Investigation of neurogenic dysphagia in commonly seen neurological diseases

Sık görülen nörolojik hastalıklarda nörojenik disfajinin araştırılması

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Abstract

Neurological disorders lead to varying degrees of impairment in the functions of vital swallowing structures, such as the cortex, cerebellum, brainstem, cranial nerves, and muscles. Neurogenic dysphagia is observed in approximately 50% of common neurological disorders such as stroke, multiple sclerosis, and Parkinson's disease. Although the pathophysiology and course of the disease vary, dysphagia may occur at any stage of swallowing, including oral preparation, oral, pharyngeal, and esophageal phases. Neurogenic dysphagia ranks among the top symptoms that restrict patients' independence in daily life activities, reduce their quality of life and increase morbidity and mortality rates. Despite being a prevalent and highly impactful symptom among patients, neurogenic dysphagia can go unnoticed among the multiple symptoms experienced by neurological disorders due to their nature. It is important to be aware of disease-specific risk factors for the early detection of neurogenic dysphagia. Overlooked dysphagia can lead to complications such as aspiration pneumonia, dehydration, malnutrition, and weight loss. Among these complications, aspiration pneumonia is the most common, requiring attention due to its recurrent hospitalizations, inpatient treatment, and high healthcare costs. Many patients exhibit common neurogenic dysphagia symptoms such as drinking liquids in small sips, cutting solid foods into small pieces, decreased appetite, and prolonged meal times. The aim of this study is to examine various aspects of neurogenic dysphagia in different neurological disorders, including its etiology, risk factors, symptoms, and prevalence.

Keywords: Demantia; multiple sclerosis; stroke; swallowing disorders

Öz

Nörolojik hastalıklar yutma için hayati öneme sahip korteks, serebellum, beyin sapı, kranial sinirler ve kaslar gibi yapıların fonksiyonlarında çeşitli derecelerde bozulmaya yol açar. Nörojenik disfaji; inme, multiple skleroz, parkinson gibi yaygın görülen nörolojik hastalıkların yaklaşık %50'sinde görülür. Hastalığın patofizyolojisi ve seyrine göre değişmekle birlikte yutmanın oral hazırlık, oral, faringeal ve özefageal fazlarının herhangi birinde disfaji ortaya çıkabilir. Nörojenik disfaji bu hastaların günlük yaşam aktivitelerindeki bağımsızlıklarını kısıtlayan, yaşam kalitelerini azaltan ve morbidite, mortalite oranlarını arttıran semptomlar arasında ilk sıralardadır. Nörojenik disfaji, yaygın görülen ve hastaları oldukça olumsuz etkileyen bir semptom olmasına rağmen nörolojik hastalıkların doğası gereği hastanın yaşadığı çoklu semptomlar arasında gözden kaçabilmektedir. Nörojenik disfajinin erken tespiti için hastalıklara özel risk faktörlerinin iyi bilinmesi önemlidir. Gözden kaçırılan disfaji ilerleyen dönemlerde aspirasyon pnömonisi, dehidrasyon, malnütrisyon, kilo kaybı gibi komplikasyonların oluşmasına neden olur. Bu komplikasyonlar arasında en sık görülen aspirasyon pnömonisi; tekrarlı hastane başvuruları, yatarak tedavi ve yüksek sağlık maliyetleri gerektirmesi nedeniyle dikkat edilmesi gereken bir durumdur. Hastaların birçoğu sıvıları çok yudumda içme, katı gıdaları küçük lokmalara bölme, iştahta azalma, yemek süresinin uzaması gibi ortak nörojenik disfaji belirtilerine sahiptir. Bu çalışmanın amacı farklı nörolojik hastalıklarda görülen nörojenik disfajinin oluşma mekanizması, risk faktörleri, semptomları ve prevelansı gibi yönlerini incelemektir.

