The Inpatient with Dysphagia

An Approach to Diagnosis and Treatment

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KEYWORDS

• Dysphagia • Disorders of swallowing • Inpatient care

KEY POINTS

- Dysphagia results from any dysfunction in the coordinated neuromuscular control of the swallow, a disruption of a patent conduit for the passage of the bolus, or loss of safe airway protection during swallowing.
- Inpatient medical needs are often complex and swift recognition, diagnosis, and treatment of dysphagia is vital to the promotion of healing.
- Collaborative evaluation and diagnosis are necessary for the development of patient-specific care, allowing the highest degree of preserved swallow function possible.

INTRODUCTION

An effective swallow is the result of a complex effort of numerous structures starting from the lips to the stomach, and any issue along the way can result in swallow dysfunction, or dysphagia. A successful swallow requires coordinated neuromuscular control at all phases of the process, a patent conduit for the passage of the bolus, and safe airway protection. Because the differential of potential pathologies of dysphagia is so large, when considering the complexity of inpatient medicine, we present an algorithm that can assist the clinician in considering the signs and symptoms and efficiently arriving at an accurate diagnosis to direct next steps in care.

It is important to acknowledge the differences in approaching the inpatient with dysphagia as compared to the outpatient. Inpatients can present with acute or rapidly evolving swallow dysfunction. They are often quite ill with multiple comorbidities and polypharmacy that can cloud the diagnostic picture. Lastly, as inpatients are often

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Abbreviations	
CP	cricopharyngeal
EGD	esophagogastroduodenoscopy
FEES	fiberoptic endoscopic evaluation of swallowing
IPC	inferior pharyngeal constrictor
LES	lower esophageal sphincter
MBBS	modified barium swallow study
RLN	recurrent laryngeal nerve
SLP	Speech Language Pathology
UES	upper esophageal sphincter
UVFP	unilateral vocal fold paralysis
ZD	Zenker's diverticulum

more fragile and potentially physically deconditioned, it is important to consider the patient's nutritional status and the obstacles to adequate nutrition that dysphagia can cause. Quick and accurate diagnosis of dysphagia for inpatients is vital to their overall health and ability to heal.

NORMAL SWALLOW FUNCTION AND THE DEFINITION OF DYSPHAGIA

First, let's explore the process of swallowing when everything is functioning correctly. Swallowing is a complex, coordinated process that ensures the safe and efficient transport of a bolus of food or liquid from the oral cavity to the stomach while protecting the airway. Understanding normal swallow physiology is foundational for diagnosing and managing dysphagia. Normal swallow function is traditionally divided into 4 phases: oral preparatory, oral transport, pharyngeal, and esophageal.[1,2] Each phase offers clinical clues to localize deficits and guide targeted interventions such as swallow therapy, dietary modifications, or surgical options.

Oral Preparatory

This voluntary phase begins with the intake of food or liquid into the oral cavity. The lips, tongue, and cheeks work together to contain the bolus, while mastication reduces solid food into a cohesive bolus. Salivary secretion, mediated by the salivary glands (parotid, submandibular, and sublingual), lubricates the bolus, aiding in its formation. The tongue plays a critical role in bolus manipulation, positioning it against the hard palate in preparation for the next phase. Deficits in this phase may be related to poor lip seal, weak mastication, poor lingual control, or dry mouth among others.[3]

Oral Transport

Also voluntary, the oral transport phase involves the propulsion of the bolus from the oral cavity toward the pharynx. The tongue initiates this by elevating and retracting, pressing the bolus posteriorly against the palate. This action triggers the transition to the involuntary phases. The duration of this phase is typically 1 to 2 seconds, depending on bolus viscosity and volume. Cranial nerve XII (the hypoglossal nerve) is central to this process, with sensory input from cranial nerve IX (glossopharyngeal) signaling bolus position. Impairments in tongue strength or sensory deficits may lead to difficulties in this phase.[3]

