www.jjgastro.com

Journal of Gastroenterology Research and Practice

Case Report

Open Access, Volume 6

Early esophageal cancer presenting with esophageal spasm: A case and its clinical significances

Ming-Xian Yan*

Department of Gastroenterology, The First Affiliated Hospital of Shandong First Medical University & Shandong Provincial Qianfoshan Hospital, Ji'nan, China.

***Corresponding Author: Ming-Xian Yan**
Department of Gastroenterology, The First Affiliated Hospital of Shandong First Medical University & Shandong Provincial Qianfoshan Hospital, Ji'nan, China.
Email: yanmingxian@sdfmu.edu.cn

Received: Jun 03, 2026
Accepted: Jun 23, 2026
Published: Jun 30, 2026
Archived: www.jjgastro.com

Abstract

This article reports a case of early esophageal cancer with esophageal spasm as the main clinical manifestation. The male patient, 58-year-old, was hospitalized due to recurrent dysphagia accompanied by vomiting. Routine laboratory tests, as well as electrocardiograms and cardiac ultrasound, showed no abnormality; upper gastrointestinal barium meal fluoroscopy revealed dilation of the upper esophagus and spasm with stenosis in the distal esophagus; Computed tomography showed fluid accumulation in the upper esophagus and stenosis in the lower esophagus; gastroscopy revealed diffuse inflammatory changes in the esophageal mucosa, and local tissue biopsy pathology reported squamous cell carcinoma in situ with possible invasion. The patient then underwent surgery, and the post-operative pathology confirmed well-differentiated mucosal squamous cell carcinoma invading the muscularis mucosa, while the remaining esophageal mucosa showed local high-grade squamous intraepithelial neoplasia, and no cancer was found in the lymph nodes. Fifteen days post-surgery, a follow-up barium meal fluoroscopy showed good patency of the esophagus with no signs of spasm. This case suggests that patients presenting as esophageal spasm should be vigilant for the possibility of early-stage cancer.

Introduction

Distal esophageal spasm is a type of esophageal motility disorder characterized by uncoordinated and high-amplitude contractions of the esophagus, leading to impairment of food bolus propulsion. The underlying mechanism involves dysrhythmia of muscular contractions and abnormal contractile activity of esophageal smooth muscle. The primary clinical manifestations include intermittent dysphagia and non-cardiac paroxysmal chest pain, with atypical symptoms such as food retention sensation and regurgitation [1]. Dietary stimuli, emotional fluctuations, and rapid eating may aggravate symptoms [2]. Long-term and recurrent esophageal spasms can lead to malnutrition and psychological disorders secondary to reduced oral intake, necessitating active therapeutic intervention. Although primary esophageal spasm is a functional disorder, pathological esophageal conditions may also induce spastic episodes. Therefore, comprehensive diagnostic evaluation is essential to exclude organic etiologies and prevent misdiagnosis-related treatment delays.

Case presentation

The patient was a 58-year-old male who began experiencing difficulty swallowing without any obvious triggers 6 months ago, accompanied by a feeling of obstruction while eating, occasional nausea, and vomiting, but no hematemesis. The symptoms appeared intermittently and were accompanied by reflux. There was no chest and abdominal pain, no bloating or diarrhea, no cough or phlegm, and no fever or chills. He did not have chest tightness or shortness of breath, and tests of stool and urine were normal. No significant weight changes had occurred in the past six months. He had no history of chronic diseases such as hypertension, coronary heart disease, or diabetes, and had no history of surgery or trauma, nor any food or drug allergies.

He had been smoking for over 20 years, approximately 10 cigarettes a day, and drunk alcohol occasionally. He denied any family history of hereditary diseases.

When he was admitted to the hospital, his body temperature, pulse, respiration and blood pressure were all normal with a body weight at 60 kg. Physical examination showed the following medical information: asthenic build with moderate nutritional status; alert and conscious; no jaundice, petechiae, or ecchymoses observed on the skin; no superficial lymph nodes found; sclera without jaundice; ears and nose unremarkable, lips without cyanosis; breath sounded in both lungs clear, no rhonchi or crackles auscultated; heart rate at 72 beats/min with regular rhythm, no pathological murmurs detected; abdomen flat, soft and non-tender; liver and spleen not palpable below the ribs; Murphy's sign negative; no percussion pain in the liver or kidney areas; bowel sounds normal; shifting dullness negative; no edema in both lower extremities.

Following hospital admission, the patient was treated with mucosal protectants and proton pump inhibitors, along with symptomatic treatment. Blood routine tests and biochemical indicators as well as tumor markers were normal. No abnormalities were found in the stool examination. The electro-cardiogram and echo-cardiogram showed no abnormalities. An upper gastrointestinal barium meal study revealed dilation of the upper esophagus, spasms in middle and lower esophagus, and a very obvious local narrowing in the lower esophagus; barium was found in small intestine in four minutes after swallowing barium. Computed Tomography (CT) showed fluid accumulation in the upper esophagus, narrowing in the lower esophagus, and no occupying lesions in the esophagus or mediastinum.