Anahtar Sözcükler: Demans; inme; multipl skleroz; yutma bozuklukları

Received/*Geliş* : 06.02.2024
Accepted/*Kabul*: 30.05.2024

DOI: 10.21673/anadoluklin.1432672

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Neurogenic dysphagia

Swallowing is a neuromotor activity of vital importance. It consists of oral preparation, oral, pharyngeal, and esophageal phases. Swallowing's neuromotor control is carried out by cranial nerves trigeminal, abducens, facial, vagus, and hypoglossal. During swallowing, coordination and control extend to 25 pairs of muscles The term "dysphagia," comes from the Greek roots "dys," meaning "difficulty," and "phagein," meaning "to eat," refers to a swallowing disorder (1). Neurological disorders are the most common cause of dysphagia. Neurogenic dysphagia describes swallowing disorders caused by the central and peripheral nervous system, neuromuscular transmission, or muscle diseases. Neurogenic dysphagia is one of the most common and dangerous symptoms of many neurological diseases. It is estimated that approximately 50% of all patients suffer from neurogenic dysphagia (2). Clinical effects of neurogenic dysphagia include aspiration pneumonia, dehydration, malnutrition, decreased quality of life, and inability to intake food orally. These complications associated with dysphagia can lead to mortality in neurological diseases (3). Understanding the complex patterns of irregular swallowing is crucial in cases of severe dysphagia. This knowledge is necessary for healthcare professionals to accurately diagnose and effectively treat individuals with neurogenic dysphagia. By understanding the neural pathways and mechanisms involved in swallowing, healthcare professionals can develop appropriate therapeutic strategies to address the specific challenges faced by patients with neurogenic dysphagia. This review aims to facilitate the recognition of the differences necessary for the appropriate diagnosis and treatment of swallowing disorders. Additionally, it aims to assist patients, caregivers, and healthcare professionals in acquiring the necessary knowledge to live with the condition.

Stroke

Stroke is a neurological condition that occurs due to occlusion or rupture of arteries supplying the brain, resulting in the sudden loss of brain functions and a neurological presentation lasting longer than 24 hours. Motor function impairments, such as dysphagia, are common in individuals experiencing vascular damage to the central nervous system (4). The incidence of dysphagia in stroke varies between 19% and 81% and is often associated with aspiration pneumonia (5, 6). The prevalence of dysphagia increases from 38% when diagnosed through clinical swallowing examinations to up to 75% when evaluated using instrumental techniques (7). Stroke patients with dysphagia have a threefold increase in the risk of aspiration pneumonia, and those with aspiration have an elevenfold increase in the risk of pneumonia (8). Advanced age, high scores on disease-specific classifications, low quality of life index score, malnutrition, low body mass index, larger lesion volume, subcortical and cortical involvement, brainstem involvement, corticobulbar system involvement, white matter involvement, dysarthria, presence of dysphonia or reduced maximum pitch, cognitive impairment or dementia, male gender, higher Glasgow Coma Scale score at admission, presence of hemorrhagic or ischemic stroke, involvement of the anterior or middle cerebral artery, large or cerebral artery involvement, small vessel occlusion, facial palsy, presence of hyperlipidemia, and thalamic lesions are predictive factors determining the prognosis of dysphagia following stroke (9).

Disruptions in swallowing physiology occur as a result of cerebral, cerebellar, or brainstem strokes, and the severity and pattern of dysphagia vary based on the lesion's localization. Dysphagia resulting from brainstem strokes (40-81%) constitutes the majority of dysphagias observed after stroke (10). Retrospective studies indicate that strokes occurring in the infratentorial region, including the brainstem (midbrain, pons, medulla), and the cerebellum, lead to more severe dysphagia compared to strokes in the supratentorial region, involving deep (thalamus and basal ganglia) or lobar (cortex and subcortical areas) intracerebral regions (11). Infratentorial lesions primarily cause dysphagia due to motor deficits, while supratentorial lesions often result in dysphagia originating from sensory afferent deficits (12). The nucleus tractus solitarius, responsible for initiating swallowing, and the nucleus ambiguus, coordinate the synchronized contraction of lingual, laryngeal, pharyngeal, and muscles. Damage to these neural pathways in the cerebellum diminishes its inhibitory effect on the cerebral cortex, leading to dysphagia (10). Cerebral lesions disrupt the inten-

tional control of chewing and bolus transfer during the oral phase. Cortical lesions involving the precentral gyrus cause impairment in contralateral tongue, lip, and facial motor control, leading to disturbances in contralateral pharyngeal peristalsis. Cerebellar lesions impact cognitive functions, resulting in concentration or selective attention deficits that negatively affect swallowing control. Brainstem lesions affect oral, lingual, and cheek sensation, the timing of pharyngeal swallowing initiation, laryngeal elevation, glottic closure, and cricopharyngeal relaxation (13). The right hemisphere contributes more to the sensory-motor integration of swallowing than the left hemisphere (14). Severe dysphagia and aspiration are more commonly observed with right hemisphere involvement, while oral disorders are more frequent with left hemisphere involvement (Figure 1) (15).

