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High- Resolution Manometry Application in Diabetic Patients with Dysphagia

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Abstract

Diabetes mellitus (DM) is a chronic metabolic disease that affects millions of people across the world. The disease affects numerous organs throughout the body, and significantly contributes to a decrease in quality of life. Many patients with diabetes complain of gastrointestinal (GI) symptoms as well, but the extent to which these symptoms correlate with abnormalities in GI motility remains to be elucidated. Esophageal manometry (EM) is believed to be the gold standard for diagnosing disorders of esophageal motility. EM assesses esophageal motility patterns by measuring pressure in the esophagus. There are two main types of manometric recording systems: the conventional EM and the high-resolution EM.

Keywords: Dysphagia, Diabetes, High-Resolution Manometry.

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1. Introduction

Diabetes mellitus (DM) is a metabolic disease, involving inappropriately elevated blood glucose levels. DM has several categories, including type 1, type 2, maturity-onset diabetes of the young (MODY), gestational diabetes, neonatal diabetes, and secondary causes due to endocrinopathies, steroid use, etc. The pathogenesis for T1DM and T2DM is drastically different, and therefore each type has various etiologies, presentations and treatments [1]. Dysphagia is defined as objective impairment or difficulty in swallowing, resulting in an abnormal delay in the transit of a liquid or solid bolus. The delay may be during the oropharyngeal or esophageal phase of swallowing. The second aspect of the definition of dysphagia is the subjective definition – the patient's sensation of a delay in transit of a liquid or solid bolus during swallowing. Both descriptions are relevant because some patients may lose the sensation of a delay in swallowing, while objective tests could show that they have dysphagia. Also, the patient's symptoms of a delay in swallowing may be potentiated or attenuated through sensory neural dysfunction [2]. Oesophageal manifestations of diabetic neuropathy, including abnormal peristalsis, and impaired lower esophageal sphincter tone, result in heartburn and dysphagia. The relationship between hyperglycemia and dysmotility is not well established.

Although many patients may have objective evidence of esophageal dysmotility or reflux, symptoms only occur in a minority of patients with diabetes. Autonomic neuropathy may manifest with gastrointestinal (GI) symptoms, as a result of the remodeling of the enteric nervous system (ENS) induced by diabetes. Loss in inhibitory and increase in excitatory enteric neurons, as well as decrease in sensory neuropeptides, may induce gastroparesis, esophageal dysmotility, constipation, diarrhea, fecal incontinence, or gallbladder atony [3]. The pathogenesis of dysphagia in diabetic patients is multifactorial and primarily attributed to autonomic neuropathy affecting the esophagus and swallowing mechanisms. Chronic hyperglycemia in diabetes leads to damage of the vagus nerve and enteric nervous system, which impairs the coordination and strength of esophageal peristalsis and the function of the lower esophageal sphincter. This can result in esophageal motility disorders such as ineffective esophageal motility, delayed esophageal clearance, and esophagogastric junction outflow obstruction. Additionally, diabetic myopathy may contribute by weakening the striated muscles involved in oropharyngeal swallowing [29]. Oesophageal manometry is the evaluation of movement and pressure of esophagus.

Conventional esophageal manometry usedprobes at every 5 cm in the esophagus to measure contraction and pressure. This was first utilized in the 1950s and had been the gold standard for diagnosing esophageal motility disorders. Recently, this technology was advanced and conventional esophageal manometry was replaced by high-resolution

esophageal manometry (HRM) as the gold standard [4]. HRM uses a high-resolution catheter to transmit intraluminal pressure data that are subsequently converted into dynamic esophageal pressure topography (EPT) plots. These transducer probes located approximately every 1 cm in the esophagus on the catheter. After the catheter is placed in the esophagus, patients get a baseline measurement and then do 10 wet swallows. From this data, a motility diagnosis can be made according to the Chicago Classification (version 3.0). Based on diagnosis, different treatments can be perused [4].