Further gastroscopy revealed that the entire esophageal mucosa showed a cloudy white change, with a rough, reddened area measuring approximately 1.5 centimeters square observed on the posterior wall of the esophagus at 25 centimeters from the incisors, exhibiting granular changes. The background staining was positive, and narrow-band imaging and magnified endoscopy showed the characteristics of capillary loops in the esophageal epithelial papillae to be marked by full vascular dilation, tortuosity, uneven thickness, and varied morphology. After iodine staining, the esophageal mucosa displayed a mottled change, with a positive pink sign at 25 centimeters from the incisors, and tissue was taken for pathological examination. Severe spasm of the lower esophagus was noted, with endoscopic ultrasound showing significant thickening of the esophageal wall's muscularis propria, with the thickest part measuring approximately 6.7 millimeter (Figure 1A-1E). The esophageal biopsy pathological report indicated squamous cell carcinoma in situ located 25 centimeters from the incisors, with the possibility of early invasion not excluded.

The patient was subsequently transferred to the esophageal surgery department for surgical treatment. During the surgery, the tumor was found in the middle segment of the esophagus, leading to an esophagectomy, esophagogastric anastomosis, and lymph node dissection. The post-operative pathological report showed well-differentiated mucosal squamous cell carcinoma, which invaded the muscularis mucosa but did not affect the proximal and distal margins. In the remaining esophageal mucosa, high-grade squamous intra-epithelial neoplasia was observed, and no cancer was found in the lymph nodes.

Fifteen days post-surgery, a barium swallow fluoroscopy of the upper gastrointestinal tract found that the esophagogastric anastomosis was patent with no evidence of esophageal spasm (Figure 1F). No postoperative chemotherapy or radiotherapy was administered.

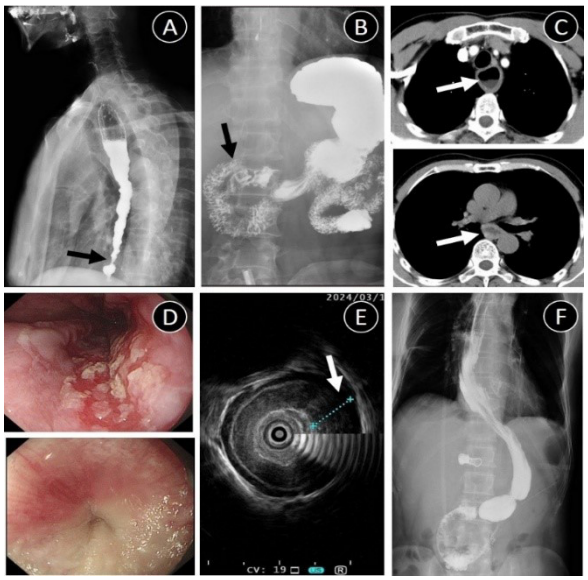

Figure 1: Patient's barium meal fluoroscopy, CT, and gastroscopy results. (A) Pre-operative barium meal fluoroscopy shows distal esophageal spasm (black arrow) and proximal esophageal dilation; **(B)** Barium meal fluoroscopy shows barium entering the small intestine after 4 minutes (black arrow); **(C)** CT shows proximal esophageal dilation and distal esophageal lumen narrowing (white arrow); **(D)** Gastroscopy shows opaque white changes in the esophageal mucosa, and distal esophageal narrowing; **(E)** Endoscopic ultrasound shows thickening of the esophageal wall (white arrow); **(F)** Post-operative barium meal fluoroscopy shows normal anastomosis with no esophageal spasm.

Discussion

Although primary esophageal spasm is a functional disorder, pathological conditions of the esophagus can also trigger esophageal spasm, necessitating careful differential diagnosis to avoid misdiagnosis and delays in treatment. The diagnostic methods for esophageal spasm include high-resolution esophageal manometry and functional lumen imaging probes, with differential diagnoses including cardiac chest pain, achalasia, and gastroesophageal reflux disease [3]. Left ventricular fibroma is reported cause esophageal spasms leading to recurrent chest pain accompanied by dysphagia in a patient [4]. The patient reported in this article presented with recurrent dysphagia and vomiting as the main clinical symptoms, without chest pain. An electrocardiogram and echocardiogram ruled out the possibility of heart disease. The CT scan of this patient showed abnormalities in the esophagus but did not reveal lesions in surrounding tissues or organs, while the upper gastrointestinal barium swallow demonstrated that the contrast agent could enter the stomach and small intestine in a relatively short time, indicating that the spasm

of the distal esophagus was transient rather than persistent. According to this case, there was no relaxation disorder of the lower esophageal sphincter, which is different from achalasia, where imaging typically shows dilation throughout the esophagus and narrowing of the lower esophagus and gastroesophageal junction, making contrast agent expulsion difficult and presenting a 'bird beak' appearance [5]. Although high-resolution esophageal manometry is the gold standard for diagnosing esophageal spasm, upper gastrointestinal imaging remains a more commonly used and convenient method for diagnosing esophageal disorders clinically. The upper gastrointestinal imaging in this patient indicated the presence of transient distal esophageal spasm, which is the pathophysiological reason for the dysphagia and symptoms of reflux and vomiting.

