Making Dysphagia Easier to Swallow: A Review for the Practicing Clinician

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CME Activity

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Learning Objectives: On completion of this article, you should be able to (1) recall how to take a clinical history for a patient with dysphagia; (2) interpret physical examination findings as they are related to differential diagnosis; and (3) recommend appropriate testing for the clinical situation.

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Abstract

Evaluating a patient with dysphagia can be a complex and daunting task. In this article, we present a practical approach to the evaluation, physical examination, and subsequent work-up of dysphagia that is applicable to practicing physicians.

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Dysphagia is a relatively common symptom, occurring in approximately 3% of the general population.[1] It is estimated that 1 in every 17 people will struggle with difficulty swallowing at some point in their lives.[2] Dysphagia increases in frequency with age, affecting approximately 10% of adults older than 65 years and approximately 50% of the institutionalized elderly population.[3,4] However, aging itself is not a typical cause of clinically significant dysphagia. Dysphagia can lead to malnutrition, aspiration, and pneumonia, and as such, an underlying cause for the symptom should be explored (Table).[5] In this article, we summarize the key features of the history and physical examination that will guide investigation and treatment of dysphagia.

PHYSIOLOGY OF DEGLUTITION

A comprehensive understanding of normal swallowing physiology is imperative for

TABLE. Causes of Dysphagia

- Oropharyngeal dysphagia
  - Neurologic
    - Cerebrovascular accidents
    - Parkinson disease
    - Amyotrophic lateral sclerosis
    - Multiple sclerosis
    - Huntington disease
    - Guillain-Barré syndrome
    - Tabes dorsalis
    - Tetanus
    - Polio
    - Tardive dyskinesia
    - Alzheimer and other types of dementia
  - Muscular
    - Myasthenia gravis
    - Muscular dystrophy
    - Hyperthyroidism
    - Hypothyroidism
    - Dermatomyositis
    - Polymyositis
  - Structural
    - Esophageal web
    - Cervical osteophytes
    - Goiter
    - Lymphadenopathy
    - Zenker diverticulum
    - Oropharyngeal tumor
- Esophageal dysphagia
  - Motility
    - Achalasia
    - Spastic motility disorders (diffuse esophageal spasm, nutcracker esophagus, jackhammer esophagus)
    - Hypocontractile motility disorders (ineffective esophageal motility disorder)
    - Scleroderma
    - Sjogren syndrome
    - Connective tissue overlap syndrome
    - Amyloidosis
  - Inflammatory
    - Erosive esophagitis
    - Eosinophilic esophagitis
  - Infectious
    - Chagas disease
    - AIDS
    - Infectious esophagitis (cytomegalovirus, herpes simplex virus, candida)
  - Structural or mechanical
    - Malignancy (esophageal, gastric, or mediastinal)
    - Foreign bodies
    - Peptic stricture
    - Esophageal ring (Schatzki ring)
    - Dysphagia lusoria (aberrant right subclavian artery)
    - Other vascular ring anomalies (enlarged left atrium or aorta, aberrant vessels)

Continued on next column

TABLE. Continued

- Esophageal dysphagia, continued
  - Other
    - Functional dysphagia
    - Pill esophagitis (nonsteroidal anti-inflammatory drugs, bisphosphonates, doxycycline, tetracycline, potassium chloride, quinidine, ferrous sulfate, ascorbic acid)
    - Decreased lower esophageal sphincter tone (medications such as nitrates, anticholinergics, benzodiazepines, morphine, calcium-channel blockers, tricyclic antidepressants)

understanding abnormal swallowing. *Deglutition* is defined as the act of swallowing in which liquids, solids, or both are transported from the mouth to the stomach by way of the pharynx and esophagus.[6] Normal swallowing occurs up to 600 times a day and each swallow takes approximately 15 seconds to complete. More than 30 muscles are involved in the process. Deglutition is divided into 4 phases: (1) preparatory, (2) oral, (3) pharyngeal, and (4) esophageal.[7]

