Revista Española de Enfermedades Digestivas
The Spanish Journal of Gastroenterology

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DOI: 10.17235/reed.2025.11227/2025
Link: PubMed (Epub ahead of print)

Please cite this article as:
Pérez de la Serna y Bueno Julio, Ruiz de León San Juan Antonio. Is chest pain in achalasia always due to spasm?. Rev Esp Enferm Dig 2025. doi: 10.17235/reed.2025.11227/2025.

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Is chest pain in achalasia always due to spasm?

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Keywords: Achalasia. Chest pain. Esophageal spasm. Esophagogastric junction. Gas trapping. Incomplete myotomy. Esophageal wall distension.

Chest pain is a common symptom in patients with achalasia, particularly in younger individuals, those with a shorter duration of symptoms (1), and those diagnosed with type III achalasia according to the Chicago Classification.

The presence, frequency, and intensity of chest pain in achalasia vary widely, ranging from occasional discomfort to daily episodes (2). Pain can occur independently of eating, drinking cold liquids, or even during sleep. It is typically described as retrosternal, radiating to the neck, jaw, and ears, or as a transfixing pain extending to the back. In some studies, it has been compared to angina pectoris. Although prevalence rates between 36% and 84% have been reported (1,2), most series estimate it between 50% and 75% (1), and it is often refractory to treatment (3).

In certain cases, imaging techniques can identify the cause of the pain, and symptom improvement is generally observed when lower esophageal sphincter (LES) relaxation (Integrated Relaxation Pressure, IRP) normalizes. However, no clear correlation has been established between pain severity and patient characteristics such as age, sex,

body mass index, or duration of achalasia (1-10). Furthermore, no associations have been found between chest pain and radiological findings, esophageal manometry topography (11), post-treatment changes based on fundoplication type and myotomy length, or clinical scores. Some studies have linked preoperative chest pain during meals to an increased risk of postoperative pain, and a positive correlation has been reported between chest pain and the Eckardt score (2).

Despite effective treatment of achalasia through various modalities (pneumatic dilation, Heller myotomy, POEM, botulinum toxin), a significant proportion of patients continue to experience episodic chest pain for years. Pain relief after treatment varies widely, with reported rates ranging from 16% to 95%. After Heller myotomy, de novo chest pain has been observed in 8.3% of patients (12). Studies with short follow-up periods (6,11) tend to report better outcomes, whereas long-term studies indicate persistent chest pain. In series with a mean follow-up of four to six years, chest pain resolution occurs in only 20%–25% of patients, despite significant improvement in dysphagia and regurgitation (1,2).

The underlying mechanisms of chest pain in achalasia remain poorly understood. Pain occurs both in untreated patients and after surgical or endoscopic interventions. Following treatment, potential causes of pain include food retention, gas trapping, partial obstruction of the esophagogastric junction due to food impaction, gastroesophageal reflux, or persistent high-amplitude contractions in type III achalasia due to incomplete myotomy.

Possible pain mechanisms include:

- Stimulation of esophageal mechanoreceptors due to vigorous and uncontrolled contractions.
- Direct activation of chemoreceptors and nociceptors in esophageal mucosa.
- Esophageal hypersensitivity, similar to that observed in patients with functional chest pain or gastroesophageal reflux disease (13).
- Hyperactivity of the longitudinal muscle (14).

The hypothesis that repetitive, high-amplitude contractions cause pain by stimulating mechanoreceptors may apply in some cases (15-17). However, this mechanism does not appear to account for the majority of cases. Arguments against this include:

- In type I and II achalasia, contractions are either absent or simultaneous and of low amplitude, making high-amplitude contractions an unlikely cause of pain.
- Most chest pain episodes are not preceded by spastic or hypercontractile esophageal contractions (10).
- The disappearance of high-amplitude contractions does not always correlate with pain resolution.

Patients with chest pain generally exhibit LES pressures and esophageal body contraction amplitudes similar to those without pain (1). In achalasia, frequent and intense episodes of esophageal shortening have been observed, and sustained contraction of the longitudinal muscle has been linked to chest pain (18). This hypothesis is difficult to assess since sustained longitudinal muscle contraction may not be associated with increased esophageal pressure (19).

Once gastroesophageal reflux and incomplete myotomy have been ruled out, poor esophageal clearance is often the primary cause of pain. This is typically associated with some degree of dysphagia (10) and related to retention and/or distension (3). Esophageal retention leads to progressive accumulation of saliva and air, causing wall distension and triggering pain. In some cases, persistent pain has resolved after endoscopic removal of retained food.