2. Epidemiology

Diabetes mellitus (DM) is a growing global health concern, with its prevalence rising steadily over the past few decades. According to the International Diabetes Federation (IDF), approximately 537 million adults (aged 20–79 years) were living with diabetes worldwide in 2021, representing about 10.5% of the global adult population. This number is projected to rise to 643 million by 2030 and 783 million by 2045 [5]. The prevalence of dysphagia is approximately 2.3% to 16% in general population. The prevalence increases with advances in age, and it is approximately 40% in people aged over 60. In hospitalized patients, approximately 14% to 18% of patients have dysphagia. In nursing homes, patients with dysphagia are in the range of 30% to 60% [6]. The global prevalence of oesophageal dysphagia in diabetic patients varies, but it ranges from 25% to 40%, depending on diagnostic criteria and study populations. The condition is often associated with diabetic autonomic neuropathy, which affects the smooth muscle function of the esophagus, leading to motility disorders such as ineffective esophageal motility or even achalasia-like syndromes.

While more prevalent in type 1 diabetics due to the longer disease duration, it is also reported in type 2 diabetes, particularly among the elderly. Despite its impact on quality of life and nutritional status, esophageal dysphagia remains underdiagnosed, highlighting the need for increased clinical awareness and standardized diagnostic approaches [27]. Tatari and Bassiouny (2018) investigated the prevalence of oropharyngeal dysphagia among diabetic patients in Egypt. In this cross-sectional study, 200 Egyptian adults with type 1 or type 2 diabetes mellitus, aged between 18 and 59 years, were screened. The results indicated that age progression and female gender were significant risk factors for dysphagia among diabetic patients. The most commonly reported symptom was "I cough when I eat." Notably, there was no significant association between the type or duration of diabetes and the presence of oropharyngeal dysphagia. This study highlights the importance of routine screening for swallowing difficulties in diabetic patients, particularly among older females, to prevent potential complications such as malnutrition and aspiration pneumonia [28].

3. Etiology of dysphagia

Dysphagia could occur during the oropharyngeal or pharyngeal phase of swallowing. Oropharyngeal dysphagia is a delay in the transit of liquid or solid bolus during the oropharyngeal phase of swallowing. It could be due to neurological, muscular or anatomical causes. The neurological causes include cerebrovascular accidents, brainstem infarctions with cranial nerve involvement, basal ganglia lesions as in Parkinson disease, head and neck injuries and surgery, multiple sclerosis, central nervous tumor, botulism, amyotrophic lateral sclerosis, supranuclear palsy and degenerative cervical spine disease. Muscular causes include polymyositis, muscular dystrophy and myasthenia gravis. Anatomical causes include Zenker diverticulum, enlarged thyroid, esophageal web, tumors, abscess, and external compression by an aortic aneurysm. Also, cervical discectomy and fusion may be associated with postoperative dysphagia [7]. Oesophageal dysphagia - could be caused by mechanical obstruction, motility disorders, rheumatological disorders and medications. Mechanical obstruction causes include rings, esophageal stricture, esophageal carcinoma and eosinophilic esophagitis. Motility disorder causes include esophageal spasm, achalasia, ineffective esophageal motility and scleroderma [8]. Rheumatological disorders include Sjogren's syndrome, systemic lupus erythematosus, mixed connective tissue disease, rheumatoid arthritis and systemic sclerosis (as part of the CREST syndrome). Medications that cause dysphagia include antipsychotic (e.g., olanzapine, clozapine), tricyclic antidepressants, potassium supplements, non-steroidal anti-inflammatory drugs, bisphosphonates, calcium channel blockers, nitrates, theophylline, alcohol, medications with immunosuppressant effects (e.g., cyclosporine) can predispose to infective esophagitis and dysphagia and opioids [9].