The process begins as food enters the mouth. Muscles of mastication and the tongue contract to mix saliva with food to generate a bolus of appropriate shape, size, and consistency. The anterior aspect of the tongue elevates to make contact with the hard palate and moves posteriorly to force the bolus into the upper pharynx. This action elevates the soft palate and seals off the nasopharynx to prevent nasal regurgitation and direct the bolus posteriorly. The suprahyoid muscles contract to move the larynx anteriorly and superiorly, whereas the epiglottis moves inferiorly to cover the trachea.[8] Pharyngeal muscles contract to push the bolus past the upper esophageal sphincter or cricopharyngeus muscle, which relaxes shortly after laryngeal elevation, allowing the bolus to move into the proximal esophagus.[8,9] Peristalsis propels the food bolus through the esophagus and across the lower esophageal sphincter.

APPROACH TO CLINICAL HISTORY

Dysphagia, or interruption to the normal process described above, is always pathologic and warrants further evaluation.

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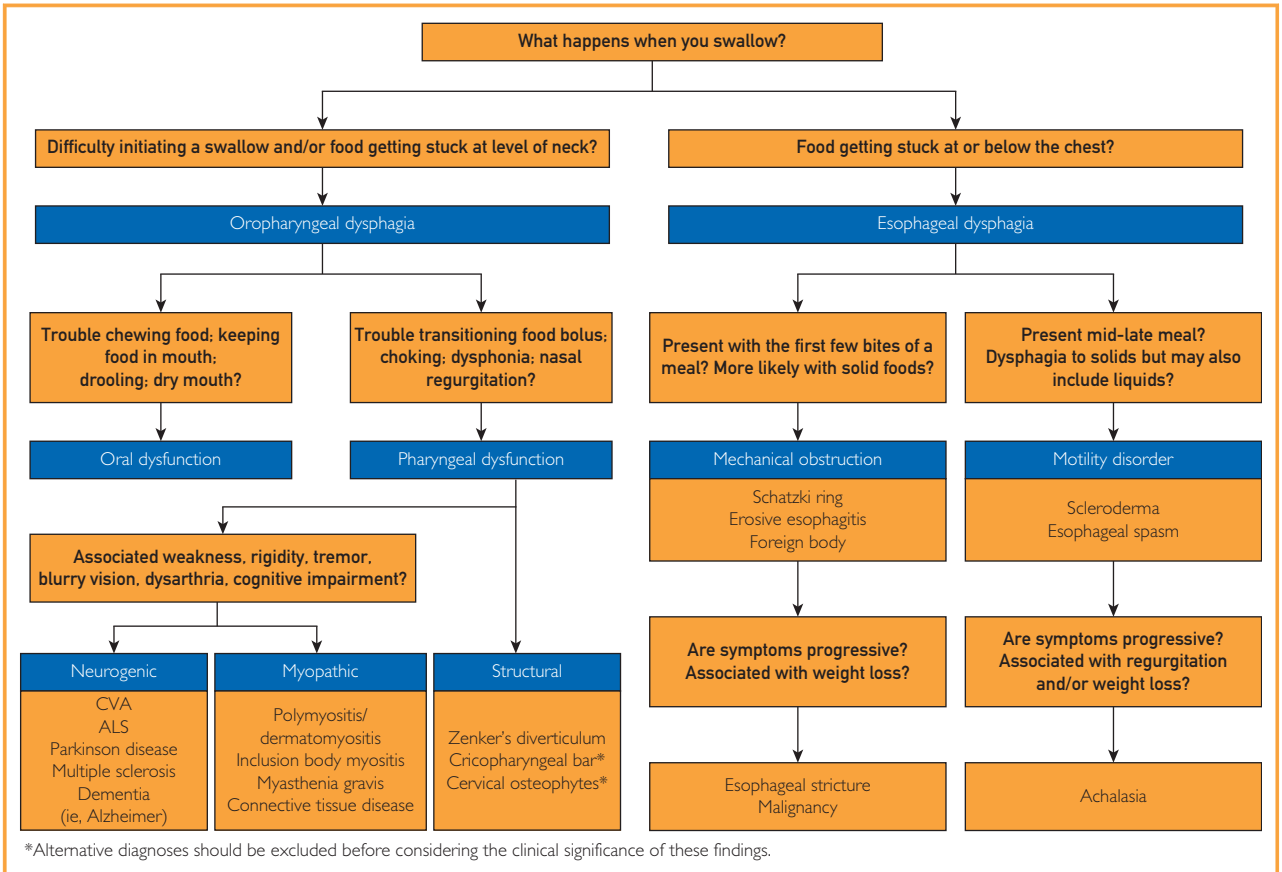