Pain due to esophageal wall distension has been demonstrated through balloon/barostat distension tests (20-22). Impedance-pH monitoring has also identified pain episodes linked to air retention in the esophageal body, with relief achieved through supragastric belching, often by drinking water. Additionally, a case of gas-trapping-induced pain has been reported following Heller myotomy and Nissen fundoplication (23). The role of gas trapping has gained attention with the identification of retrograde cricopharyngeal dysfunction syndrome, in which inadequate upper esophageal sphincter function impairs belching, leading to chest pain (24).

In patients with altered pain perception and esophageal hypersensitivity, similar to functional chest pain, neuromodulators such as tricyclic antidepressants or serotonin reuptake inhibitors may be beneficial (25).

Therefore, attributing chest pain in achalasia solely to esophageal spasm offers a limited perspective on the broad spectrum of possible causes. Once cardiovascular pathology has been ruled out, identifying the cause of pain should be based on the patient's clinical presentation, particularly in those who have already received treatment.

Follow-up after any therapeutic intervention in achalasia patients should include a clinical evaluation between one and three months post-treatment, along with timely complementary tests based on symptoms:

1. Detailed symptom assessment, primarily using the Eckardt score. It is crucial to evaluate pain intensity and duration, dietary restrictions due to symptoms, triggering factors, previously attempted treatments, associated conditions, and ongoing therapies.
2. High-resolution esophageal manometry, preferably with impedance measurement. This test provides information on changes in motor activity, the esophagogastric junction, and esophageal clearance post-treatment.
3. 24-hour esophageal pH monitoring with impedance, which helps assess reflux presence or persistent esophageal retention in the patient's new clinical state.
4. Barium radiographic studies, offering insights into the progression of esophageal dilation, esophageal morphology, esophagogastric junction function, contrast passage into the stomach, and esophageal clearance rate compared to previous studies.
5. Endoscopic examination, which identifies potential structural mucosal alterations and evaluates resistance at the esophagogastric junction. This procedure is mandatory if impaction is suspected.
6. Impedance planimetry (EndoFlip®), a useful tool for measuring the diameter and compliance of the cardia, as well as detecting changes from previous assessments.

By integrating clinical findings with these diagnostic tests, a more precise understanding of the pain's underlying mechanism can be achieved. This enables a tailored therapeutic approach, selecting from various medical treatments (calcium channel blockers, nitrates, phosphodiesterase type 5 inhibitors, proton pump inhibitors, pain modulators), as well as surgical or endoscopic options. Such an

approach minimizes unnecessary or ineffective treatments over prolonged periods.

In most cases, sufficient clinical improvement is achieved, allowing patients to regain their quality of life and improve their dietary habits and nutrition. However, it is important to remember that the absence of peristalsis and the resulting difficulty in esophageal clearance—especially when meals are consumed too quickly, without sufficient liquid intake, or include large, dry solid food pieces—can lead to temporary retention or impaction. These episodes may resolve spontaneously through air expulsion (belching) or, in some cases, require endoscopic intervention.

References

1. Eckardt VF, Stauf B, Bernhard G. Chest pain in achalasia: Patient characteristics and clinical course. Gastroenterology. 1999; 116:1300–1304.

2. Olvera-Prado H, Peralta-Figueroa J, Narváez-Chávez S, et al. Predictive factors associated with the persistence of chest pain in post-laparoscopic myotomy and fundoplication in patients with achalasia. Front Med (Lausanne). 2022 Oct 14;9:941581. doi: 10.3389/fmed.2022.941581.

3. Sasaki A, Obuchi T, Nakajima J, et al. Laparoscopic Heller myotomy with Dor fundoplication for achalasia: long-term outcomes and effect on chest pain. Dis Esophagus. 2010 May;23(4):284-9. doi: 10.1111/j.1442-2050.2009.01032.x.

4. Papo M, Mearin F, Castro A, et al. Chest pain and reappearance of esophageal peristalsis in treated achalasia. Scand J Gastroenterol. 1997;32:1190–4. doi: 10.3109/00365529709028145.

5. Nylander DL. Chest pain in achalasia is an age-dependent phenomenon. Gastroenterology. 1999; 117:1259. doi: 10.1016/S0016-5085(99)70429-4.

6. Perretta S, Fisichella P M, Galvani C, et al. Achalasia and chest pain: effect of laparoscopic Heller myotomy. J Gastrointest Surg. 2003; 7: 595–8.