3.1. Physical Examination

As oropharyngeal dysphagia may be just one of many manifestations of various neurological conditions such as stroke and Parkinson disease, therefore a complete neurological examination is important when a neurological disorder is suspected. Examination of the neck is also important to exclude any mass lesion that might cause obstruction, or tenderness including lymphadenopathy which might point to an infective or inflammatory process. In contrast, physical examination is generally non-contributory in oesophageal dysphagia; however, it is important to examine the skin and joints for features of connective tissue disorders such as systemic sclerosis, given the association of these conditions with oesophageal hypomotility. The oral cavity should be inspected for dentition, evidence of xerostomia and of infective conditions such as candidiasis. Noting major chest and spine deformities may provide clues for underlying syndromes. Complications of dysphagia such as malnutrition, weight loss and pulmonary complications may also be evident on physical examination [10].

3.2. Investigations

A barium swallow is an imaging modality that uses real-time fluoroscopy and barium to evaluate esophageal dysphagia. It is helpful in assessing any morphologic and motility abnormalities in the pharynx and esophagus. Over the years, several advancements have been made in technique of the barium swallow [11]. Barium swallow has been shown to be useful in diagnosing esophageal webs, rings, and diverticulae. Schatzki rings with a diameter of less than 13 mm are usually associated with dysphagia [12]. Esophageal tumors appear as intraluminal or intramural filling defects on barium swallow. Some studies have also reported that double-contrast barium swallow sensitivity in diagnosing esophageal cancers is more than 95%. A barium swallow is also helpful in identifying extrinsic compression of the esophagus [13]. A double-contrast barium swallow is also useful in assessing

esophageal motility. "Bird's beak" and the "megaesophagus" appearance on barium swallow associated with primary achalasia. Narrowing on barium swallow extending more than 3.5 cm above gastroesophageal junction, which appears as a "rat-tail sign," is commonly associated with secondary achalasia "Corkscrew" or "rosary beard" appearance on barium swallow characteristically associated with diffuse esophageal spasm [14]. Esophagogastroduodenoscopy (EGD) is one of most essential diagnostic and therapeutic modalities in management of the dysphagia.

EGD may conclude the diagnosis in patients with unrevealing imaging studies. Therapeutic intervention, including stricture dilation and esophageal biopsy, can be performed during EGD. Esophageal mass requires a biopsy to establish a diagnosis. However, even in patients with a normal-appearing esophagus, biopsies in middle and lower esophagus are recommended to evaluate for eosinophilic esophagitis (EoE). In high-risk individuals, lower esophageal assessment for the esophagitis and Barrett's esophagus can be performed. Dysphagia due to gastric cardiac pathologies is often missed with other imaging modalities, and it can be diagnosed with retro-flexed view evaluation during an EGD [15]. Endoscopy helps to diagnose esophageal structural abnormalities such as mucosal abnormalities, rings, retained food, strictures, and masses. Esophageal strictures and gastroesophageal reflux disease (GERD) are most common EGD findings. A biopsy can be done during endoscopy to rule out underlying malignancy. Studies have shown that multiple mucosal biopsies have a sensitivity of 96% in diagnosing esophageal cancer. Complications associated with EGD include infection, bleeding, and esophageal perforation [16]. Manometry is a valuable modality that is useful in diagnosing esophageal dysphagia and is particularly helpful in patients in whom a motility disorder suspected. HRM is more sensitive than conventional manometry [10].

4. HRM protocol and analysis

4.1. Specimen Collection

Patients are brought into the clinic on the day of the test. They are instructed to avoid certain medications, including H2-blockers, proton pump inhibitors, calcium channel blocks, nitrates, opioids, sedative medications, and even caffeine for at least 24 hours. If patients have undergone previous esophageal surgeries including gastric fundoplication, Heller myotomy, the per-oral esophageal myotomy (POEM), pneumatic dilations (PD), or even botulinum injections, these may falselyalter the findings on HRM. Finally, patients with large hiatal hernias or peptic strictures may have false HRM findings [17]. Patients should also fast for a minimum of six hours before the test. The catheter is inserted into the esophagus. With up to 36 sensor probes, the HRM catheter is positioned at the upper esophageal sphincter (UES), the lower esophageal sphincter (LES), and the throughout the esophageal body [18].