FIGURE 1. Diagnostic cascade for dysphagia. ALS = amyotrophic lateral sclerosis; CVA = cerebrovascular accident (stroke).

This evaluation begins with a careful history to guide a cost-effective work-up. The approach to the clinical history is broken down into a series of questions that help narrow the differential diagnosis.[10] Before exploring these questions, it is important to clarify with the patient what is meant by difficulty swallowing. Dysphagia needs to be distinguished from *globus sensation*, a feeling of a "lump in the throat" that can be present both during eating and fasting, and *odynophagia*, pain with swallowing. After the true nature of the issue is clarified as dysphagia, we recommend proceeding with the following questions (Figure 1).

What Happens When You Try to Swallow?

This question helps distinguish between oropharyngeal and esophageal dysphagia. A good clinical history can decipher the type of dysphagia in approximately 80% of cases.[11] If the patient describes difficulty initiating swallowing, nasopharyngeal regurgitation, or choking, this is suggestive of oropharyngeal dysphagia. If the patient describes food getting stuck after the food bolus has left the oropharynx, this suggests esophageal dysphagia. However, patient perceived localization has low diagnostic specificity if food is described as getting stuck at the level of the neck, as this can also result from lower esophageal obstruction.[7] Deciphering between oropharyngeal and esophageal dysphagia is a key step.

If the cause is determined to be most likely oropharyngeal, a good next question is as follows.

Do You Have Any Trouble Chewing Your Food?

This question addresses whether the oral preparatory phase is normal. Asking the patient if it is a challenge to chew food helps sort out true dysphagia from oral cavity pathology,

which is important as treatment is different. This is especially true in the elderly patient who has absent or poor dentition, poorly fitting dentures, xerostomia, weakened muscles of mastication, temporomandibular joint arthritis, altered oropharyngeal sensation, and altered level of attention during feeding that could be mistaken for dysphagia. Oral lesions, trauma, or mucositis may also inhibit normal mastication.

If the first question points the clinician away from oropharyngeal and toward esophageal dysphagia, a good next question is as follows.

Do You Have Difficulty Swallowing Solids, Liquids, or Both?

This question helps differentiate between a motor issue and a mechanical one. Dysphagia that is limited to solids suggests a mechanical obstruction and stenosis of the esophageal lumen to a diameter less than 15 mm.[12] Various mucosal diseases can narrow the lumen via mechanisms such as fibrosis, inflammation, or neoplasia.[12] Foreign bodies can also be responsible for dysphagia limited to solids. Mediastinal disease can create narrowing by extrinsic compression or direct invasion of the esophageal lumen.[12] Dysphagia that includes both liquids and solids may represent a motility problem. Examples of motility disorders include achalasia and scleroderma.

After determining whether the pathology is mechanical or motor, establishing a timeline is next.

What Is the Symptom Duration and Frequency?