Pharyngeal

This involuntary phase is rapid (approximately 1 second) and critical for airway protection. The swallow reflex is triggered as the bolus passes the anterior tonsillar pillars,

activating the swallowing center in the medulla via cranial nerves IX (glossopharyngeal) and X (vagus). The soft palate elevates, which seals the nasopharynx and prevents nasal regurgitation. The larynx is pulled superiorly and anteriorly, which causes epiglottic inversion to close the laryngeal inlet. Additionally, the arytenoids and vocal folds adduct during swallow as an added layer of airway protection. The upper esophageal sphincter (UES), which is composed of the cricopharyngeus muscle and the inferior pharyngeal constrictor muscle, relaxes allowing bolus passage. Impairments in this phase, such as incomplete UES relaxation, weakness of the pharyngeal constrictor muscles, or inadequate laryngeal closure may increase aspiration risk while impairing swallow function.[4]

Esophageal

Fully involuntary, the esophageal phase of swallowing transports the bolus through the esophagus to the stomach via peristalsis. Primary peristalsis, initiated by the pharyngeal swallow, moves the bolus downward, while secondary peristalsis clears residual material if needed. The lower esophageal sphincter (LES) relaxes to permit entry into the stomach, then contracts to prevent reflux. This phase, lasting 8 to 20 seconds, is governed by the vagus nerve and the enteric nervous system. Disorders including achalasia or esophageal dysmotility can disrupt this phase, leading to symptoms such as regurgitation or chest pain.[5]

PATHOPHYSIOLOGY OF DYSPHAGIA

As the swallow mechanism is inherently complex, the differential for dysphagia is also vast and frequently multifactorial in nature. Although etiologies span multiple categories, we present a condensed discussion of several broad categories to help simplify the approach to dysphagia in the inpatient setting. The categories include neuromuscular, structural, lack of airway protection, inflammatory, and other.

Neuromuscular Causes of Dysphagia

In earlier sections, we discussed how effective swallow function is fully reliant on a coordinated, complex neuromuscular system of control and sensation. The brain and brainstem are inextricably linked to functional swallowing.[6] In fact, the central nervous system recruits 30 muscles via a combination of 8 cranial and peripheral nerves to coordinate all aspects of successful and safe swallowing.[7] Numerous neuromuscular diseases such as myasthenia gravis, amyotrophic lateral sclerosis, Parkinson's disease, multiple sclerosis, and stroke affect swallow function. By their nature, these diseases have the potential of impacting various and often multiple components of the deglutination process. It is important to emphasize here that although dysphagia is common in older adults, it should never be considered a normal condition of aging alone. Treatment of the underlying condition can be difficult in these cases and the symptoms can be progressive. Identifying early signs of dysphagia may allow early interventions and preserve serviceable swallowing for as long as possible, postponing the need for gastric tube placement or tracheostomy in patients at high risk for aspiration.

Primary esophageal neuromuscular dysfunction and dysmotility also occur, including diffuse esophageal spasm and achalasia.[8] Diffuse esophageal spasm is characterized by uncoordinated contractions along the esophagus that do not efficiently advance the food bolus to the LES. This disease is poorly understood, but may be attributed to reduced inhibitory neuron innervation.[9] Achalasia, on the other hand, causes abnormal hypercontractile tone of the lower esophagus due to inflammatory degradation of the neurons in the myenteric plexus.[10]

Structural Causes of Dysphagia

Zenkers's diverticulum

The cricopharyngeal (CP) muscle is a muscle that functions as part of the UES.[8] The CP muscle is tonically contracted and arises from the most distal aspect of the inferior pharyngeal constrictor (IPC) muscle.[11] Inadequate CP muscle compliance leads to a mechanical obstruction at this physiologic area of stenosis and may misdirect the bolus toward the laryngeal inlet or slow its progress.

CP dysfunction is commonly associated with another condition known as a Zenker's diverticulum (ZD). Anterolaterally, the transition point from oblique IPC muscle fibers and horizontal CP muscle fibers creates an area of weakness known as the Kilian-Jamieson Triangle, which is inherently prone to the formation of diverticula. Currently, the leading theory for ZD development attributes the mucosal layer outpouching to increased luminal pressure stemming from reduced or absent CP relaxation. Over time, the evolving CP dysfunction and increased intraluminal pressure preferentially direct the food bolus to the diverticulum once it becomes large enough. Patients tend to develop progressive aspiration, halitosis, gurgling, regurgitation, globus, weight loss, and dysphonia.