4.2. Procedure

The patient is then placed in supine position and does a baseline swallow followed by 10 swallows of water (5 mL each) with at least 30 seconds in between. During swallow, sensors detect multiple parameters including the integrated relaxation pressure (IRP), distal contractile integer (DCI), contractile deceleration point (CDP), and distal latency (DL) to produce color pressure topography plots [19].

4.3. Indications

Patients that present with symptoms of dysphagia, odynophagia, gastroesophageal reflux (GERD), or non-cardiac chest pain are typically worked up for esophageal pathology. Patients typically first undergo upper gastrointestinal (GI) swallow study or an EGD to rule out structurallesions or masses. Once structural lesions or masses have been excluded, motility disorders of the esophagus are considered. The gold standard in the evaluation of esophageal motility disorders is HRM, which has replaced conventional manometry [20].

4.4. Potential Diagnosis

Based on the results of HRM, the patient will be classified into 1 of 4 categories based on the Chicago III Classification: [21]

(1) Incomplete LES relaxation (achalasia or esophagogastric junction (EGJ) outflow obstruction).
(2) Major motility disorders (distal esophageal spasm, hypercontractile or jackhammer esophagus and absent contractility).
(3) Minor motility disorders (ineffective esophageal motility or fragmented peristalsis).
(4) Normal esophageal motility.

4.5. Normal and Critical Findings

The first category in the Chicago Classification is incomplete LES relaxation, which includes achalasia and EGJ outflow obstruction. Achalasia is defined as the absence of esophageal peristalsis with incomplete LES relaxation. In the Chicago Classification, the diagnosis of achalasia is based on an elevated IRP 4s in combination with failed peristalsis or spasm. The IRP is a measure of the relaxation of the EGJ, with typical values (less than 15.0 mmHg), although this is catheter specific. During normal swallows, the LES at the EGJ relaxes to allow the food bolus into the stomach. If the LES fails to relax, this is indicated by elevated IRP. The IRP 4s is a software mean calculation of the EGJ pressure in the lowest 4 seconds of the 10-second swallow after UES opening [22]. If patients have an IRP greater than the upper limit of normal (ULN) along with failed peristalsis or a spasm, then they are consideredto have achalasia. Achalasia is then further divided into three distinct subclasses based on the pattern of contractility in the esophageal body. In type I (classic achalasia), no pressure waves are recorded in the distal esophagus as there is 100% failed peristalsis. Failed peristalsis is defined by DCI less than 100 mmHg cm/s for type I achalasia. Type II is characterized by at least 20% pan-esophageal pressurizations with no normal peristalsis. Type II achalasia is the most prevalent subtype of achalasia. These patients may have a distal latency of under 4.5 seconds, but the diagnosis of type II achalasia is dependent on >20% swallows with pan-esophageal pressurizations.

In Type III, at least 20% of swallows reveal rapidly propagating or spastic simultaneous contractions with a distal latency of below 4.5 seconds and no normal peristalsis. [13] These spasms are typically distal on the esophageal body and donot have the pan-esophageal pressurizations seen with type II achalasia. If patients have high IRP, indicating failed LES relaxation, with weak peristalsis or do not fit into achalasia

subclasses I-III, then they areconsidered to have EGJ outflow obstruction. EGJ outflow obstruction is largely a manometric diagnosis but has a rising incidence after the advent of HRM [10]. The second category of the Chicago Classification is major disorders in peristalsis, which include distal esophageal spasm, hypercontractile or jackhammer esophagus, and absent peristalsis. Distal esophageal spasm (DES) is diagnosed on HRM in patients with normal IRP, normal DCI, but distal latency less than 4.5 seconds. Distal latencyis measured from the UES swallow induced relaxation to the contractile deceleration point (CDP). Normal distal latency is greater than 4.5 seconds, with anything shorter than 4.5 seconds is considered esophageal spasm. The CDP is the point on which peristaltic wave velocity slows, demarcating peristalsis from ampullary emptying.