This question allows the clinician to stratify the time course as acute, intermittent, or progressive. Acute suggests foreign body, whereas intermittent can be indicative of esophageal spasm if the problem is motor in nature or esophageal luminal narrowing if the problem is mechanical. If the symptoms are progressive, concern is heightened for motor issues such as scleroderma or achalasia or for mechanical issues such as strictures or malignant neoplasms.[13] Timing of dysphagia during the course of eating can also provide further information. Dysphagia that occurs within the first few bites of a meal can represent a ring or stricture. Asking the patient how they have managed the symptom can elucidate timing of the pathology. Some patients present only when adaptive behaviors fail them. Patients who have become a slow eater, chew more carefully, or manipulate their head or neck to facilitate swallowing are those who have suffered from dysphagia for a longer period of time.

Inquiring about accompanying issues can bring clarity to the diagnosis. Ask the patient the following question.

What Are the Associated Symptoms?

Associated symptoms that frequently accompany oropharyngeal dysphagia include coughing, choking, drooling, nasal regurgitation, and repetitive swallowing.[12] They may present with episodes of aspiration pneumonia.[13] Halitosis and regurgitation can be a sign of a Zenker diverticulum or achalasia. Ptosis is seen with myasthenia gravis. Hemiparesis can occur with stroke. Pill-rolling tremor, rigidity, and bradykinesia are seen in Parkinson disease. Weight loss is concerning for malignancy or an advanced disease process. Chronic heartburn can be a manifestation of scleroderma, gastroesophageal reflux disease, or peptic stricture.[14]

Lastly, to be comprehensive, collect an allergy and medication history to ensure there are no confounders. A history of environmental or food allergies may be suggestive of eosinophilic esophagitis (EoE). Review the medication list for drugs that can impair swallowing by causing xerostomia, direct esophageal mucosal injury, or decreased lower esophageal sphincter tone to cause reflux.[15] Medications that can directly damage the esophagus include potassium chloride, tetracycline antibiotics, clindamycin, trimethoprim-sulfamethoxazole, quinidine, nonsteroidal anti-inflammatory drugs, ferrous sulfate, alendronate, and zidovudine.[13] Numerous psychiatric medications can cause xerostomia. Opiate medications can alter esophageal motor function in various ways that can lead to dysphagia.[16]

PHYSICAL EXAMINATION

The physical examination may provide important clues in elucidating the underlying etiology. Examine the oral cavity. Incorrectly sized dentures or dry mucous membranes

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may affect a patient's ability to chew. Assess for facial muscle wasting that could weaken the ability to swallow. Check for cervical lymphadenopathy and other anatomical changes that could alter the transit of food. Assessment of cranial nerve function, speech, weakness, or gait disturbance can provide clues to the underlying neurological disease that affects swallowing mechanics. Tongue fasciculation can be an early sign of amyotrophic lateral sclerosis. Inspect the body for cachexia and evidence of weight loss suggestive of neoplasm.[14] Listen for delayed transit of the food bolus by auscultating the epigastrium for a gurgling sound associated with swallowing; in patients with achalasia, this sound can be delayed for several minutes (or not present at all).[14] The skin should be examined for lesions such as sclerodactyly, telangiectasias, or calcinosis that may be present in scleroderma, as well as the characteristic "shawl" sign rash that could be suggestive of dermatomyositis.

Perform a bedside swallow evaluation to visually assess the process of deglutition. Have the patient sip a small amount of water. Assess that they are able to keep the fluid in the mouth without any drooling or choking. Ask the patient to swallow, and assess that the patient is able to completely mobilize the bolus without any residual fluid in the oral cavity. Ensure that there is appropriate and symmetrical rise of the thyroid cartilage, and evaluate for choking or the need for double swallowing. Note if the patient endorses pain or a sensation of the bolus "getting stuck," and attempt to localize from where this sensation arises. Repeat the swallow examination, and check how long it takes for the patient to initiate swallowing after fluid enters the mouth. After the patient finishes swallowing, check for changes in phonation that may be associated with fluid retention by asking the patient to phonate the letter "e." This basic screening test is reported to be 95% sensitive for the presence of dysphagia.[17]