Cervical osteophytes

Cervical osteophytes are benign bony growths emerging from the cervical vertebrae typically seen in the elderly. While osteophytes are generally asymptomatic, when they arise in select levels of the spine, they may cause swallowing issues. Interestingly, it is estimated that 10% to 30% of the population have cervical osteophytes, yet only a subset of these (6%–30%) develop clinical dysphagia.[12] The spinal levels of greatest relevance are C3 to C7 as they are located adjacent to the oropharynx, hypopharynx, and UES. Depending on their size and location, these bony protrusions can affect swallowing in several ways, including altering epiglottic motion, hyolaryngeal elevation, and vocal cord mobility.[13]

Radiation changes

Primary radiation with or without chemotherapy for primary hypopharyngeal and laryngeal head and neck cancers is the treatment of choice for organ preservation. Inherent to radiation treatment are both acute and delayed onset toxicity sequelae that contribute to dysphagia. The peritreatment effects associated with radiation are inflammatory in nature given the underlying mechanism of action for this treatment modality. Hydroxyl free radical-mediated DNA damage leads to apoptosis and acute inflammation, which can cause dysphagia secondary to odynophagia (painful swallowing) or acute mucosal swelling as seen in mucositis.[8] Long after treatment concludes, chronic inflammation dominates, leading to radiation-induced fibrosis. Stiffening of the involved musculature, xerostomia, and even poor mucosal sensation all affect the highly coordinated deglutination process outlined in previous sections.[14,15] Patients are prone to malnourishment resulting in weight loss, with complaints of prolonged mealtimes, aspiration, coughing, difficulty initiating swallow, globus, and regurgitation.[16]

Other structural causes of dysphagia

Structural etiologies of dysphagia can also be classified by the presence of a physical barrier to the esophageal lumen. Examples of benign processes that can do this include foreign body, food impaction, esophageal stricture, esophageal rings, hiatal and paraesophageal hernias, and benign neoplasms.[8] Strictures are bands of fibrosis that can arise from several causes, namely radiation, infection, eosinophilic esophagitis, and caustic ingestion. Esophageal rings are circumferential congenital or acquired bands composed of mucosa and/or muscle, most commonly identified at the

squamocolumnar junction to form the so-called Schatzki ring. Hiatal and paraesophageal hernias may lead to dysphagia due to the accompanying esophageal reflux symptoms as well as external compression of the esophagus and/or LES by the herniated gastric fundus.

Tumors along the upper aerodigestive tract can also cause structural blockages of swallow function when they become large enough. In the pharynx and larynx, squamous cell carcinoma is the most common malignancy. In the esophagus, the 2 most common esophageal malignancies include squamous cell carcinoma and adenocarcinoma.[8] Depending on their size and location, these masses may obstruct or disrupt the esophageal lumen and/or coordinated peristalsis. Airway protection can also be affected concurrently. A malignant head and neck cancer should always be considered for progressive symptoms (sometimes rapid), in the presence of a neck mass, unexplained weight loss, or hemoptysis.

Lack of Airway Protection as a Cause of Dysphagia

Glottic insufficiency

Glottic insufficiency refers to the inability of the vocal folds to oppose or contact one another adequately. In this setting, a glottic gap occurs during swallowing, disrupting one of the key anatomic airway protection mechanisms and predisposing the patient to experiencing dysphagia.[17] In addition, glottic insufficiency leads to a diminished cough as the poor vocal fold apposition does not allow for a build-up of subglottic pressure to adequately expel aspirated contents.[18]