Aspiration, the most severe symptom of dysphagia, is observed in approximately 40% of acute stroke patients. Silent aspiration, primarily caused by reduced sensory input or decreased cough reflex, constitutes the majority of these cases. Stroke-related pneumonia, due to its association with increased mortality, disability rates, and prolonged hospital stays, is a crucial symptom that requires attention (16). Dysphagia leads to malnutrition, dehydration, low-grade fever, loss of appetite, weight loss, wet voice, feeling of blockage in the throat, and airway obstruction. During the oral preparation stage, chewing disorders, prolonged chewing, weak lip seals, drooling while chewing, or solid food pocketing in the cheeks may occur. In the oral propulsion stage, difficulty initiating swallowing is observed, and in the pharyngeal stage, symptoms such as inability to swallow, choking, nasal regurgitation, coughing, shortness of breath, and pain during swallowing are prevalent. In the esophageal phase, symptoms such as belching or indigestion due to reflux, recurrent pneumonia, bad taste in the mouth upon waking, heartburn, chest pain, or discomfort may occur (17).

As a result, one of the common causes of dysphagia is considered to be stroke. Stroke can have significant adverse effects on swallowing physiology, impacting both oral and pharyngeal stages of swallowing. The severity and pattern of dysphagia following cerebral, cerebellar, or brainstem strokes vary depending on the localization of the brain lesion.

Dementia

The prevalence of dementia, and dysphagia associated with dementia increases with aging. Various assessments are conducted in individuals suspected of dementia, but establishing a definitive diagnosis takes time. When dementia is mentioned, cortical changes related to cognitive impairments are typically what come to mind. However, upon examining MRI images from the early stages, damage to the brainstem region involved in swallowing control is observed (18).

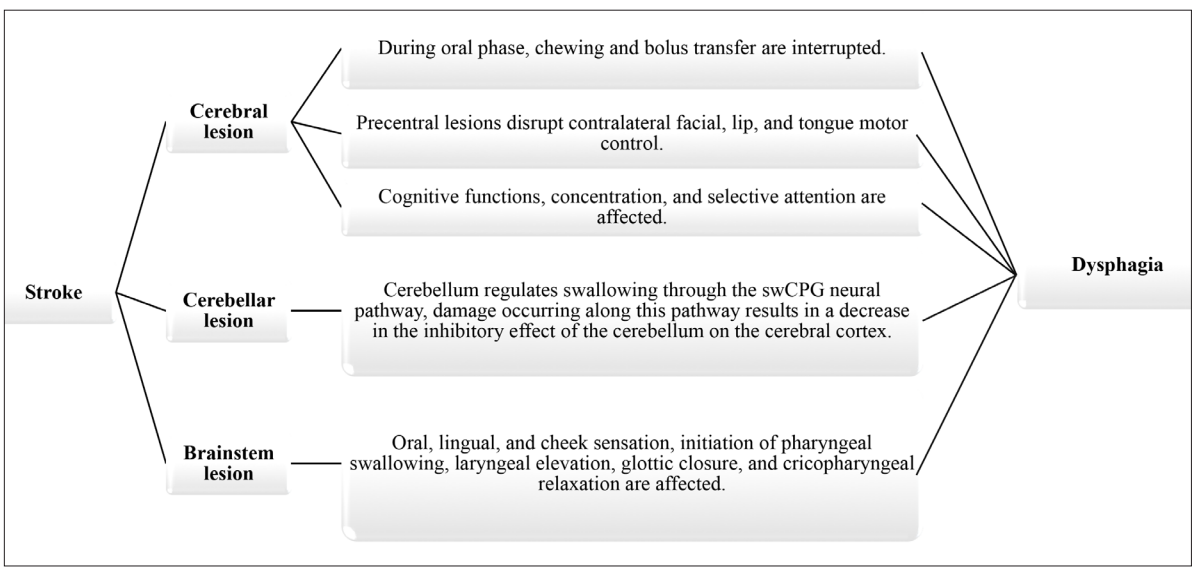

Figure-1. Pathophysiology of Dysphagia in Stroke

Swallowing difficulties observed in the early stages of dementia in patients are attributed to the natural aging process (19). This situation leads to delayed diagnosis of dysphagia in dementia patients and can result in an increase in the complication rate, potentially culminating in mortality in advanced stages (20). Dementia patients experience problems with dysphagia such as increased leakage duration during chewing, prolonged initiation time of swallowing, premature bolus spillage, and decreased bolus clearance (21). Although the prevalence of dysphagia in dementia patients is estimated to be around 45% on average, it varies depending on clinical features such as dementia type, disease stage, and process (22). Apart from those listed below, neurogenic dysphagia is also experienced in frontotemporal and lewy body types of dementia (23).

