Dysphagia

Section 6 Alterations in Gastrointestinal Function | Part 2 – Cardinal Manifestations & Presentation

KEY CLINICAL POINTS

1 Dysphagia is defined as difficulty with the transit of food or liquid from the mouth to the hypopharynx or through the esophagus.

2 Subclassified by location (oral, pharyngeal, esophageal) and mechanism (structural vs. propulsive/motor).

3 Intermittent dysphagia to solids suggests structural issues; constant dysphagia of both solids and liquids suggests motor abnormalities.

4 Rapid progression (weeks to months) raises concern for neoplasia; slow/stable progression (years) indicates benign processes like Schatzki rings or eosinophilic esophagitis.

5 Hoarseness preceding dysphagia indicates a primary laryngeal lesion; hoarseness following dysphagia suggests recurrent laryngeal nerve involvement by malignancy.

6 Scleroderma presents with absent peristalsis and a weakened LES, predisposing to peptic stricture formation.

7 Eosinophilic esophagitis is most prevalent in Caucasian males aged 20–40 years with a history of atopy.

8 Cricopharyngeal bars are common radiographic findings; often asymptomatic, requiring exclusion of other etiologies before treatment.

9 Oropharyngeal dysphagia is characterized by nasal regurgitation, aspiration, and cough with swallowing.

10 Esophageal dysphagia is typically localized to the chest or neck.

11 Food impaction is a hallmark of structural dysphagia.

12 The esophageal lumen must be >13 mm to avoid common solid food dysphagia; however, it can occur in larger diameters with motor dysfunction or poor mastication.

DEFINITION & OVERVIEW

- **Definition:** Dysphagia is difficulty with swallowing, referring to problems with the transit of food or liquid from the mouth to the hypopharynx or through the esophagus.
- **Classification by Location:** Oral, pharyngeal, or esophageal.
- **Classification by Mechanism:**
  - **Structural:** Caused by an oversized bolus or a narrow lumen.
  - **Propulsive (Motor):** Due to abnormalities of peristalsis or impaired sphincter relaxation after swallowing.

ETIOLOGY & PATHOPHYISIOLOGY

Physiology of Swallowing

- **Oral Phase:** Voluntary; includes preparation (mastication/salurine) and transfer (tongue pushes bolus into pharynx).
- **Pharyngeal Phase:** Centrally mediated; involves larynx elevation, UES opening, and peristaltic clearance.
- **Esophageal Phase:** Primary peristalsis involves sequenced inhibition followed by contraction of musculature along the entire length of the esophagus.
- **UES Function:** Remains closed at rest due to cricopharyngeus muscle contraction; opens during swallowing via cessation of vagal excitation and contraction of suprahyoid/geniohyoid muscles.

Neuromuscular Apparatus

- **Striated Muscle (Oral, Pharynx, UES, Cervical Esophagus):**
  - Innervated by cranial nerves V, VII, IX, and X.
  - UES: Composed of cricopharyngeus muscle and adjacent inferior pharyngeal constrictor.
- **Smooth Muscle (Distal Esophagus, LES):**
  - Controlled by myenteric plexus.
  - Excitatory neurons: Acetylcholine and substance P.
  - Inhibitory neurons: Vasoactive intestinal peptide and nitric oxide.
- **LES Function:**
  - At rest: Contracted due to excitatory ganglionic stimulation and intrinsic myogenic tone.
  - During swallowing: Deglutitive inhibition causes relaxation until the peristaltic sequence is complete.

Pathophysiology of Dysphagia

- **Structural vs. Propulsive:**
  - Structural: Caused by oversized bolus or narrow lumen.
  - Propulsive (Motor): Due to peristalsis abnormalities or impaired sphincter relaxation.
- **Scleroderma:** Characterized by absent peristalsis and a weakened LES → risk of peptic stricture.
- **Cricopharyngeal Bar:** Common radiographic finding; often asymptomatic.

→ Must rule out other etiologies before treatment.

CLINICAL FEATURES

Oropharyngeal Dysphagia

- **Key Symptoms:** Nasal regurgitation, aspiration, cough with swallowing (may indicate tracheoesophageal fistula), and food impaction.
- **Hoarseness Timing:**
  - Preceding dysphagia → Primary laryngeal lesion.
  - Following dysphagia → Recurrent laryngeal nerve compromise (likely malignancy).
- **Clinical Nuance:** Patients with oropharyngeal dysphagia often have greater difficulty managing liquids than solids.

Esophageal Dysphagia

- **Anatomy:**

  - Cervical esophagus: pharyngoesophageal junction to suprasternal notch.
  - Thoracic esophagus: suprasternal notch to diaphragmatic hiatus.
  - Dimensions: Length 18–26 cm; lumen 2 cm (AP) imes 3 cm (lateral).
- **Clinical Thresholds:**
  - Solid food dysphagia common when lumen <13 mm.
- **Propulsive Disorders:**
  - Absent peristalsis: Complete absence of contraction or nonperistaltic, disordered contractions.
  - Distal Esophageal Spasm (DES): Normal LES function; disordered motility in esophageal body.
  - Scleroderma: Absent contractility + severe weakness of the LES.

Temporal and Progression Patterns

- **Rapid Progression (weeks/months):** High suspicion for neoplasia.
- **Slow/Stable Progression (years):** Suggests benign process (e.g., Schatzki ring, eosinophilic esophagitis).
- **Food Type:**
  - Solid only → Structural dysphagia (Exception: Scleroderma patients often have solid-only dysphagia despite motor issues).
  - Solid and liquid → Motor abnormality.
- **Clinical Correlation:** Chest pain with a history of heartburn suggests peptic stricture or esophageal adenocarcinoma.