The recurrent laryngeal nerve (RLN) innervates numerous intrinsic laryngeal muscles and has a sensory role to the vocal fold surface and the infraglottis.[19] Dysfunction of this nerve can result in unilateral vocal fold paralysis (UVFP) and is a common cause of glottic insufficiency. In the hospital setting, patients found to have UVFP often have a history of recent neck or thoracic surgery or a recent intubation event. The new complaint of dysphonia or excessive coughing with the reinitiation of diet prompts evaluation, which typically reveals UVFP and signs of aspiration. This particular clinical picture is a common scenario where otolaryngologists are consulted for indirect laryngoscopy to confirm the UFVP and for consideration of vocal cord injection to improve dysphonia.[20] Although iatrogenic injury to the RLN is commonly associated with UVFP, it may also occur following intubation trauma, neck trauma, infection, autoimmune disease, or from an idiopathic condition.[21–23] Considering this wide underlying differential, it is important for the inpatient clinician to consider laryngeal pathology as an underlying cause of dysphagia.

Epiglottic dysfunction

Flexible and dynamic, the epiglottis is a cartilaginous, leaf-like structure extending from the oropharynx to the larynx. The epiglottis plays a critical role in protecting the endolarynx during deglutination. Retroflexion of the epiglottis is due in part by hyolaryngeal elevation, downward propulsion force of the food bolus, and the muscular and ligamentous connection allowing the epiglottic petiole to contact the arytenoid cartilages and form a seal.[8] The epiglottis is one of the 4 key airway protection mechanisms during deglutination along with the aryepiglottic folds, false vocal folds, and the true vocal folds.[24] Pathologic insults to the normal functioning of the epiglottis leading to incomplete retroflexion, prolonged retroflexion, stiffening, and horizontal axis tilt can contribute to dysphagia.[8] Therefore, factors that disrupt epiglottis flexibility or ability to fully retroflex such as cartilaginous calcification with age, trauma, connective tissue disease, radiation, mass, and hypopharyngeal crowding in the setting of osteophytes and surgical hardware can lead to symptomatic dysphagia as well.[25]

Inflammatory Causes of Dysphagia

Esophageal dysphagia can also occur in the setting of various inflammatory conditions that include but are not limited to eosinophilic esophagitis, infectious esophagitis, and caustic ingestion. Eosinophilic esophagitis, as the name implies, results from an inflammatory eosinophilic infiltrate of the esophagus, leading to dysphagia symptoms, classically characterized by recurrent food impaction events. Infectious esophagitis may occur as well and should always be suspected in acute dysphagia patients who are immunocompromised. Infectious esophagitis is typically caused by common pathogens (cytomegalovirus, herpes simplex virus, and candida).[8] Finally, caustic injury may result in direct immediate inflammation and delayed mucosal stricture depending on the properties and volume of the ingested agent.

Other Etiologies

There are also several etiologies that do not perfectly fit in any category. For example, some systemic diseases, such as scleroderma are frequently associated with swallow dysfunction. Greater than 70% of patients suffering from scleroderma experience end organ dysfunction secondary to systemic fibrosis leading to distal esophageal muscular atrophy and LES incompetence.[26] Medication side effects can also affect swallow function. See **Fig. 1** for many examples of xerostomia-inducing medications, which can frequently cause a subjective swallowing difficulty for many patients. Chronic opioid use may also affect swallowing in many patients, thought to affect motility over time.[27]

THE APPROACH TO EVALUATION AND DIAGNOSIS

Given the diverse possible etiologies for dysphagia, a systematic workup is essential to identify the underlying cause, localize the dysfunction, and guide management. The next section outlines the approach to dysphagia evaluation, organized into 3 key sections: history and physical examination, diagnostic studies, and consultation considerations.