The pathophysiological mechanism of esophageal motility disorders is related to esophageal neuro-muscular lesions, which cause disturbances in esophageal movement and regulation. This is associated with local chronic inflammatory stimuli and immune dysregulation of the esophagus [5,6]. Multiple sclerosis, as an inflammatory disease, leads to dysfunction of pre-ganglionic and post-ganglionic nerve fibers in the intramural nerve plexus, increasing the risk of developing achalasia or esophageal spasm [7]. These chronic inflammatory stimuli and immune dysregulations also increase the risk of esophageal tumors. Studies have demonstrated that the risk of esophageal squamous cell carcinoma in patients with achalasia is 10 to 50 times higher than that of the general population, with cancerous lesions primarily occurring in the mid and lower segments of the esophagus; the average time from the onset of dysphagia symptoms to the diagnosis of esophageal squamous cell carcinoma is about 22 years [8,9]. Additionally, gastroesophageal reflux increases the risk of esophageal adenocarcinoma [10]. Therefore, although esophageal spasm is mostly a manifestation of esophageal functional disorders, for patients with a long duration of symptoms and those of older age, endoscopic examination should be performed to rule out organic lesions of the esophagus.

In this case, the endoscopic examination revealed significant esophageal mucosal lesions, with post-operative pathology indicating early squamous cell carcinoma and high-grade squamous intra-epithelial neoplasia, suggesting that the patient's esophageal spasm was not merely a functional disorder but was related to significant organic lesions of the esophagus. It is speculated that the patient's long-term inappropriate eating habits or reflux stimulation caused secondary lesions in the esophageal mucosa, thereby leading to carcinogenesis, while esophageal spasm was merely an external sign of the esophageal disease. Although acid reflux, heartburn, or retrosternal pain are typical clinical features of gastroesophageal reflux or esophageal mucosal lesions, some patients may not present prominent symptoms, leading to delayed diagnosis and treatment, allowing the lesions to progress insidiously.

Conclusion

This case suggests that for some patients presenting with swallowing difficulties, even if there is clinical evidence of esophageal spasm, a detailed examination through endoscopy and accurate tissue biopsy are still necessary to rule out the possibility of early esophageal tumors, in order to avoid delays in treatment that could affect prognosis.

References

1. Roman S, Hebbard G, Jung KW, Katz P, et al. Chicago classification update (v4.0): Technical review on diagnostic criteria for distal esophageal spasm. Neurogastroenterol Motil. 2021; 33: e14119.
2. Zaher EA, Patel P, Atia G, et al. Distal esophageal spasm: An updated review. Cureus 2023; 15: e41504.
3. Vasireddy AR, Leggett CL, Kamboj AK. Esophageal motility disorders: A concise review on classification, diagnosis, and management. Mayo Clin Proc. 2025; 100: 332-339.
4. O'Brien D, El-Andari R, Alomran M, et al. Left ventricular fibroma presenting as esophageal spasm: A case report. J Card Surg. 2022; 37: 3421-3425.
5. Provenza CG, Romanelli JR. Achalasia: diagnosis and management. Surg Clin North Am. 2025; 105: 143-158.
6. Nikaki K, Sawada A, Ustaoglu A, et al. Neuronal control of esophageal peristalsis and its role in esophageal disease. Curr Gastroenterol Rep. 2019; 21: 59.
7. Kim Y, Shibli F, Fu Y, et al. Multiple sclerosis is associated with achalasia and diffuse esophageal spasm. J Neurogastroenterol Motil. 2023; 29: 478-485.
8. Sandler RS, Nyrén O, Ekbom A, et al. The risk of esophageal cancer in patients with achalasia. A population-base d study. JAMA. 1995; 274: 1359-1362.
9. Tustumi F, Bernardo WM, da Rocha JRM, et al. Esophageal achalasia: a risk factor for carcinoma. A systematic review and meta-analysis. Dis Esophagus. 2017; 30: 1-8.
10. Lagergren J, Bergström R, Lindgren A, et al. Symptomatic gastroesophageal reflux as a risk factor for esophageal adenocarcinoma. N Engl J Med. 1999; 340:825-831.