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Diagnosis and medical management of esophageal dysmotility

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ABSTRACT

Esophageal motility disorders are often suspected in patients with dysphagia and noncardiac chest pain. Even though structural evaluation is important, the primary diagnostic tool is esophageal manometry. With the advent of high-resolution esophageal manometry, the Chicago classification has emerged as the primary scheme to categorize identified manometric abnormalities. However, although some manometric abnormalities associated with achalasia and distal esophageal spasm are well defined, the clinical significance of many identified manometric findings is less clear. Consequently, an understanding of the manometric findings defined by the Chicago classification is important. Furthermore, given the ambiguity associated with many of these findings, medical therapy is often challenging. This review attempts to provide a concise update on interpretation of high-resolution esophageal manometry specifically using the Chicago classification scheme. In addition, we highlight the current evidence for pharmacologic treatment for identified manometric abnormalities.

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1. Introduction

Dysphagia, chest pain, heartburn, and regurgitation constitute the major symptoms of esophageal dysfunction. When patients present with dysphagia, the first consideration is typically whether a structural abnormality is present. This is often assessed with diagnostic tests, which may include barium esophagram and esophagogastroduodenoscopy (EGD). However, when a structural cause is ruled out, a motility disorder of the esophagus is considered.

Understanding an esophageal motility disorder starts with understanding the physiology of normal deglutition. A normal swallow is defined by initial relaxation of both the upper and lower esophageal sphincter (LES), followed by a coordinated peristaltic contraction of the esophageal body that clears the bolus followed by closure of both sphincters. Esophageal dysmotility is defined by an impairment of the neuromuscular function responsible for this coordinated esophageal function. The muscular component of the esophageal wall is composed of skeletal and smooth muscle. Striated muscle comprises the proximal 5%, and mixes in the middle 35%-40% with an increasing proportion of smooth muscle distally. The distal 50%-60% of the esophagus is composed entirely of smooth muscle. Both the striated and smooth muscle portions of the esophagus contain a myenteric plexus between the longitudinal and circular muscles. In the smooth muscle portion, these enteric neurons are the relay neurons between the vagus and the smooth muscle. In the esophageal body and LES, vagal fibers synapse on myenteric plexus neurons with excitatory neurons. These mediate contraction of both longitudinal and circular muscle layers via nicotinic cholinergic receptors and inhibitory neurons largely via nitric oxide. There is a progressively increased proportion of inhibitory input distally in the esophagus, resulting in sequentially timed contraction, that is, peristalsis, within the esophageal body. In contrast, the LES maintains a tonic contraction at rest, with a swallow resulting in prolonged inhibition constituting LES relaxation.

Consequently, dysfunction at many levels of esophageal control can result in an esophageal motility disorder. Although the diagnostic approach is somewhat similar regardless of etiology, therapeutic options can differ depending on the unit of dysfunction. This article focuses on the diagnostic evaluation of specific esophageal motility disorders as well as available pharmacologic therapeutic options.

2. Diagnostic evaluation

2.1. Initial evaluation

Esophageal dysmotility is typically heralded by symptoms of dysphagia, most often for both solids and liquids, although solid food dysphagia may start first. These symptoms can be intermittent and may be associated with chest pain, heartburn, and

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regurgitation. When an esophageal motility disorder is suspected, diagnosis often involves multiple modalities. A structural evaluation to rule out mechanical causes of dysphagia is paramount. This can be achieved with barium esophagram and EGD. In addition, findings on these examinations may suggest an underlying esophageal motility disorder. As an example, the finding of an epiphrenic diverticulum on esophagram is associated with an underlying esophageal motility disorder in 80% of cases. Both examinations may also demonstrate esophageal dilation with retained food and secretions accompanied by a tight LES. However, when esophageal dysmotility is suspected, esophageal manometry is an essential part of the evaluation.

2.2. Esophageal manometry

Esophageal manometry represents the gold standard for evaluation of esophageal motility disorders. Although esophageal manometry has existed as a diagnostic tool since the 1950s, the advent of high-resolution esophageal manometry (HRM) has expanded our understanding of esophageal motor function. HRM uses an increased number of esophageal pressure sensors positioned 1 cm apart to allow intraluminal pressure to be monitored as a continuum spanning from the pharynx to the esophagus. This in turn allows manometric data to be displayed as pressure topographic plots and facilitates the simultaneous analysis and therefore coordination of different contractile segments of the esophagus. The result is a more precise and detailed definition of contractile characteristics of the esophagus [1].

Esophageal manometry is performed by transnasal placement of the HRM catheter. Positioning is confirmed by identification of 2 high-pressure zones, representing the upper and LES. After a 30-second baseline period, recording is performed to assess the esophagogastric junction (EGJ) at rest; the patient is given a series of ten 5-ml water swallows spaced 20-30 seconds apart. Results are displayed as a color-coded topographic map, with high pressures demonstrated in red and orange and low pressures in blue and green. Time is demonstrated along the *x*-axis, whereas the length of the esophagus along the *y*-axis (Figure 1). The Chicago classification has emerged as the primary means by which esophageal manometric findings on HRM are categorized. This classification scheme uses several basic metrics to provide a standardized categorization of nonobstructive dysphagia. We discuss the interpretation and categorization of HRM studies using the Chicago classification later [2].