Alzheimer's disease

Alzheimer's disease, which constitutes 60-70% of all dementia cases, is characterized by progressive cognitive, behavioral, and neuropsychiatric symptoms attributed to hippocampal and bilateral temporal-parietal lobe involvement (24). In Alzheimer's disease, the sensory aspect of swallowing is affected due to neurofibrillary tangles originating from neuritic plaques and olfactory pathway involvement. Dysphagia develops in 84% to 93% of moderate to severe Alzheimer's patients due to impaired cognitive functions, leading to visual recognition inability of bolus and oral-tactile agnosia. Consequently, symptoms such as decreased smell and taste sensations during the oral phase, impaired perception of food in the mouth, inability to initiate voluntary swallowing, and delayed oral transit of liquids are observed (25). The presence of dysphagia in Alzheimer's patients leads to increased hospital admissions, inpatient treatment, and healthcare expenditures (26). Therefore, it is important to be vigilant against risk factors such as difficulty in oral/labial residue clearance and bolus cleaning for early detection of dysphagia (27).

Vascular dementia

In the second most common type of dementia, vascular dementia, the motor aspect of swallowing is affected more than the sensory aspect (28, 29). Disturbances are observed in the chewing of semi-solid food and the formation of the bolus due to the involvement of the trigeminal and hypoglossal cranial nerves responsible for chewing and tongue movements. Patients with vascular dementia experience problems not only in the oral phase but also in the motor components of the pharyngeal phase, including hyolaryngeal movement and epiglottic inversion. Compared to Alzheimer's disease, vascular dementia is associated with a higher incidence of overall aspiration and silent aspiration (29).

Parkinson's disease

Parkinson's disease (PD) is a complex neurodegenerative progressive disorder that affects both motor and non-motor functions. In pathophysiology, dopaminergic neuron loss occurs due to the decrease in dopamine in the basal ganglia and the accumulation of cytoplasmic residues containing the insoluble α-synuclein (αSyn) protein, called Lewy bodies, in more than one cell type (30). Dysphagia, which is difficulty in swallowing, is a common non-motor symptom experienced by people with PD. It can affect any stage of the disease and any of the oral, pharyngeal, esophageal phases It can affect any phase of swallowing, including oral, pharyngeal, and esophageal stages of the disease (31). Swallowing disorders can occur in the early stages or even in the prodromal stage of PD, even if they are typically more noticeable in advanced stages (31, 32). Unlike stroke, dysphagia in PD often worsens with the progression of the disease and appears approximately 10-11 years after the onset of motor symptoms (33, 34). The prevalence of dysphagia in PD varies depending on disease duration, stage, and assessment method (35). There are differences between the prevalence of dysphagia reported subjectively (35%) and that confirmed by instrumental methods (85%) (33). Due to deficiencies in sensorimotor functions and a decreased protective cough reflex, many individuals with PD may not be aware of their swallowing issues. This lack of awareness often leads to asymptomatic dysphagia in the early stages, subsequently resulting in silent aspiration and/or penetration (36, 37, 38). However, several symptoms and signs can contribute to the early diagnosis of dysphagia in PD. These include prolonged meal duration, difficulty swallowing tablets, a sensa-