The normal CDP is within 3 cm of the LES. The HRM software calculates the CDP and distal latency [1]. Hypercontractile or jackhammer esophagus is defined as having a distal contractile integral (DCI) of more than 8000 mmHg cm/s. DCI is a multiplication of length, duration, and amplitude of contractions. Basically, it is the force of peristalsis. A DCI less than 450 mmHg cm/s indicates weak peristalsis, whereas a DCI higher than 8000 mmHg cm/s indicates hypercontractile peristalsis. Absent contractility is when there is a complete failure of peristalsis with normal IRP. These patients typically have a DCI of less than 100 mmHg cm/s [23]. The third category of the Chicago Classification is minor disorders of peristalsis, which include ineffective esophageal motility or fragmented peristalsis. Ineffective esophageal motility is diagnosed when >50% of swallows are ineffective, as defined by failed (DCI of less than 100mmHg cm/s) or weak (DCI 100 mmHg cm/s to 450 mmHg cm/s) peristalsis. These patients have normal IRP and distal latency. Fragmented peristalsis is defined as >50% of swallows with a large break (>5 cm) between peristaltic contractions and not having ineffective esophageal motility. Minor disorders of peristalsis are conditions with impaired esophageal bolus transit [24].

4.6. Complications

Complications of HRM are rare. Placing the HRM nasogastric sensory catheter may cause discomfort in the nose or throat. During placement of the catheter, patients may experience a gagging sensation that may lead to emesis. Caution should be exercised when placing the catheter in patients who have recently had esophageal surgery or in patients with esophageal varices. Finally, there have been rare incidence of esophageal perforation in patients with severe achalasia during HRM [25].

5. HRM findings if diabetic patients with dysphagia

The study conducted by Muroi et al. (2021), which investigated esophageal motility disorders in diabetic patients with dysphagia using high-resolution manometry (HRM) found that 60% of diabetic patients with dysphagia exhibited esophageal motility disorders, significantly higher prevalence compared to 29.6% in non-diabetic patients. Notably, minor disorders such as ineffective esophageal motility (IEM) and fragmented peristalsis were more common in diabetic patients (45% vs. 11%). Additionally, diabetic patients showed lower values in distal contractile integral (DCI) and integrated relaxation pressure 4s (IRP 4s), indicating impaired esophageal clearance. The presence of esophageal motility abnormalities in diabetic patients was also associated with higher incidences of diabetic complications, including neuropathy, retinopathy and nephropathy [26].

6. Summary

Diabetes mellitus (DM) affects nearly all different body systems including the gastrointestinal system. Above half of diabetic patients report gastrointestinal symptoms including esophageal ones. The prevalence of esophageal symptoms, such as heartburn and dysphagia, is estimated to be between 25% and 87%. These are thought to be due to esophageal motility disorders and gastroparesis esophageal manometry (EM) is believed to be the gold standard for diagnosing disorders of esophageal motility. EM assesses esophageal motility patterns by measuring pressure in the esophagus. There are two main types of manometric recording systems: the conventional EM and the high-resolution EM. In diabetic patients, EM revealed up to 65% of Eosophageal Motility Disorders (EMD). Major patterns of EM abnormalities are incomplete lower esophageal sphincter (LES) relaxation, delayed peristalsis progressing, abnormal amplitude in distal peristaltic waves, or high incidence of simultaneous contractions (>10%). Pathophysiology of Esophageal Motility Disorders (EMD) in patients with DM seems to be multifactorial. Main mechanisms described include hyperglycemia as well as autonomic neuropathy (AN). In fact, several studies have reported that uncontrolled Diabetes Mellitus is associated with a higher frequency of EMD. Esophageal dysfunctions occur frequently in patients with diabetic autonomic neuropathy. The prevalence of EMD in diabetic patients with the AN varies widely ranging from 13% to 70%.

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