DIFFERENTIAL DIAGNOSIS

Structural Causes

- **Common:** Schatzki's rings, eosinophilic esophagitis, peptic strictures.
- **Other:** Zenker's diverticulum, cricopharyngeal bar, neoplasia, cervical web, osteophytes, congenital abnormalities, post head and neck surgery, chemotherapy mucositis, radiation, corrosive injury, infection, cerebral palsy, brainstem tumor, Guillain-Barré, paraneoplastic, scleroderma, surgical stenosis, hiatal hernia, lichen planus, ringed esophagus, congenital esophageal stenosis.

Propulsive (Motor) Causes

- **Conditions:** Cerebral vascular accident, polymyositis, myasthenia gravis, achalasia (primary and secondary), diffuse esophageal spasm, eosinophilic esophagitis, Behcet's syndrome, bullous pemphigoid, Crohn's disease, mixed connective tissue disorders, oculopharyngeal muscular dystrophy, Huntington's chorea syndrome, sarcoidosis, radiation esophagitis.

Myogenic Causes

- **Conditions:** Myasthenia gravis, polymyositis, oculopharyngeal muscular dystrophy, Huntington's chorea syndrome, and myotonic dystrophy.

Neurogenic Causes

- **Conditions:** Cerebrovascular accidents, Parkinson's disease, amyotrophic lateral sclerosis, brainstem tumor, Guillain-Barré, multiple sclerosis, and post-polio syndrome.

DIAGNOSTIC APPROACH

1. **History:**
    – Location: Suprasternal notch (30% are oropharyngeal) vs. Chest (esophageal).
    – Circumstances: Solid only (structural) vs. Solid/Liquid (motor).
    – Progression: Rapid → Neoplasia; Slow → Benign.
    – Associated Symptoms: Hoarseness, cough with swallowing (tracheoesophageal fistula?), odynophargia (infection/pill-induced).
    – Risk Factors: Atopy (eosinophilic esophagitis), immunocompromised status (Candida, HSV, CMV, Kaposi's, lymphoma), prior surgery/radiation.
2. **Physical Examination:**
    – Assess for bulbar or pseudobulbar palsy (dysarthria, dysphonia, ptosis).
3. **Imaging and Endoscopy:**
    – Fluoroscopy: Requires conscious/cooperative patient; assesses pharyngeal phase, bolus retention, and UES opening.
    – Direct Laryngoscopy: Assess structural abnormalities requiring biopsy.

MANAGEMENT & TREATMENT

1. **Initial Assessment:** Determine if dysphagia is related to motor disorders, structural disorders, or reflux disease (GERD).
2. **Diagnostic Pathway:** Utilize the algorithm in Figure 47-2 (Flowchart 1) to determine specific management based on location and consistency.
3. **Structural Management:** Address mechanical obstructions such as Schatzki rings, peptic strictures, or tumors.
4. **Motor Management:** Address motility issues like achalasia or systemic sclerosis.

SPECIAL CONSIDERATIONS

Immunocompromised States

- **Considerations:**
    – Opportunistic infections: Candida, HSV, CMV.
    – Tumors: Kaposi's sarcoma, lymphoma.

Atopy

- **Eosinophilic Esophagitis:** High suspicion in patients with atopy; most prevalent in Caucasian males aged 20–40 years.

KEY PEARLS & HIGH-YIELD POINTS

- **Hoarseness Timing:** Pre-dysphagia → Larynx; Post-dysphagna → Recurrent laryngeal nerve.
- **Scleroderma:** Characterized by absent peristalsis and weak LES → peptic stricture.
- **Cricopharyngeal Bar:** Common finding; often asymptomatic. Rule out other causes before treatment.
- **Eosinophilic Esophagitis:** High prevalence in Caucasian males (20–40 years) with atopy.
- **Suprasternal Notch:** Distal dysphagia is referred proximally approximately 30% of the time.

FLOWCHARTS

Approach to the patient with dysphagia (Flowchart 1)

1. **Identify Location/Symptoms:**
    - Oropharyngeal: If associated with nasal regurgitation, muscle weakness, or ENT symptoms.
    - Esophageal: If localized to throat/chest.
2. **Determine Consistency (for Esophageal):**
    - Solid and liquid → Propulsive (Motor) pathway.
    - Solid only → Structural pathway.
3. **Sub-classify Propulsive (Motor):**
    - Neurogenic: Myasthenia gravis, ALS, etc.
    - Myogenic: Achalasia, Systemic sclerosis, etc.
4. **Final Outcomes:**
    - Oropharyngeal Structural: Berner's diverticulum, Cricopharyngeal web, Zenker's diverticulum, etc.
    - Oropharyngeal Pulsive: Cervical vascular, Parotitis, etc.
    - Esophageal Structural: Esophageal cancer, Esophageal web, etc.
    - Esophageal Neurogenic: Myasthenia gravis, ALS, etc.
    - Esophageal Myogenic: Achalasia, Systemic sclerosis, etc.

Clinical Value of Patient History (Flowchart 2)

1. **Purpose:** Use history to provide a presumptive diagnosis or limit the differential diagnoses.
2. **Key Data Points:**
    - Location → Oropharyngeal vs. Esophageal.
    - Circumstances → Structural vs. Propulsive/Motor.
    - Progression → Benign vs. Malignant.