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REVIEW

Modern insights into the pathophysiology and treatment of pseudoachalasia

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Abstract

Background Secondary achalasia or pseudoachalasia is a clinical presentation undistinguishable from achalasia in terms of symptoms, manometric, and radiographic findings, but associated with different and identifiable underlying causes.

Methods A literature review was conducted on the PubMed database restricting results to the English language. Key terms used were "achalasia-like" with 63 results, "secondary achalasia" with 69 results, and "pseudoachalasia" with 141 results. References of the retrieved papers were also manually reviewed.

Results Etiology, diagnosis, and treatment were reviewed.

Conclusions Pseudoachalasia is a rare disease. Most available evidence regarding this condition is based on case reports or small retrospective series. There are different causes but all culminating in outflow obstruction. Clinical presentation and image and functional tests overlap with primary achalasia or are inaccurate, thus the identification of secondary achalasia can be delayed. Inadequate diagnosis leads to futile therapies and could worsen prognosis, especially in neoplastic disease. Routine screening is not justifiable; good clinical judgment still remains the best tool. Therapy should be aimed at etiology. Even though Heller's myotomy brings the best results in non-malignant cases, good clinical judgment still remains the best tool as well.

Keywords Esophagus · Esophageal motility disorders · Esophageal achalasia · Bariatric surgery · Stomach neoplasms · Pseudoachalasia

Introduction

The modern concept of achalasia, and this term as a matter of fact, is credited to Lendrum [1] when associated the disease to an incomplete relaxation of the lower esophageal (LES) sphincter (LES) in 1937. Chicago Classification in its 4.0 version in 2021 [2], the most accepted classification for esophageal motility disorders based on high-resolution manometry, defines achalasia by an abnormal relaxation of the LES, as defined manometrically as an elevated median integrated relaxation pressure and total failed peristalsis. The Classification also categorized achalasia into three different subtypes based on esophageal pressurization during swallows: Type I is characterized by no pressurization, Type II by panesophageal pressurization occurring in ≥ 20% swallows, and Type III by premature/spastic contractions in ≥ 20% swallows.

Primary achalasia is understood as an idiopathic disease with loss of esophageal myenteric ganglia [3, 4]. Some patients, however, develop a clinical presentation undistinguishable from achalasia in terms of symptoms, manometric, and radiographic findings, but associated with identifiable underlying causes. This condition is known as secondary achalasia or pseudoachalasia, first described by Olgivie in 1947 [5]. Different etiologies for pseudoachalasia were described: neoplastic (mainly cardia cancer, but also malignant mesothelioma, breast cancer, sarcoma, and other tumors) [5–9], post-Nissen fundoplication [10], bariatric surgery [4, 11, 12], amyloidosis [13], etc.

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Very interestingly, the interest in pseudoachalasia oscillated along time. Initial studies were numerous with a high concern for the disease, then for some time, this theme was left aside, but with the ascension of bariatric surgery, pseudoachalasia returned to the focus of studies again to decrease once more after changes in the trends for bariatric techniques. This pendulum of interest brought some insights into the pathophysiology and treatment of pseudoachalasia.

Methods

A literature review was conducted on the PubMed database restricting results to the English language. Key terms used were "achalasia-like" with 63 results, "secondary achalasia" with 69 results, and "pseudoachalasia" with 141 results. References of the retrieved papers were also manually reviewed.

Etiology

More than 25 different causes for pseudoachalasia have been described [14, 15]. Most causes are linked to esophagogastric junction outflow obstruction or neuronal damage. In achalasia series, pseudoachalasia comprises 1.5–5% of all cases [16–18].

Neoplastic obstruction

Tumors at the cardia were the first and most described causes for pseudoachalasia [16, 17]. It is the most common cause in compilations of literature series, comprising 70% of the cases, with a preponderance of cardia cancer in 50% and other malignancies in 20% [16]. It is virtually impossible to estimate the number of patients with a tumor that presents with pseudoachalasia since esophageal manometry and barium swallow will not be part of the workup of these individuals and symptoms overlap.

Tumoral invasion, intrinsic or extrinsic, could limit esophagogastric junction outflow, leading to peristalsis deterioration. A tumor may also invade the esophageal submucosa and destroy the esophageal myenteric plexus precluding adequate LES relaxation [14, 15].

Paraneoplastic syndrome

Cancer may lead to pseudoachalasia beyond the mechanical hypotheses. Paraneoplastic syndrome may encompass secondary achalasia. Pathophysiology is linked to the release by the tumor of neurotoxic substances [19], antibodies, or myenteric plexus infiltration by lymphocytes and plasma cells leading to neuronal degeneration [20].

The most common tumors associated with pseudoachalasia are small-cell lung carcinoma; however, other neoplasms are also associated, such as anaplastic lung adenocarcinoma, lymphoma, and ovarian papillary serous adenocarcinoma [18, 20–22].

Postoperative

Pseudoachalasia has been described after different operations at the cardia or proximal stomach. It may be diagnosed very precociously [23], with an average of 4 months after the index operation [24], or very late [25] although one may question if very later an idiopathic achalasia developed independent of the operation—metachronous primary achalasia [24]. It must be emphasized that some authors described a reversible esophageal ileus following antireflux surgery with 50% of aperistalsis on a postoperative day 1 [26]. Thus, diagnosis of pseudoachalasia very early or very late in evolution must be cautiously done.

