

Dysphagia in rheumatological disorders

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Abstract

Dysphagia can be seen in rheumatological diseases. Due to life-threatening complications, early diagnosis and treatment of dysphagia is important. However, sufficient data is not available for the diagnosis and treatment of dysphagia especially in the group of rheumatological diseases. In this paper, the presentation of dysphagia in rheumatological diseases will be reviewed.

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Key words: Dysphagia; Swallowing; Inflammatory rheumatological disease; Non-inflammatory rheumatological disease; Rehabilitation

Core tip: Although dysphagia symptoms are common in rheumatological diseases, these conditions are often overlooked. Both the diagnosis and treatment of them is an issue to be considered carefully as they will lead to an apparent improvement in the patient's quality of life. Health professionals should be made aware of this issue.

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INTRODUCTION

Swallowing is a complex function enabling forwarding of the material and saliva in the mouth into the stomach. Dysphagia is associated with impaired swallowing function and can be defined as the difficulty or failure in conveying foods or liquids from the mouth to the stomach. Swallowing problems may develop in connection with a large variety of diseases in all age groups from a newborn to an elderly^[1]. Swallowing problems are encountered in inflammatory or non-inflammatory rheumatological diseases due to the disease itself or the treatment administered^[1,2]. Aspiration, pneumonia, malnutrition, increased mortality, prolonged hospitalization, advanced disability, declined quality of life and isolation from the society may accompany the difficulty in swallowing. Early diagnosis and treatment of dysphagia is important due to such life-threatening complications. In this paper, the presentation of dysphagia in rheumatological diseases will be reviewed.

INFLAMMATORY RHEUMATICAL DISEASES

Scleroderma

It has been reported that 87% of patients with progressive systemic sclerosis complained from dysphagia. Even during the first examination, 60% of patients have complained about dysphagia^[3-6]. Early diagnosis and treatment of dysphagia in this patient group is important in terms of patient care.

Perioral skin and temporomandibular joint limitation may lead to difficulty in opening the mouth and glosal papillae, and mucous membrane atrophy to impaired taste and eating problems, which in turn results in serious weight loss. Esophagus dysfunction in this disease, on the other hand, has a complex, multifactorial characteristic which is variable according to the stage of the disease^[3-6].

Impairment of the motor function of the esophagus has been linked to more severe reflux and possible recurrent episodes of micro aspiration and lung damage. The

degree of mucosal damage diagnosed by endoscopy was also worse in patients with aperistalsis. In the scleroderma patients, abnormal reflux was found in 91%. Presence of chronic reflux may result in Barrett's metaplasia, chronic pharyngitis-laryngitis and aspiration pneumonia in these patients. It may also lead to muscle atrophy and fibrosis in 2/3 portion of the lower esophagus as well as to motility damage^[7]. The findings obtained include decreased resting lower esophageal sphincter (LES) pressure, impaired LES relaxation, and increased non-peristaltic contraction alongside decreased esophageal peristaltic contraction amplitude. Cholinergic function abnormalities may also be seen at various severities depending on the stage of the disease. There are data indicating a relationship between the dysmotility in esophagus and the Raynaud phenomenon. Esophagus involvement shows itself also in the CREST syndrome with heartburn, regurgitation and chronic intermittent dysphagia to both liquid and solid foods. It was reported that eosinophilic fasciitis may cause esophageal dysfunction in cases involving systemic sclerosis^[6].

Sjögren's syndrome

Swallowing difficulties are frequently (32%-85%) encountered complaints in the primary Sjögren syndrome (SS). Exocrine gland infiltration causes dysphagia symptoms in these patients. The manifestations from the gastrointestinal system in patients with SS include mucosal dryness, accelerated dental decay, and enlargement of the major salivary glands, as well as dysphagia, nausea, epigastric pain, and dyspepsia. The oral-pharyngeal phase and esophageal phase of swallowing is being affected. It is difficult to establish a relationship between the severity of symptoms and the severity of disease involvement^[2,4,6].

The data obtained in the long term were meant to reveal the esophageal disorders in SS^[8]. Patients consult us with esophagus atrophy and motor coordination problem as a result of an inflammation of the esophageal exocrine gland. Such patients apparently have abnormal esophagus motility. They seem to have a LES pressure higher than that in healthy people, an upper esophagus sphincter (UES) impairment and a decreased ability of contraction in the upper 1/3 of esophagus. Peristaltic contraction velocity decreases and its duration increases in the lower and middle parts of esophagus^[8-10].