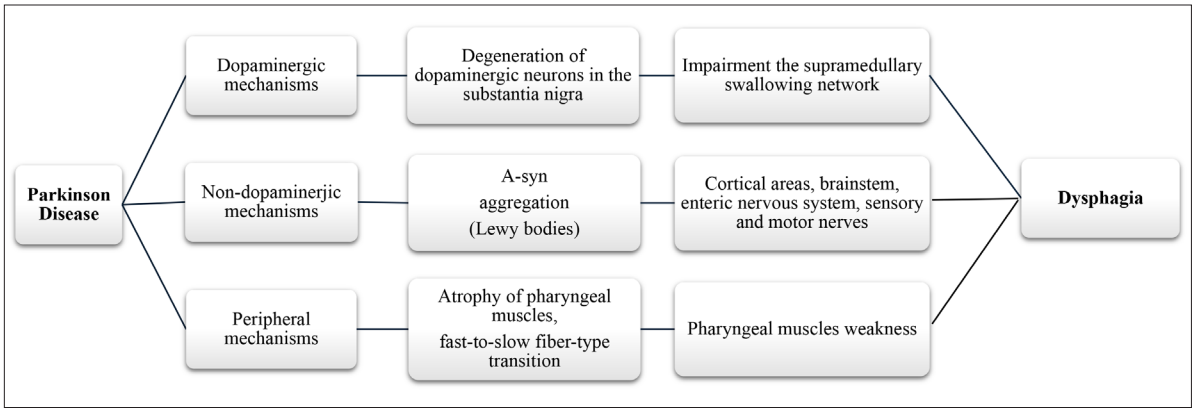

Figure-2. Pathophysiology of Dysphagia in Parkinson Disease

tion of food sticking or persisting in the throat after swallowing, coughing and choking during food and liquid intake, changes in voice (e.g., gurgling voice), and weight loss or low body mass index during recurrent chest infections (39). Even if not exhibiting these symptoms, Parkinson's patients in Hoehn and Yahr stages 4 and 5, with dysphagia-related weight loss or a body mass index <20 kg/m2, drooling or sialorrhea, and clinical predictors such as dementia, should be assessed for screening (40).

In the development of dysphagia in PD, both non-dopaminergic and dopaminergic mechanisms may be involved (41). The extrapyramidal dysfunction arising from the degeneration of dopaminergic neurons in the substantia nigra plays a significant role in the pathophysiology of dysphagia in PD patients by impairing the supramedullary swallowing network crucially involved in swallowing. This hypothesis is also supported by the observation that some PD patients show a significant improvement in swallowing function after L-Dopa and that deep brain stimulation may affect the supramedullary swallowing network by modulating dopaminergic pathways (42). Lewy bodies, which are αSyn aggregates involved in the pathology of PD, have been found to accumulate in various non-dopaminergic cortical areas, including 'central pattern generators' in the medulla, brainstem, enteric nervous system and motor nerves innervating the pharyngeal muscles, which can disrupt the swallowing pattern (43). Peripheral mechanisms, such as atrophy of pharyngeal muscles and fast-to-slow fibre-type transition, also contribute to swallowing dysfunction (Figure 2) (44, 45).

The hallmark features of dysphagia in PD include repetitive or festinated tongue movements, decreased coordination and speed of mastication, significantly prolonged oropharyngeal transit time, pharyngeal spillage, and delayed initiation of swallowing (40, 46). Additionally, studies report increased swallowing frequency for clearing residues in the pharynx, esophageal dysmotility and slowed hyolaryngeal movements. Deficiencies in airway protection may manifest as decreased cough airflow rates, reflex, and airway obstruction due to sensory impairments. (33, 46). Dysphagia can significantly affect the quality of life in patients with PD. Swallowing problems adversely affects well-being, self-confidence, and social integration, leading to frustration and social isolation (39, 46).

In conclusion, dysphagia occurs in PD through dopaminergic, non-dopaminergic, and peripheral mechanisms. Dysphagia can be seen at any stage of the disease, but as the disease progresses, the symptoms worsen and negatively affect the quality of life. Dysphagia in PD causes aspiration pneumonia, leading to severe complications and even death.

Chorea, dystonia, and Wilson disease, which are included under the term movement disorders, are other diseases in which neurogenic dysphagia is observed (23).

Multiple sclerosis

Multiple sclerosis (MS) is the most common neurodegenerative disease of the central nervous system, characterized by chronic inflammation and demyelination (47). The formation of plaques in areas such as the cortex, cerebellum, brainstem, and cranial nerve

regions, which have a functional impact on swallowing, as a result of inflammation, leads to dysphagia in patients with MS (48).