TESTING

In cases of oropharyngeal dysphagia, the criterion standard test is the videofluoroscopic swallow study (modified barium swallow),[13,18] which provides real-time information on swallowing mechanics (Figure 2). It

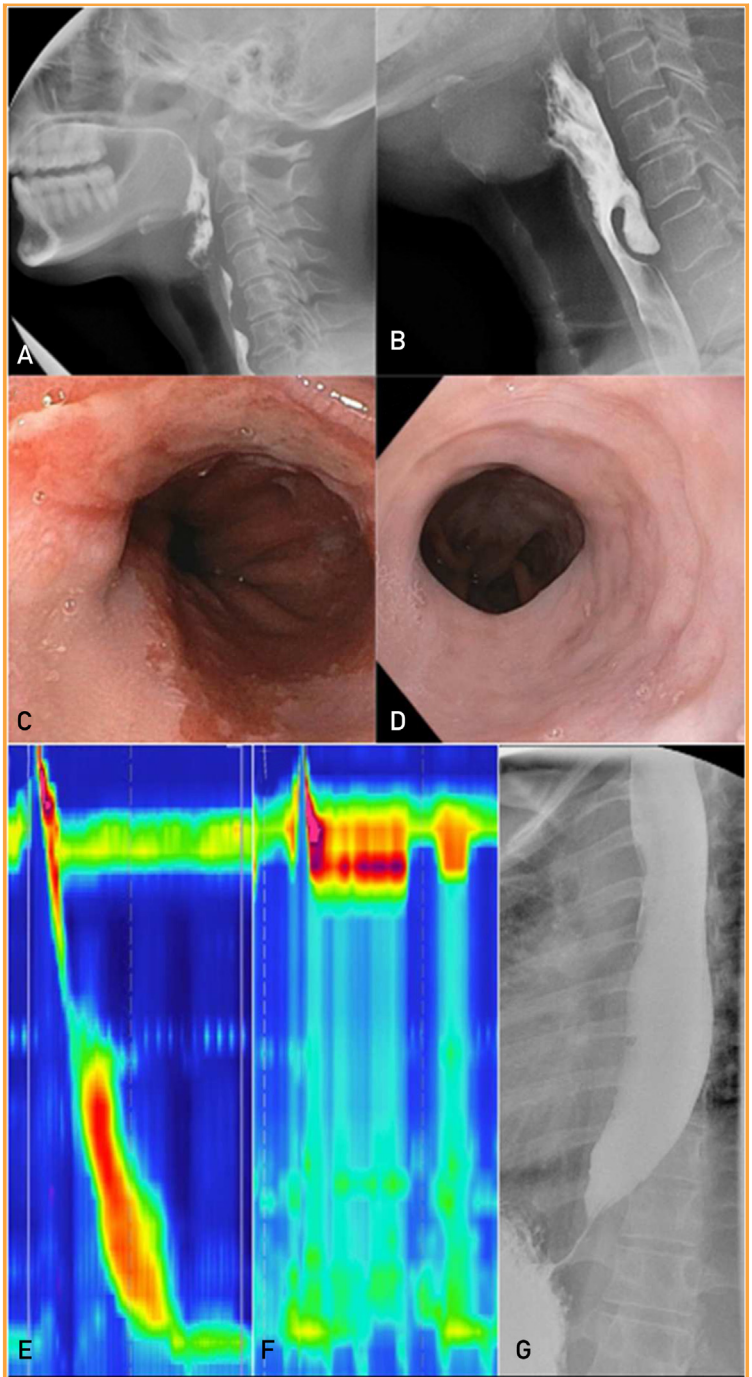

FIGURE 2. Sample of diagnostic studies for causes of dysphagia. A, Videofluoroscopic swallow study showing hypopharyngeal residue in patient with dermatomyositis. B, Zenker diverticulum. C, Esophagogastroduodenoscopy showing severe erosive esophagitis (Los Angeles classification D). D, Esophagogastroduodenoscopy showing partially obstructive Schatzki ring. E and F, High-resolution esophageal manometry showing normal swallowing (panel E) compared with lack of peristalsis and hypertensive lower esophageal sphincter of achalasia (panel F). G, Barium esophagram of a patient with achalasia showing a dilated esophagus with "bird beak."