It has been reported that multi-factorial mechanisms lead to dysphagia in SS^[6,11]. The results were tried to be explained by lack of saliva, esophageal dysmotility (36%-90%), esophageal web, achalasia, exocrine gland involvement, low grade myositis, and parasympathetic function damage. The relationship between the severities of xerostomia and dysphagia is controversial^[2,4,6,8].

Idiopathic inflammatory myopathies

Dermatomyositis (DM) and polymyositis (PM) are classified as idiopathic inflammatory myopathies. Dysphagia can be a serious problem in these patients. Dysphagia in this patient group is associated with the severity of

the disease and is also an indicator of a poor prognosis. Involvement of the striated muscles of the oropharynx and upper esophagus occurs in 10%-15% of patients, and may lead to dysphagia (10%-15%), regurgitation, and aspiration pneumonia. In addition, there may be ventilatory dysfunction due to involvement of the diaphragm and intercostal muscles. Dysphonia with a nasal speech quality may be noted^[12]. In PM/DM patients, the triggering of the swallowing reflex for the voluntarily initiated swallow was normal while the pharyngeal phase of swallowing was significantly prolonged. The cricopharyngeal sphincter muscle electromyography (EMG) demonstrated severe abnormalities in halves of the patients investigated. These findings demonstrated the weakness of the striated oropharyngeal muscles. Cricopharyngeal (CP) sphincter muscle was affected less frequently and showed either hyperreflexic or hyporeflexic states during swallowing. It is concluded that the pharyngeal stage of oropharyngeal swallowing is mainly involved in patients with PM/DM^[13]. Decreased pharyngeal muscle strength, palate elevation disorder, tongue weakness, cricopharyngeal muscle dysfunction, and sphincter closing problem are among the findings seen in this group of patients. The oropharyngeal swallowing problem was in 20% of the patients, esophagus involvement was in 1/3 of proximal part and there was esophagus dysmotility. There was a slowdown in the speed of gastric motility. Malignity of gastrointestinal system and nasopharynx can also be seen^[6].

Pharyngeal involvement in juvenile DM was also found to be associated with poor prognosis. Weakness in oropharyngeal, laryngeal and esophageal muscles cause swallowing dysfunction^[14]. In the absence of a more accurate assessment method to determine which children with active JDM are most at risk of swallow dysfunction and aspiration, all children with active dermatomyositis should be referred for speech and language assessment and videofluoroscopic swallow study (VFSS)^[14].

Dysphagia is more common in inclusion body myositis (IBM) than in the other inflammatory myopathies and is reported to be occurring in 38%-84% of patients. Moreover, its outcome is worse in patients with IBM than in those with either poly- or dermatomyositis and its contribution to aspiration pneumonia associated respiratory failure may be the most common cause of death in people with IBM^[15]. Dysphagia was frequently the presenting clinical symptom in patients who had IBM-associated dysphagia, observed more often in women and was usually refractory to medical and nonsurgical treatment^[15].

Common dysphagia symptoms were sensation of food sticking in the throat and coughing during meals. The patients noted difficulty with dry foods, solids, and thin liquids most frequently. Clinical oral examination findings typically showed normal lingual range of motion, strength, and coordination.

The most common videofluoroscopic abnormalities are residual pharyngeal pooling, tongue base weakness, airway penetration, reduced pharyngeal constrictor con-

traction, CP muscle dysfunction described as a prominent CP muscle with poor relaxation and narrowing in the upper esophagus, and impaired laryngeal elevation. Aspiration was revealed in eight patients (35%). Prominent, tight CP muscle was noted in all nine patients who underwent a barium swallow. Common pharyngoesophageal manometry findings included low amplitude pharyngeal constrictor contraction (75%), normal resting tone and relaxation of the UES (82%), and diminished inferior esophageal sphincter pressure (42%)^[15].