The prevalence of dysphagia in MS patients varies between 21% and 90% (48, 49), with an average estimate suggesting that approximately one out of every three patients has swallowing difficulties (50). Many factors, such as age, affected region, and disease type, influence the prevalence and severity of dysphagia in MS patients (51). It has been reported that MS patients with involvement of the brainstem, forebrain, and central pattern generator regions responsible for controlling swallowing function are more likely to experience dysphagia than other patients (52, 53). Studies investigating the relationship between disease type and prevalence have shown that patients with progressive forms such as primary progressive MS (PPMS) and secondary progressive MS (SPMS) are more affected by dysphagia compared to those with relapsing-remitting MS (RRMS) (48, 54). The clinical course of dysphagia may vary for different MS types. In RRMS, dysphagia can last from 2-3 weeks to 2-4 months during exacerbations and can become chronic during remission periods, whereas for progressive types, there is a continuous increase in the severity of dysphagia (55). Another factor influencing the prevalence of dysphagia is the method of evaluating swallowing function. When evaluated with instrumental techniques, dysphagia prevalence increases to 65.7%, compared to 38.4% diagnosed through clinical swallowing examinations (51).

In the early stages of the disease, dysphagia can go unnoticed among multiple symptoms, and unless serious complications such as aspiration pneumonia develop, dysphagia may be neglected (56), with only 2% of patients diagnosed with dysphagia receiving treatment (57). This delay in intervening in dysphagia may lead to more severe dysphagia and increased disability and mortality rates in the advanced stages of the disease (58, 59). To detect overlooked dysphagia earlier, it is important to be aware of risk factors such as the use of certain medications in MS treatment, the initial symptom being swallowing problems, a high EDSS score (>8), and having motor and cerebellar symptoms, as well as disease duration and progressive disease course (60, 61).

Common signs of dysphagia in MS patients are frequent sips to drink fluids, dividing food into small morsels, and weight loss (60). Dysphagia typically stems from glottal closure issues, weakness in soft palate/laryngeal movements, and penetration during the pharyngeal phase. In rare cases, delayed swallowing reflex or residue transportation during the pharyngeal phase can lead to penetration (58, 62). In some instances, dysphagia is characterized by delayed swallowing reflex and difficulty in bolus manipulation

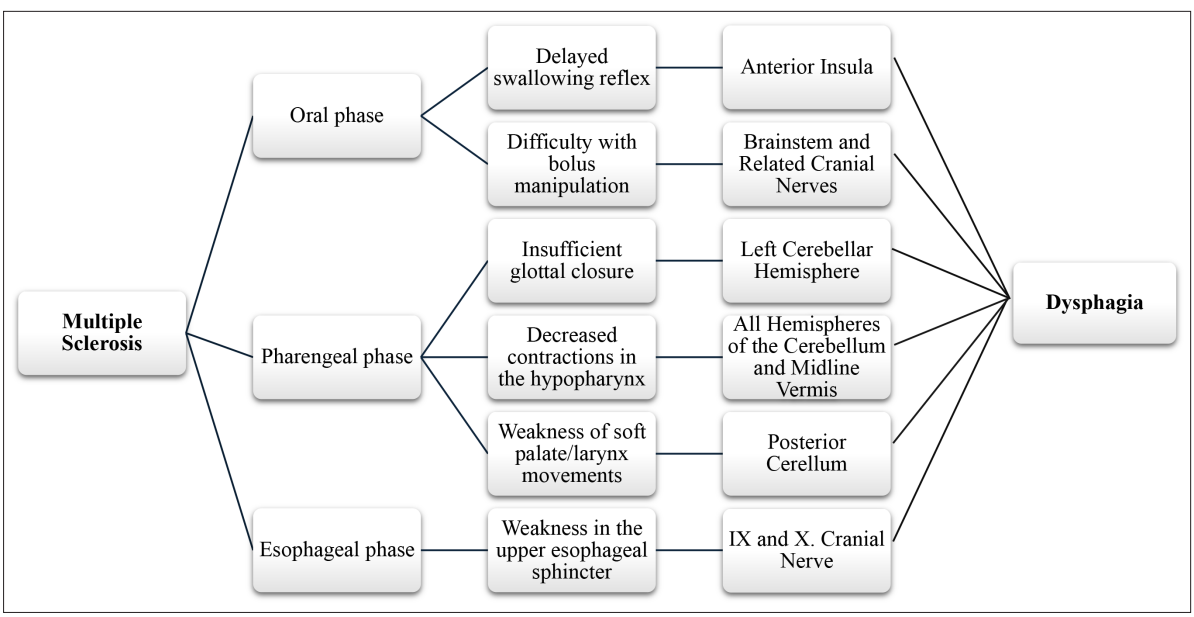

Figure-3. Pathophysiology of Dysphagia in Multiple Sclerosis

during the oral phase. In the advanced stages of MS, dysphagia may be linked to reduced contractions in the hypopharynx or dysfunction of the upper esophageal sphincter (61). Additionally, dysphagia in the oral phase may arise from cerebellar problems, while dysfunction in brainstem function, cognitive functions, and/or cerebellar disorders may contribute to dysphagia in the pharyngeal phase (Figure 3) (63).