is also useful to determine risk of aspiration, which allows for prescription of an appropriate texture-based diet. After the diagnosis of oropharyngeal dysphagia is made, further testing may be indicated to determine whether it is primarily a neurological or muscular problem. A referral to neurology and other subspecialists should be considered.

In cases of esophageal dysphagia, the most helpful test is esophagogastroduodenoscopy (EGD). The procedure both affords the potential for visualization of a specific diagnosis such as a ring or stricture and provides therapeutic intervention such as esophageal dilation. If no structural abnormalities are present, midesophageal biopsies are oftentimes indicated to rule out EoE.[19] Reflux esophagitis in the absence of any other explanation for esophageal dysphagia warrants a trial of acid suppression medication.[20] Esophagogastroduodenoscopy is also useful in evaluating the infectious causes of dysphagia and ruling out malignancy.[21]

For patients with achalasia, the findings on endoscopy can vary on the basis of severity of disease. Mild forms can have a mostly normal-appearing esophagus, with a "popping" sensation felt by the endoscopist as the scope passes into the stomach because of increased lower esophageal sphincter tone. Reduced or absent peristalsis can also be observed. Patients with severe disease may exhibit a "sigmoid" esophagus with retained food and fluids.[22] Esophagogastroduodenoscopy in patients with scleroderma may reveal a patulous or completely open gastroesophageal junction, often with concurrent reflux esophagitis.[23]

If EGD reveals no obvious pathology, a barium contrast study may help clinicians identify structural abnormalities, including subtle rings, intraluminal narrowing, or extrinsic compression on the esophagus. This test may also reveal the classic "birds beak sign" of distal tapering of the esophagus in achalasia or the "corkscrew" esophagus seen in diffuse esophageal spasm.[24-26] A barium esophagram can also reveal a prominent cricopharyngeal bar. The clinical significance of this finding can be challenging to discern because it can be observed in more than 50% of patients with gastroesophageal reflux in the absence of dysphagia.[27]

If a motility disorder is suspected on the basis of endoscopy or barium studies, high-resolution esophageal manometry is indicated. A transnasal probe is introduced into the esophagus across the gastroesophageal junction, with pressure sensors located proximally every centimeter.[28] A typical protocol records the results of resting pressures as well as the pressures recorded on the basis of 10 swallows of water in the supine position. The Chicago Classification, a tool for categorizing esophageal motility disorders on the basis of the appearance of their high-resolution manometry plots, incorporates these findings and allows clinicians to identify specific pathologies.[29]

In some instances, a patient may be receiving dual antiplatelet therapy or chronic anticoagulation; these medications may alter the approach to testing if the cause of dysphagia is suspected to be esophageal. Pursuing a barium esophagram before EGD may help identify patients who need to hold these agents before endoscopic therapy if clinically safe to do so (Figure 3).