Systemic lupus erythematosus

In the systemic lupus erythematosus (SLE) patient group, we often encounter gastrointestinal complaints associated with the disease itself or the treatments given. In this patient group where systemic symptoms are in the foreground, mucosal ulcer (50%), decreased salivation, esophagus ulcer-perforation, decreased esophageal motility (72%), isolated abnormal peristalsis in esophagus, stricture, and reflux may be seen. The complaints are mild and vary in line with the disease activity. These findings are thought to emerge as a result of muscle inflammation and/or vasculitis-related damage. If vasculitis is present, more severe complaints and symptoms may be seen. Involvement can occur in any place in the gastrointestinal system. The region that is involved most frequently is the oral cavity. Erythematous lesions or discoid ulcers occurring in the hard palate, buccal mucosa or vermilion border regions cause dysphagia and odynophagia. The upper 1/3 region of esophagus is affected more. Abnormal peristalsis in proximal or distal esophagus is one of the leading pathologies. LES involvement is not seen much in this patient group^[2,4-6].

Rheumatoid arthritis

One third of the patients in this rheumatologic disease group mention about the dysphagia symptoms. Temporomandibular joint involvement and Sicca syndrome cause impaired chewing function and difficulty in swallowing. Esophageal involvement is associated with pharmaceutical therapies that last much longer than the disease itself. Atlantoaxial subluxation and vasculitis (1%) are rare but may lead to esophageal problems (dysmotility, fibrosis, stricture and ulceration)^[2,4-6]. Rheumatoid nodule or laryngeal synovitis may also create a dysphagia symptom^[16]. Accumulations of esophagus amyloid and pseudo-achalasia may appear as complications in RA patients.

Eosinophilic infiltration of gastrointestinal tract (esophageal and pharyngeal) in RA appears as a rare and interesting involvement. Eosinophilia in RA patients can be associated with allergies, drugs (gold, penicillamine, sulfasalazine, methotrexate), disease activity, rheumatoid vasculitis and parasitic infections^[17].

Children with Juvenile rheumatoid arthritis (JRA) rarely report temporomandibular joint pain, which may be due to pain avoidance mechanisms resulting in compromised masticatory function. Micrognathia, loss of a mandibular condyle and jaw retrusion seem to be associ-

ated with dysphagia symptoms in the child patient group. Other findings also encountered include decrease/impairment in esophagus distal peristalsis (30%-58%), decrease in LES tonus, esophageal ulcers (Feltz's send). A complaint of reflux may arise in connection with these findings^[18].

Other

Dysphagia symptoms that are pill-induced or associated with the presence of esophagitis are rarely seen in the *Seronegative arthropathy* group. Annulus fibrosis and longitudinal ligament bone formation may lead to dysphagia symptoms in patients with ankylosing spondylitis.

Sarcoidosis can affect the oesophagus in different ways. Stenosis of the distal oesophagus due to direct granulomatous involvement, or extrinsic compression by enlarged hilar and mediastinal lymph nodes, may both cause dysphagia. Sarcoid infiltration of the distal oesophagus can give rise to achalasia, and granulomatous myositis of the cricopharyngeal muscle causing dysphagia has also been reported. Barrett's oesophagus can also occur in sarcoidosis.

Since *vasculitis* can produce any type of a vessel involvement in any organ, gastrointestinal-esophageal involvement is also a diagnosis that should be considered in vasculitis such as Behçet's disease. Oral and esophageal ulcers may cause dysphagia symptoms. Esophageal manometry was found abnormal in a third of the cases. Mucosal lesions, odynophagia associated with esophagus involvement, bleeding, and ulcerations may occur in other types of vasculitis such as Wegener granulomatosis^[2,4-6].

In inflammatory rheumatologic diseases, abnormal motility of esophagus may be associated with the worsening of pulmonary functions and the severity of reflux^[7].

NON-INFLAMMATORY RHEUMATIC DISEASES

Due to the close relationship between esophagus and cervical spine, the presence of broad anterior osteophyte (spondylosis or DISH) causes dysphagia symptoms. Dysphagia is probably the most common cervical manifestation associated with DISH and was reported by various specialties. DISH was the cause of dysphagia in 17%-28% of patients over 60 years of age referred for dysphagia evaluation. Obstruction frequently occurs at C5-6 and more rarely at C4-5, C2-3 and C3-4. This location is particularly vulnerable to local pressure because the osteophytes compress the relatively immobile portion of the esophagus at the level of the cricoid cartilage. Larynx and hyoid elevation becomes a problem for epiglottic movement and bolus movement. Conditions other than cervical osteophytes may induce dysphagia, such as strictures, oesophagitis, cardiospasm, diverticula, motility disorders, benign or malignant tumors, and other C-spine disorders^[19].