In conclusion, dysphagia associated with MS can affect the sensorimotor cortex, cerebellum, brainstem, and cranial nerves depending on the localization of inflammation and demyelination in the central nervous system. Dysfunction in these structures, each vital for swallowing function, can lead to a range of problems such as oropharyngeal sensory deficits, weakness in muscles involved in chewing and swallowing, impaired initiation of voluntary swallowing, disruption of the swallowing reflex, and disturbance in the timing and sequencing of activation of structures involved in swallowing, as well as coordination problems among interacting structures. Complications related to dysphagia can occur in MS patients. Dysphagia should be screened for early using subjective and objective assessment methods regardless of the presence of symptoms in MS patients with various risk factors.

Bacterial and viral meningoencephalitis, tetanus, poliomyelitis and post-polio syndrome, which are included under the term ınflammatory diseases of the central nervous system, are other diseases in which neurogenic dysphagia is observed (23).

Brain tumors

Dysphagia is a prevalent complication of brain tumors, and it is directly associated with the effects of the lesion, tumor resection, and the outcomes of chemotherapy and radiation therapy. Dysphagia observed in brain tumors can lead to complications such as aspiration pneumonia, dehydration, and inadequate nutrition, significantly impacting long-term quality of life and mortality (64). Among all cancer types, dysphagia is most commonly seen in head and neck cancers, with an incidence rate of 89%. Swallowing function activates a complex communication network in the brain, involving bilateral sensorimotor cortex, premotor area, primary motor area, supplementary motor area, insular cortex, inferior frontal gyrus, cingulate gyrus, temporal lobe, inferior parietal lobule, precuneus, cerebellum, basal ganglia, and brainstem. Due to the intricate nature of swallowing, dysphagia can emerge from a tumor-induced lesion in the brain. The nucleus ambiguus, responsible for generating motor responses in muscles, and the nucleus tractus solitarius (NTS), responsible for initiating and maintaining swallowing based on bolus characteristics (size, texture, and temperature), are crucial structures for swallowing (23). In infratentorial brain tumors, compared to supratentorial brain tumors, penetration, aspiration, residue accumulation in the valleculae and piriform sinuses, and weakened swallowing reflex are more common, leading to an increased incidence of dysphagia. When comparing the incidence of dysphagia between benign and malignant tumors, there is no significant difference. Dysphagia arising from brain tumors is known to share similar characteristics with dysphagia resulting from stroke, as both conditions are outcomes of neurological interactions occurring in the same region (64). Tumor-related neurogenic dysphagia is also observed in metastases, neoplastic meningitis, and paraneoplastic syndromes under the umbrella term of brain tumors.

Neuromuscular diseases

Neuromuscular diseases (NMDs) are pathologies involving motor units and sensory nerves, often rooted in genetic factors. The clinical course and manifestations of NMD vary depending on the underlying pathology, with common symptoms including voluntary muscle weakness, muscle cramps, changes in muscle tone, balance problems, and difficulty swallowing (65). Dysphagia in adult NMDs typically affects the oropharyngeal phase, usually due to weakness in vocal cords and pharyngeal muscles. Dysphagia in NMD individuals contributes to decreased quality of life and may lead to increased morbidity and mortality (2, 66). Dysphagia is prevalent across a broad spectrum of NMDs, accompanying the clinical course and manifestations of the disease. Pathologies within this spectrum include Oculopharyngeal Muscular Dystrophy (OPMD), Myotonic Dystrophy (MD), Facioscapulohumeral Muscular Dystrophy (FSHD), Duchenne

Muscular Dystrophy (DMD), Myasthenia Gravis (MG), Amyotrophic Lateral Sclerosis (ALS), , and Spinal Muscular Atrophy (SMA) (67).

