h because his PD symptoms often worsened around lunchtime. In addition, familiar homemade tastes and between-meal snacks helped him to easily consume the number of calories needed every day.

However, despite the good results in this case, the clinical time course of the current patient might not be standard in the home-based intervention setting. One reason for the good clinical course was that the patient enthusiastically ate food and exercised, and we could increase the number of calories needed per day as planned and his weight increased. It could have been very difficult and time consuming if the amount of oral intake or exercise had not been maintained or increased as planned. Another reason was the ideal and smooth cooperation between hospital and home care. Information on the patient was received from the hospital SLP before discharge, and we were able to understand his condition and desired results. Consequently, we could carry out customized rehabilitation and nutritional support from the day of discharge.

The patient returns for follow-up visits to the hospital once a month, but refused blood analysis to prevent overnutrition and refeeding syndrome. Furthermore, it is difficult to measure tongue pressure for diagnosis of dysphagia in the home-based intervention setting because it is not covered by insurance. Moreover, it is also difficult to measure muscle mass by dual-energy X-ray absorptiometry in the home-based intervention setting. Consequently, we used CC measurement, which is positively correlated with skeletal muscle mass and skeletal muscle index, as a surrogate marker[13] to diagnose the current patient with sarcopenia.

The results of the current study demonstrate the potential utility of home-based combined therapy with rehabilitation and aggressive nutrition management for sarcopenic dysphagia in PD patients. For PD patients suffering from sarcopenia and dysphagia, healthcare workers may need to regularly intervene to improve the patient’s nutritional status and sarcopenic dysphagia.

In conclusion, sarcopenic dysphagia may be a complication of PD, and home-based combined therapy with rehabilitation and aggressive nutrition management may be effective for patients with this condition.

CONFLICTS OF INTERESTS

The authors certify that there are no conflicts of interest with any financial organization regarding the material discussed in this article.

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