Skeletal deformity/basilar invagination may lead to oropharyngeal dysphagia symptoms in Paget's disease al-

though very rarely.

COMPLICATIONS OF MEDICAL TREATMENT IN RHEUMATIC DISEASES

Not only rheumatic diseases themselves but the drugs used in treating these diseases may also cause impairment in swallowing functions. Gold, penicillamine, sulfasalazine, methotrexate and other cytotoxic drugs can cause stomatitis and oral ulcers. Similarly, non-steroidal anti-inflammatory drugs, corticosteroids and bisphosphonates can cause mucosal erosion, oesophagitis and oesophageal ulceration^[2,20]. Psychological and behavioural adverse events of low- to medium-dose glucocorticoids during ≥ 1 mo for inflammatory diseases were most frequently reported, followed by gastrointestinal events such as dysphagia. Whereas “pill oesophagitis” may cause identical symptoms to gastroesophageal reflux disease with retrosternal chest pain and possibly dysphagia, odynophagia is usually the dominant complaint^[3]. Steroid therapy and other immunosuppressive agents may also impair deglutition by predisposing to candidiasis of the upper intestinal tract^[2,20].

ASSESSMENT

A good assessment is a must for the success of the treatment in patients with dysphagia. Speech language therapists assume a big task in the assessment and treatment of swallowing disorders. A physiatrist and/or a radiology specialist may be helpful in performing the assessment. However, assessment of a patient should be conducted in a multidisciplinary way^[1].

At the stage of assessing patients, whether or not there is a swallowing disorder, possible local and anatomic causes of dysphagia (oropharyngeal, esophageal), airway protection capability (*e.g.*, aspiration risk), oral feeding functionality, alternative methods for regulating eating, an additional specific diagnostic test and need for consultation should all be considered. Anamnesis, queries about the drugs (sedative, antispastic, anticholinergic, *etc.*) and disease, background, family history, physical examination and imaging methods are used in an effort to arrive at a diagnosis. Failure or delay when initiating oropharyngeal swallowing, postnasal regurgitation or nasal regurgitation, cough indicating aspiration, apnea, presence of residue in the mouth and swallowing that necessitates repetition to clear the mass from hypopharynx may serve as guidelines for an oropharyngeal swallowing problem. A sensation of sticking behind the sternum during swallowing, painful swallowing, heartburn, and lack of pharyngeal symptoms also require investigation in terms of esophageal dysphagia. Weight loss and prolonged eating time are also important symptoms. A history of pulmonary infection undergone by the patient may be enlightening.

Some of the patients who were diagnosed with gastroesophageal reflux consult polyclinics with a complaint of dysphagia. Assessments of these patients do not re-

veal any significant pathology other than symptoms of reflux. They also need to be assessed in terms of esophageal motility problems.

During the physical examination, oropharyngeal condition, pulmonary system, musculoskeletal system, mental condition, speech and voice quality, cranial and reflex examination (gag reflex, cough reflex, swallowing reflex, touch and taste stimulation and pathologic reflex) and motor control are assessed. Biochemical parameters concerning nutrition should also be reviewed.

Diagnostic tests are started with a clinical or bedside swallowing assessment. The bedside assessment starts with a water and ice assessment. Various volumes of water (3, 5 and 10 mL of water) and food with various viscosities (nectar-like, honey-like, cracker, *etc.*) are tested. After swallowing, a change in the patient's color, wet voice, presence of food residues, and coughing can be enlightening for us in terms of aspiration. A bedside assessment gives us limited information on the function and mechanism of swallowing; its reliability is moderate. The VFSS is accepted as the gold standard. Clinical indicators do not reveal aspiration in inflammatory rheumatologic diseases particularly in children. Thus, although it is an unpleasant test in terms of radiation, money and time, VFSS still remains to be the most viable method today.

The fiberoptic endoscopic evaluation of swallowing (FEES) also gives fairly reliable information other than its assessment insufficiency at the oral phase. If a problem is being suspected particularly in oropharyngeal swallowing, these tests will help us for making a diagnosis^[21].