History and Physical

There are several key features in the history that will help narrow the etiology of dysphagia. Many published algorithms recommend differentiating between oropharyngeal and esophageal dysphagia as the first step of the workup.[28,29] This is because these types of dysphagia have differing etiologies and treatment. Clues on history

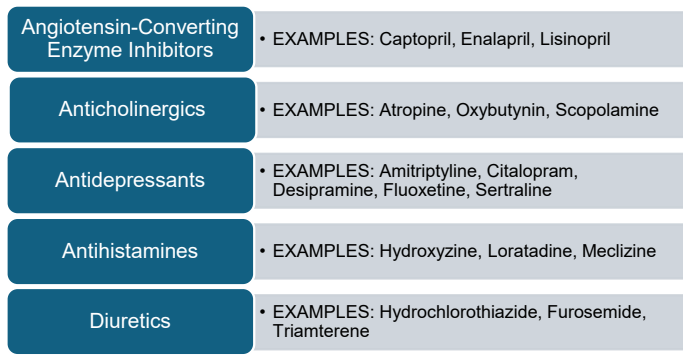

Fig. 1. Examples of xerostomia-inducing medications. (*Information from* Refs.[39,40])

alone can be helpful in steering the inpatient clinician toward an initial diagnosis, and a complete history is essential. A comprehensive review of systems should inquire about symptoms including hemoptysis, voice changes, pain, neurologic changes, weight loss, and infectious symptoms such as fevers or chills. Difficulty initiating swallow or coughing suggests oropharyngeal dysphagia while the sensation of food sticking lower in the chest can suggest esophageal pathology.[30,31] Rapid onset may point to an acute neurologic change or infectious etiology, whereas an insidious onset may point to a progressive etiology such as a mass or neuromuscular disorder. The presence of pain is helpful as pain likely indicates an inflammatory etiology or an invasive tumor. The presence of regurgitation of undigested food or halitosis may suggest a structural problem causing a retained food bolus. Voice changes may be due to the presence of an obstructive mass or vocal cord paralysis.[32] Dysphagia with certain textures can also be revealing. If solids are worse than liquids, then a structural cause is suggested. If both solids and liquids are affected, a neuromuscular etiology may be present.

Special attention should also be given to the patient's medical history, including past surgery or radiation of the head or neck, autoimmune disorders, neurologic disorders, and past smoking and/or alcohol use. Lastly, a full medication review is needed, as xerostomia can exacerbate oral dysphagia and is a common side effect of many medications (see **Fig. 1** for examples). As mentioned prior, chronic opioid use is also associated with dysphagia, including spastic dysfunction and dysmotility.[27]

When considering an algorithm for the diagnosis and management of the inpatient with dysphagia, we found good use in first using the history to determine whether inpatient versus outpatient management is indicated (**Fig. 2**). Chronic, stable symptoms that are not relevant to the acute medical issue requiring hospitalization may often be evaluated as an outpatient. Progressive symptoms or symptoms that are hindering recovery from the admitting diagnosis, such as malnutrition preventing recovery from an infectious illness, do deserve consideration for diagnosis and treatment as an inpatient. A second chart designed to guide diagnosis based on initial historical findings is included (**Fig. 3**).

A comprehensive physical examination includes a thorough oral cavity, oropharyngeal, and neck examination including examination within the mouth and the oropharynx for any abnormalities. Palpation of the neck for lymphadenopathy or neck masses should be performed. A cranial nerve examination focused on cranial nerves V, VII, IX, X, and XII should be included as well. If signs are suggestive of a mass or obstructive lesion within the pharynx, fiberoptic laryngoscopy should be performed as part of the physical examination. Laryngoscopy is crucial when considering concomitant laryngeal pathology, malignancy, or aspiration risk.[32]

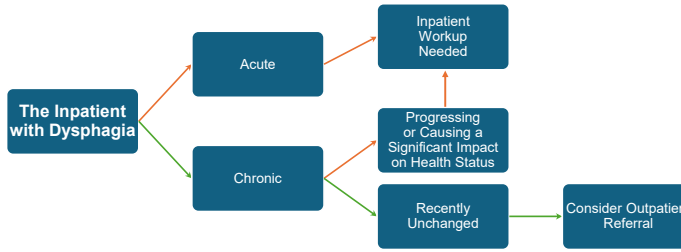

Fig. 2. Our approach to the inpatient with dysphagia.