7. Omura N, Kashiwagi H, Tsuboi K, et al. Therapeutic effects of a laparoscopic heller myotomy and Dor fundoplication on the chest pain associated with achalasia. Surg Today. 2006; 36:235–40. doi: 10.1007/s00595-005-3122-4.

8. Arain MA, Peters JH, Tamhankar AP, et al. Preoperative lower esophageal sphincter pressure affects outcome of laparoscopic esophageal myotomy for achalasia.

J Gastrointest Surg. 2004; 8:328– 34. doi: 10.1016/j.gassur.2003.09.011

9. Omura N, Kashiwagi H, Yano F, et al. Effect of laparoscopic esophagomyotomy on chest pain associated with achalasia and prediction of therapeutic outcomes. Surg Endosc. 2011; 25:1048–53. doi: 10.1007/s00464-010-1314-5.

10. Wuller C, Bessell JR, Watson DI. Chest pain before and after laparoscopic cardiomyotomy for achalasia. ANZ J Surg. 2011; 81:590–594. doi: 10.1111/j.1445-2197.2010.05577.x.

11. Nicodème F, de Ruigh A, Xiao Y, et al. A comparison of symptom severity and bolus retention with Chicago classification esophageal pressure topography metrics in patients with achalasia. Clin Gastroenterol Hepatol. 2013 Feb;11(2):131-7; quiz e15. doi: 10.1016/j.cgh.2012.10.015..

12. Hatafuku T, Maki T, Thal A P. Fundic patch operation in the treatment of advanced achalasia of the esophagus. Surg Gynecol Obstet. 1972; 134: 617–24.

13. Savarino EV, Salvador R, Ghisa M, et al. Research gap in esophageal achalasia: a narrative review. Dis Esophagus. 2024 Jul 31;37(8):doae024. doi: 10.1093/dote/doae024. PMID: 38525929.

14. Wolff R R, Defilippi CC. Nuevas técnicas para el estudio de la función esofágica. Gastroenterol. latinoam 2008; 19 (3): 228-233.

15. Sanderson DR, Ellis FH Jr, Schlegel JF, et al. Syndrome of vigorous achalasia: clinical and physiologic observations. Dis Chest. 1967; 52:508–517.

16. Bondi JL, Godwin DH, Garrett JM. “Vigorous’’ achalasia. Its clinical interpretation and significance. Am J Gastroenterol. 1972; 58:145–154.

17. Bramer S, Ladell A, Glatzel H, et al. Medical management of painful achalasia: a patient-driven systematic review. Dis Esophagus. 2024 Apr 27;37(5): doae005. doi: 10.1093/dote/doae005.

18. Holloway R. Esophageal Ultrasonography: A New View on Esophageal Motility. Am J Gastroenterol. 2007; 102: 146-148.

19. Balaban D H, Yamamoto Y, Liu J, et al. Sustained esophageal contraction: a marker of esophageal chest pain identified by intraluminal ultrasonography. Gastroenterology. 1999; 116: 29-37. doi: 10.1016/s0016-5085(99)70225-8.

20. Barish CF, Castell DO, Richter JE. Graded esophageal balloon distention. A new provocative test for noncardiac chest pain. Dig Dis Sci. 1986 Dec;31(12):1292-8.

21. Remes-Troche JM, Attaluri A, Chahal P, et al. Barostat or dynamic balloon distention test: which technique is best suited for esophageal sensory testing? Dis Esophagus. 2012 Sep-Oct;25(7):584-9.

22. Carlson DA, Kahrilas PJ, Pandolfino JE. Repetitive antegrade contractions on high-resolution manometry: A physiologic pattern related to sustained esophageal distention in Abelchia. Neurogastroenterol Motil. 2025 Feb;37(2):e14934. doi: 10.1111/nmo.14934.

23. Alcala-Gonzalez LG, Aguilar-Cayuelas A, Quiroga S, et al. Recurrent symptoms after achalasia treatment: The value of impedance analysis. United European Gastroenterol J. 2024 Oct 28. doi: 10.1002/ueg2.12692.

24. Bastian RW, Smithson ML. Inability to belch and associated symptoms due to retrograde cricopharyngeus dysfunction: diagnosis and treatment. OTO Open. 2019; 15;3(1):2473974X19834553. doi: 10.1177/2473974X19834553.

25. Aggarwal M, McMichael J, Murthy SC, et al. Neuromodulators are effective for treatment of spastic chest pain after heller myotomy for achalasia. J Neurogastroenterol Motil. 2021 Oct 30;27(4):655-656. doi: 10.5056/jnm21113.