A barium esophagram is important for the diagnosis of osteophyte, dilatation, stricture, Zenker's diverticulum, achalasia and presence of a lump. Esophageal dysfunction is frequently seen in autoimmune and inflammatory rheumatologic diseases. Esophageal manometric examination is a specific and sensitive method to assess the dysmotility of esophagus. The sensitivity of VFSS and FEES examinations is lower in this patient group. Esophagitis, Barrett's esophagus and presence of stricture can be revealed with a gastric endoscopy and gastroesophageal reflux disease with a 24-h ph-meter examination. Esophagus scintigraphy may be useful in these patients for assessment of esophagus dysmotility, esophageal emptying and reflux.

GENERAL PRINCIPLES IN TREATMENT

The treatment objective in these patients is to reduce the risk of aspiration, to ensure daily calorie intake, to increase oral feeding varieties for both nutrition and patient satisfaction and to raise quality of life. It is decided after the examinations whether the patient's way of food intake will be oral, through a nasogastric tube (NGT) or through percutaneous endoscopic gastrostomy (PEG)/jejunostomy (PEJ). In all viscosities, if there is more than 10% aspiration, the patient should not be fed orally. If the patient cannot eat safely and effectively, methods of

feeding through a tube will be tried. This is not a treatment method, but a strategy devised to protect the patient from negative clinical conditions (dehydration, malnutrition, aspiration). It should be made certain whether or not the patient is taking his/her daily calorie requirement. It may be difficult to ensure that he/she is taking sufficient calories depending on the duration of eating food (cognitive defect). As a general approach, if a short-term non-oral feeding is targeted, NGT will be chosen, if non-oral feeding for a longer period than 2-3 wk is considered, PEG or PEJ will usually be used. However, the healthcare staff that monitors the patient and the patient should make this decision together.

Treatment of dysphagia in inflammatory rheumatologic diseases is a difficult task. Although there is recovery in parallel with disease activity in some groups, it is hard to achieve this success at all times. Over three fourths of the patients received aggressive immunosuppression, but the benefit seems to be ineffective consistent with literature. The effect of the drugs used for treating the disease on dysphagia varies. While response to the treatment is limited in scleroderma, the decline in the severity of the disease in SLE patients results in an improvement also in dysphagia symptoms. The success of IVIG and steroid therapies is variable. The effect of TNF-alpha inhibitor drugs is unknown. If it is thought that the dysphagia is associated with the medication used by the patient, pharmaceutical rearrangements should be made. The treatment of gastroesophageal reflux may be useful in terms of securing oral hygiene, candida treatment, LES pressure-related approaches, dysphagia symptoms, and complications. Fortunately, although dysphagia symptoms are frequent, severe dysphagia and its complications are seen rarely. A multidisciplinary approach increases success in treatment.

In addition to the management of underlying disease, the treatment includes special dietary regimen, rehabilitation and even interventional surgical procedures, if necessary. Treatment options included swallowing strategies (exercise, special techniques, diet modifications), CP myotomy (Zenker's diverticulum, achalasia), pharyngoesophageal dilation, electrical stimulation to swallowing muscles, and botulinum toxin injection for the treatment of UES (in the presence of sphincter relaxation problem). It is decided after the assessment as to what treatment(s) is appropriate. The treatments may be grouped basically as compensatory and therapeutic approaches. Compensatory approaches are applied to control the way through which food will travel, to remove the symptoms and to prevent aspiration. Compensatory approaches include postural technique (eating positions), oral-sensory motor improvement techniques, and adjustment of the volume and viscosity of the bolus. Swallowing compensation and feeding techniques were recommended to over half of the patients, but their effectiveness remains uncertain in these diseases. The limited data available on this issue make it difficult for us to decide as to what treatment is more effective. Therapy procedures include special swallowing maneuvers (supraglottic, super-supraglottic,

effortful swallowing, Mendelshon maneuver), bolus control exercises, jaw-tongue-lip-pharyngeal joint range of motion exercises, jaw, tongue and lip strengthening exercises, Shaker exercises, Masako maneuver, and breathing exercises. Adaptive devices and instruments are also used for eating safety and control. When assessing patients, an appropriate treatment plan is prepared by trying different maneuvers, viscosities and volumes at the same time.

In conclusion, swallowing problems in rheumatologic diseases appear to be a not very well known condition. Studies with long follow-up periods to be included in the literature in the future will provide us with more information. With the present data, it is quite difficult to comment on either the diagnosis or the treatment. It is important that healthcare staff is made aware of the fact that patients with dysphagia should be directed to rehabilitation units. In this way, a noticeable improvement can be achieved in patients' quality of life through small touches.

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