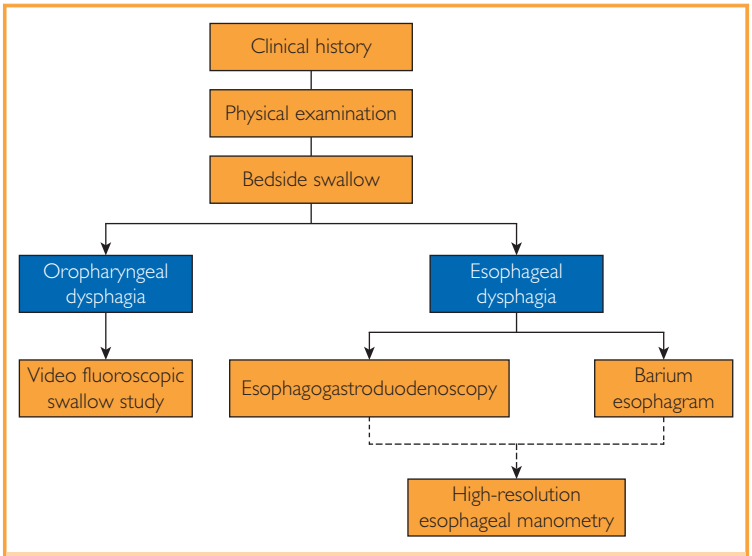

FIGURE 3. Work-up of dysphagia.

MANAGEMENT

Management is focused on the underlying disorder and often guided by subspecialty clinicians. A speech pathologist can guide a patient through compensatory interventions

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in the management of oropharyngeal dysphagia. These include postural adjustment to maximize effectiveness of swallowing and modifications to the food rate and amount to enhance the likelihood of successful swallowing. Eating in an upright position, using the chin tuck (widens valleculae, displaces tongue base, and epiglottis posteriorly), and using the head turn (directs bolus to the stronger side after the patient suffers a stroke causing unilateral pharyngeal weakness) are all strategies to assist in swallowing. Eating slowly, taking a smaller amount of food per bite, thickened liquids (reduced tendency to spill over tongue base), and swallowing mindfully are methods of combating oropharyngeal dysphagia. Rehabilitative exercises that focus on jaw, lips, and tongue range of motion and strengthening can also be helpful.

For esophageal dysphagia, treatment should target the underlying etiology. Upper endoscopy can often lead to a therapeutic intervention as previously mentioned; it could also lead to a specific diagnosis such as EoE. A patient who is found to have increased eosinophils (>15 per high-power field) on esophageal biopsies should begin twice daily acid suppression therapy with a repeat endoscopy in 6 to 8 weeks to rule out proton pump inhibitor–responsive eosinophilia. If symptoms persist and biopsies obtained from the distal and midesophagus continue to reveal eosinophilia, a diagnosis of EoE should be suspected. Treatment options include a 6-food elimination diet under the guidance of a dietician or swallowed corticosteroids (budesonide or fluticasone).[30] A gastroenterologist should be involved in the care of patients with EoE and refractory symptoms, a history of food impaction, or evidence of narrowing on EGD or barium swallow.

Patients with motility disorders such as achalasia require multidisciplinary care, including gastroenterology and thoracic surgery to determine the best treatment approach. In the absence of profound comorbidities, pneumatic dilation, surgical myotomy, and peroral esophageal myotomy should be considered. Although injection of botulinum toxin into the lower esophageal sphincter may provide temporary relief of symptoms, this option requires careful consideration, as it can potentially limit further interventions such as peroral esophageal myotomy. A comprehensive discussion of management of esophageal motility disorders is beyond the scope of this review and often requires the expertise of a gastroenterologist.

CONCLUSION

Dysphagia and its work-up can often appear complex and time intensive, as there are numerous associated conditions. As such, a methodical evaluation can ease diagnosis and treatment. One example of a clinical approach is described in this review. Following the steps in this evaluation should help narrow the differential diagnosis and define the type of dysphagia involved to further direct testing. Referral to gastroenterology should be considered when endoscopic treatment is required or in cases of complex esophageal dysphagia. Because of the multidimensional nature of dysphagia, referrals to other professionals in the fields of neurology, otolaryngology, speech language pathology, and oncology may also be merited.

Abbreviations and Acronyms: **EGD** = esophagogastroduodenoscopy; **EoE** = eosinophilic esophagitis

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