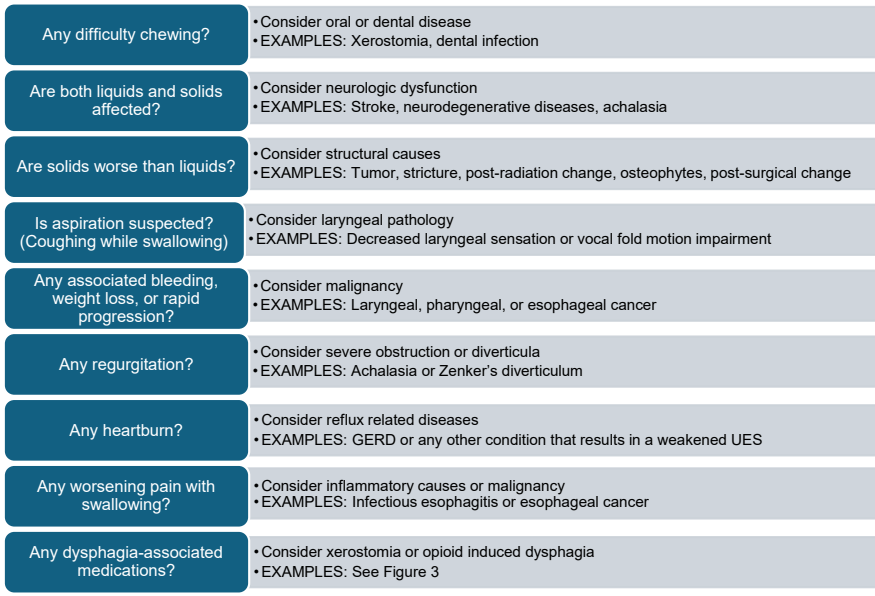

Fig. 3. A guide to the differential based on the patient's history. (*Information from* Refs.[40,41])

Studies

Depending on the history and physical examination, the next step in workup may include either structural or functional evaluations of the relevant anatomy. The choice of study depends on the suspected site and etiology of dysphagia. The first step for the evaluation of oropharyngeal dysphagia is often a swallow study, either modified barium swallow study (MBSS) or a fiberoptic endoscopic evaluation of swallowing (FEES).

MBSS is considered by some to be the gold standard for evaluation of dysphagia.[33] This study requires the patient to consume contrast under fluoroscopy, providing a dynamic evaluation of all phases of swallowing. This study allows for the identification of aspiration, residue, weakness, or blockages that may be contributing to dysphagia.[34] FEES is an alternative method of analysis that involves using a flexible endoscope to view the pharynx while the patient swallows a variety of food consistencies (eg. thins, puree, and soft). This allows for the assessment of any pharyngeal or laryngeal anatomic abnormalities, the presence of laryngeal penetration, and secretion burden.[35] The oral and esophageal phases are not well characterized with this study, so if there is concern for dysfunction during these phases of swallow then an MBSS is preferred. FEES may be preferred in some cases as it can be performed at bedside, does not require radiation exposure, and also uses real food and liquids.

The workup of esophageal dysphagia as a first line may include either an esophagram or an esophagogastroduodenoscopy (EGD). The esophagram is noninvasive and can reveal information about structural or motility abnormalities. EGD allows for the identification of mucosal abnormalities and allows for biopsy. If there is concern for a motility disorder of the esophagus, manometry may be obtained.[36]

Cross-sectional imaging of the brain, head, neck, and chest may be obtained if there is concern for any masses which may be causing compression, or additional imaging

is indicated for the suspected underlying etiology, such as appropriate MRIs if stroke is on the differential.[34]

Consultation

Multidisciplinary assessment is often required for accurate diagnosis and management of dysphagia. The specific consultations required depend on the suspected etiology. If there is concern for oropharyngeal dysphagia, Speech Language Pathology (SLP) consultation should be initiated. SLP is able to perform a bedside swallow assessment and determine if further testing with either MBSS or FEES is necessary.[37] If abnormalities are discovered, further consultation or workup may be suggested at that time. If there is no further workup or medical intervention necessary, the speech language pathologist may also be able to offer swallow therapy and counsel patients on helpful compensatory strategies.