After assessing the LES during the 30-second rest period, analysis begins with the first water swallow. Initiation of the swallow is marked by opening of the upper esophageal sphincter (UES) and EGJ, represented by a change in color from high pressure (red and orange) to low pressure (blue and green). EGJ relaxation is assessed first by measuring the integrated relaxation pressure (IRP). The IRP is the lowest mean pressure at the EGJ for 4 noncontinuous seconds measured during the 10-second window following UES opening. Normal relaxation of the EGJ is reflected by an IRP less than 15 mm Hg. Esophageal peristalsis is then evaluated within the esophageal body, propagating proximally to distally (Figure 1). The presence or absence of peristaltic activity is assessed first. If peristalsis is present, the swallow can then be assessed for spasm. Premature contraction or spasm is defined in part by shortening of the distal latency (DL). The DL is measured as the time from UES opening to the contractile deceleration point, which is represented as the inflexion point in the contractile front. The contractile deceleration point represents the termination of esophageal peristalsis and the onset of ampullary emptying of the distal esophagus [3] (Figure 2). The DL essentially is a measure of adequacy of inhibitory neuromuscular function, as normal peristalsis depends on a neural gradient with an increasing proportion

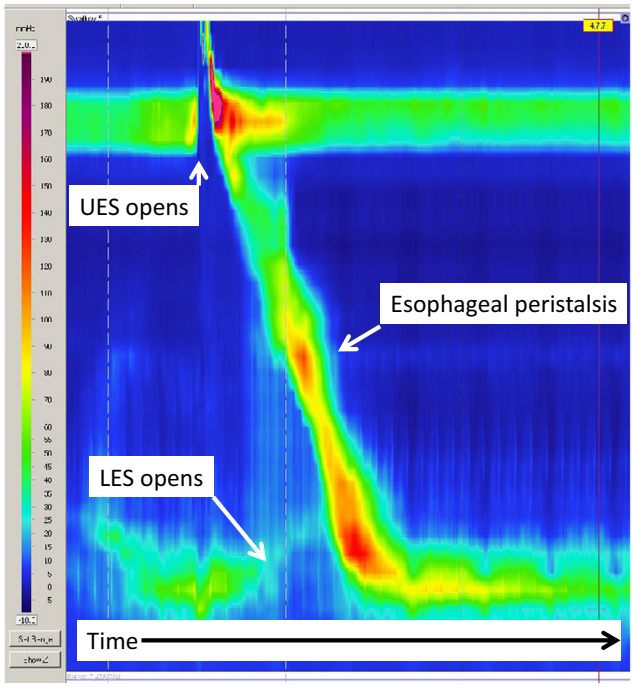

Fig. 1. Color topographic plot of a normal swallow on HRM. Normal swallow on HRM displayed as color-coded topographic map. Time is along the *x*-axis and length of the esophagus along the *y*-axis. The opening of the UES marks the start of the swallow, followed by LES opening and then esophageal peristalsis from proximal to distal. (Color version of figure is available online.)

of inhibitory ganglionic neurons in the distal esophagus. A short DL less than 4.5 seconds consequently represents early contraction of the distal esophagus and defines a spastic contraction [3-5] (Figure 2). Contractile vigor is assessed next by calculating the distal contractile integral (DCI). This metric uses an algorithm to integrate pressure, distance, and time along the esophagus to summarize the contractile vigor of the smooth muscle esophagus. Finally, peristaltic integrity is measured as breaks in the 20-mm Hg isobaric contour during a swallow. Each swallow is then classified using these metrics (Table 1), and the 10 swallows are taken together to classify the manometric study in an algorithmic fashion (Figure 3). This framework is employed to discuss specific manometric abnormalities as defined by the Chicago classification later.

3. Achalasia

Achalasia is a relatively rare condition, with an estimated annual incidence of 1 in 100,000 and prevalence of 1 in 10,000 [6]. Achalasia is the result of degeneration of ganglion cells in the myenteric plexus of the distal esophageal body and LES. Although the exact etiology is unclear, this appears to be an inflammatory autoimmune process precipitated by an environmental trigger. Recent data have suggested this may be infection with a herpes virus [7-9]. Rarely, achalasia can occur owing to a known cause such as Allgrove syndrome or Chagas disease [10,11]. The inflammatory process leads to a disproportionate loss of excitatory neurons compared with inhibitory neurons. This imbalance leads to poorly regulated simultaneous contractions in the esophageal body and incomplete LES relaxation [12]. The primary presenting symptom is dysphagia, typically for solids and liquids [13,14]. However, patients often learn to adapt by employing maneuvers to assist with food passage and often report minimal weight loss. As a result, clinical presentation and diagnosis is often delayed for several years. Regurgitation and chest pain also are frequently

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