OPMD, although a prototypical adult-onset rare hereditary myopathy, presents dysphagia as the initial symptom in 32% of individuals around their fifties (68, 69). Patients often complain of difficulty swallowing solid foods, the sensation of throat blockage, and symptoms like coughing and choking during meals (68, 70, 71). In Myotonic Dystrophy, prolonged contractions and relaxations of swallowing muscles such as the sternocleidomastoid, masticatory muscles, and cricopharyngeal (upper esophageal) sphincter result in dysphagia. The prevalence of dysphagia in MD ranges from 25% to 80%, depending on the duration and severity of the disease (72, 73). FSHD patients develop dysphagia due to weakness in the muscles around the mouth, leading to difficulty in bolus formation. While 25% of patients report symptoms like prolonged meal times and fear of choking, most do not complain of dysphagia (74). In DMD, increased tongue thickness leads to impairment in the oral phase of swallowing. Delayed oral phase problems during swallowing and pharyngeal residues are also believed to reduce tongue strength (75, 76). Approximately 15% of MG patients develop fatigue-related dysphagia, characterized by weakness associated with effort and increased residue with repeated swallowing attempts. In advanced stages of the disease, myasthenic crises occur in 50% of individuals, potentially leading to aspiration due to dysphagia (77). ALS, characterized by progressive degeneration of lower and upper motor neurons, manifests early weakness in head and neck muscles due to corticobulbar pathway involvement. About 30% of patients present with dysphagia at diagnosis, which progresses with the disease (78). Dysphagia in ALS presents as premature spillage and a combination of hypopharyngeal residues in different areas (3). In patients with corticobulbar involvement, swallowing difficulties typically start with decreased tongue mobility, reduced pharyngeal contraction, and difficulty manipulating the bolus. Aspiration due to reduced laryngeal elevation is also common (79, 80).

In conclusion, dysphagia caused by voluntary muscle weaknesses, affecting chewing and swallowing muscles, is one of the clinical symptoms of NMD. Particularly, as the disease progresses and affects esophageal phase muscle activations, dysphagia severity can impact quality of life, morbidity, and mortality rates.

Traumatic brain injuries and spinal cord injuries

Dysphagia is a common complication of traumatic brain injuries (TBIs) and spinal cord injuries. TBIs result in diffuse axonal injury, brain contusion, and/or hematoma-related damage, leading to pathophysiological mechanisms such as edema, increased intracranial pressure, hypoxia, ischemia, and inflammation. Particularly, cervical spine injuries and surgeries, especially at the C3-C5 level, can cause neurogenic dysphagia (81).

Psychogenic dysphagia

Psychogenic dysphagia is observed during adulthood and is more frequent in females. Unlike other types of dysphagia, typical complications such as aspiration pneumonia are not seen in psychogenic dysphagia, and no pathology is usually detected in neurological examinations. It is commonly encountered in individuals with depression, anxiety, and an introverted personality (23). Psychogenic dysphagia encompasses conditions such as globus pharyngis and phagophobia, often confused with eating disorders, arising from psychosocial stress (81).

Conclusion

Dysphagia is a multifaceted and pervasive issue with significant implications for individuals across various neurological conditions. Neurogenic dysphagia, stemming from disorders affecting the central and peripheral nervous systems, neuromuscular transmission, or muscle diseases, presents a range of challenges impacting both quality of life and mortality rates. From strokes to dementia, Parkinson's disease to multiple sclerosis, and various neuromuscular diseases, dysphagia manifests differently in each condition, reflecting the complex interplay of neural pathways and muscular functions involved in swallowing. Understanding the distinct patterns and mechanisms of dysphagia in each neurological disorder is crucial for accurate diagnosis and effective management. Healthcare profes-

sionals must navigate through the intricate neural networks and motor processes implicated in swallowing to develop tailored therapeutic strategies. Moreover, early detection and intervention are paramount to mitigate complications such as aspiration pneumonia, dehydration, malnutrition, and diminished quality of life associated with dysphagia.

Furthermore, dysphagia extends beyond purely neurological origins, encompassing traumatic brain injuries, spinal cord injuries, and even psychogenic factors. Each presents unique challenges that necessitate comprehensive assessment and targeted interventions. In addressing dysphagia, collaboration among healthcare providers, caregivers, and patients themselves is essential. Education and awareness initiatives can empower individuals to recognize symptoms, seek appropriate care, and implement strategies to manage dysphagia effectively. Overall, by deepening our understanding of the complexities surrounding dysphagia in neurological disorders and beyond, we can strive towards improved outcomes, enhanced quality of life, and better support for those affected by this challenging condition.

Conflict-of-interest and financial disclosure

The authors declare that they have no conflict of interest to disclose. The authors also declare that they did not receive any financial support for the study.

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