If there is oropharyngeal dysphagia with concern for a structural or surgically correctable etiology, otolaryngology consultation is indicated. This includes tumors of the head or neck, vocal fold paralysis, UES dysfunction or ZD.[38] Otolaryngology consultation may include laryngoscopy to visualize the pharynx and larynx and biopsy of any suspicious lesions. Laryngoscopy can also be an important aspect of aspiration evaluation, if this is suspected as part of the patient's dysphagia.[38]

Gastroenterology consultation is essential for the workup of esophageal dysphagia. EGD is frequently offered to fully assess for mucosal or structural abnormalities. Additionally, if there is concern for motility-related or reflux-related dysphagia, then manometry or pH studies may be ordered, respectively, in collaboration with gastroenterology.[33]

Depending on the suspected etiology, other consultation may also be needed. A pulmonology consult may be indicated if recurrent aspiration from dysphagia has caused aspiration pneumonia or other pulmonary sequelae. Neurology consultation might be obtained for any patient with a suspected neurologic cause of dysphagia including cerebrovascular or neuromuscular etiologies. Nutrition consultation is frequently needed to optimize nutritional status, especially if chronic dysphagia has led to a need for enteral feeding if oral intake is unsafe. Particularly for inpatients who have other medical comorbidities, considering nutritional status is vital to their overall ability to heal and recover.[28]

The workup for dysphagia requires a comprehensive history and physical examination, targeted diagnostic studies, and appropriate consultations. In inpatients, these scenarios are often complex, and the importance of good communication and early collaboration should not be underestimated. Speedy identification of aspiration risk or malignancy is critical to the promotion of the best possible outcomes and to the prevention of complications such as pneumonia, malnutrition, or metastatic spread.

THE APPROACH TO MANAGEMENT

Similar to the approach to evaluation and diagnosis, the approach to developing a treatment plan for swallowing disorders is often multidisciplinary. Given the breadth of etiologies that can contribute to dysphagia, treatment approaches can include a variety of options including simple observation, swallow therapy, nutritional supplementation, and medical and surgical intervention. Medical management is typically focused on treating the underlying condition. For example, antifungals or antibiotics may be needed in the treatment of esophagitis, proton pump inhibitors for reflux-mediated disease, or antiparkinsonian medications for the management of Parkinsons. Surgical options to relieve an obstruction include dilation of esophageal stricture

or myotomy of an obstructive CP bar. Surgical treatments can also restore airway protection for appropriate patients, such as vocal cord injections for patients with glottic insufficiency, to improve safe swallow function. Botulinum toxin injections are used when the muscles of swallowing are too spastic or tight, such as in achalasia. Regardless of the treatment initiated, it is crucial to prioritize the nutritional status of the inpatient with dysphagia, ensuring the diet is adequate or that nutrition is supplemented in other ways when needed, such as via a nasogastric tube or gastric tube.

A collaborative approach among treatment teams is essential when addressing dysphagia symptoms in a patient-specific manner. Open channels of communication within these teams are of maximum benefit to treating a patient in a timely manner and for the clear delineation of a safe diet. Multidisciplinary care involving swallow therapy before and after surgical procedures for dysphagia can also be extremely beneficial and should be considered when possible.

IN SUMMARY

Dysphagia results from dysfunction at any stage of the complex neuromuscular process that is swallowing. The differential is vast, and our pages here only scratch the surface of possibilities. We present an algorithm to help steer the inpatient clinician when encountering these challenging patients, collaborating with all teams involved, arriving at an accurate diagnosis, and initiating proper management.

CLINICS CARE POINTS

- Initiate inpatient workup and treatment of any inpatient with dysphagia when symptoms are acute, progressing, or significantly impacting the patient's health status.
- Inpatients with dysphagia require careful consideration, as the nutritional implications on the patient's overall health and ability to recover can be extensive.
- As the differential and treatment options for dysphagia is vast, multidisciplinary care and coordination among treatment teams is essential to the determination of a safe diet, the optimization of nutritional status, and the preservation of swallow function.

DISCLOSURES

The authors have nothing to disclose.

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