Pseudoachalasia may occur in about 3% of the cases after a Nissen fundoplication [24]. One case after magnetic ring sphincter augmentation has been reported [27]. Adjustable gastric banding, fortunately falling into disuse, is an operation with a high risk of postoperative pseudoachalasia [28–35]. Pseudoachalasia is rare after Roux-en-Y gastric bypass and sleeve gastrectomy [36]. Again, the incidence of pseudoachalasia post-bariatric surgery may be lower than reported since cases may correspond to classic achalasia as esophageal manometry is not recommended in the preoperative work-up of these patients [37], and achalasia in the morbidly obese population may be asymptomatic [38, 39].

One hypothesis proposed for pathophysiology is vagal nerve injury during the operation with LES denervation [24]. There are, however, strong arguments against this proposal. First, there are some case reports [40–42], but pseudoachalasia is rare following surgical vagotomy, an operation extensively performed during peptic ulcer surgery. Also, periesophageal fibrosis cannot be excluded as a cause since there are cases after highly selective vagotomy that excludes cardiac denervation [43]. Moreover, vagal damage is more common after reoperations at the esophagogastric junction [44] and paraesophageal hernia repair [45], but we could not find cases reported after these operations.

Another hypothesis proposed for pathophysiology is chronic outflow obstruction. Experiments in cats show an achalasia-like motility pattern when the distal esophagus of cats is partially obstructed by a band [46, 47]. A mechanical obstruction or anatomic failure of the fundoplication, however, cannot be demonstrated in most cases of post-Nissen pseudoachalasia [24]. We can parallel this finding with a series of conversion from total to partial fundoplication. Most of the patients in these series do not have a mechanical obstruction or anatomic failure and are unresponsive

to endoscopic dilatation [27, 48]. Total fundoplication is probably just an obstacle to the esophageal peristalsis in this small percentage of individuals. Very interestingly, we could not find cases reported after a partial fundoplication.

Parallel with achalasia

Pseudoachalasia may give some insights into achalasia pathophysiology and vice versa. Traditionally, the combination of LES dysfunctional relaxation and aperistalsis guided the diagnosis. Advances in esophageal manometry improved the understanding and classification of achalasia. New variants of achalasia were proposed, with incidences that are not insignificant [2, 49]. These variants question the all or nothing at all concept for aperistalsis but, more importantly, suggest that LES dysfunction may be the initial cause, similar to pseudoachalasia, at least in postoperative cases. Thus, esophagogastric junction outflow obstruction, a disease per se according to the Chicago Classification, may be an achalasia variant or early form [50].

Apart from the LES, dysmotility exclusively of the esophageal body such as esophageal spasm can degenerate in achalasia. This deterioration occurs independently of LES dysfunction [51]. This may parallel some cases of pseudoachalasia with disease limited to the esophageal body sparing the LES such as aortic aneurysm compressing the esophagus [52]. These cases, however, are anecdotal cases, most without a previous workup to exclude the epiphenomenon of a conventional achalasia misdiagnosed until the presentation of the second disease [52].

Diagnosis

We mentioned before that pseudoachalasia has a clinical presentation undistinguishable from achalasia in terms of symptoms, manometric, and radiographic findings. For postoperative cases, the correct diagnosis demands preoperative demonstration of peristalsis and complete relaxation of the LES [24] (Fig. 1) since 45% of patients with untreated achalasia had been prescribed acid-suppressing medications on the assumption that gastroesophageal reflux disease was the cause of the symptoms [53], and misdiagnose of achalasia in patients that undergo antireflux operations may reach 4% [54].

Some claim that pseudoachalasia symptoms may be of shorter duration with more pronounced weight loss and older age [8, 14, 15, 17, 55], but this is an occurrence usually in neoplastic pseudoachalasia due to cancer and not due to esophageal disease.

Some authors tried to find distinguishable manometric traits, such as compartmentalized pressurization or Chicago type [55, 56]. These findings, however, overlap with achalasia and are not extremely common or pathognomonic of pseudoachalasia.

Image tests (upper endoscopy, barium swallow, endoscopic ultrasound, and tomography) may obviously demonstrate the presence of a tumor as the cause, although some cases are not diagnostic [8, 18, 57, 58]. A feature in barium swallow that some authors point out is a longer segment of distal narrowing with a less marked esophageal dilatation [57–59].

Upper endoscopy may fail to reveal neoplastic infiltration in up to 60% of the cases and tomography in 20–100% [60–63].

Interestingly, fluoroscopy magnetic resonance seems to be a promising method [64].

Treatment

Since several etiologies could induce pseudoachalasia, management of this condition is variable.

Therapy is directed towards oncologic treatment in cases secondary to neoplasms. Symptoms may resolve with tumor remission and in some cases, peristalsis is restored [65–67]. Achalasia treatment may be used in cases of unresectable tumors in patients with severe symptoms. Outcomes of these procedures are shown in Table 1.

Precocious relief of reversible esophagogastric junction obstruction, if possible, may resolve the problem and even bring motility back to normal. This has been reported in cases of deflation or removal of adjustable gastric banding [28, 30, 31, 35] or drainage of a pseudocyst compressing the cardia [78]. Relief cannot be obtained if obstruction is chronic (over 8 years) [29].

Endoscopic forceful dilatation of the cardia is probably the most used treatment irrespective of etiology (Table 1). In our personal experience and others, dilatation after Nissen fundoplication is inefficient, and myotomy is necessary [68, 70]. Some believe that dysmotility may be reversible depending on the period for symptoms onset and dilatation with eventual anatomic repair is sufficient if symptoms occur early after a fundoplication [23] Conversion to partial fundoplication seldom solves symptoms [24, 71] (Table 1).

Heller's myotomy and partial fundoplication always show good outcomes (Table 1), except perhaps with certain underlying conditions such as the coexistence of diffuse leiomyomatosis [79].

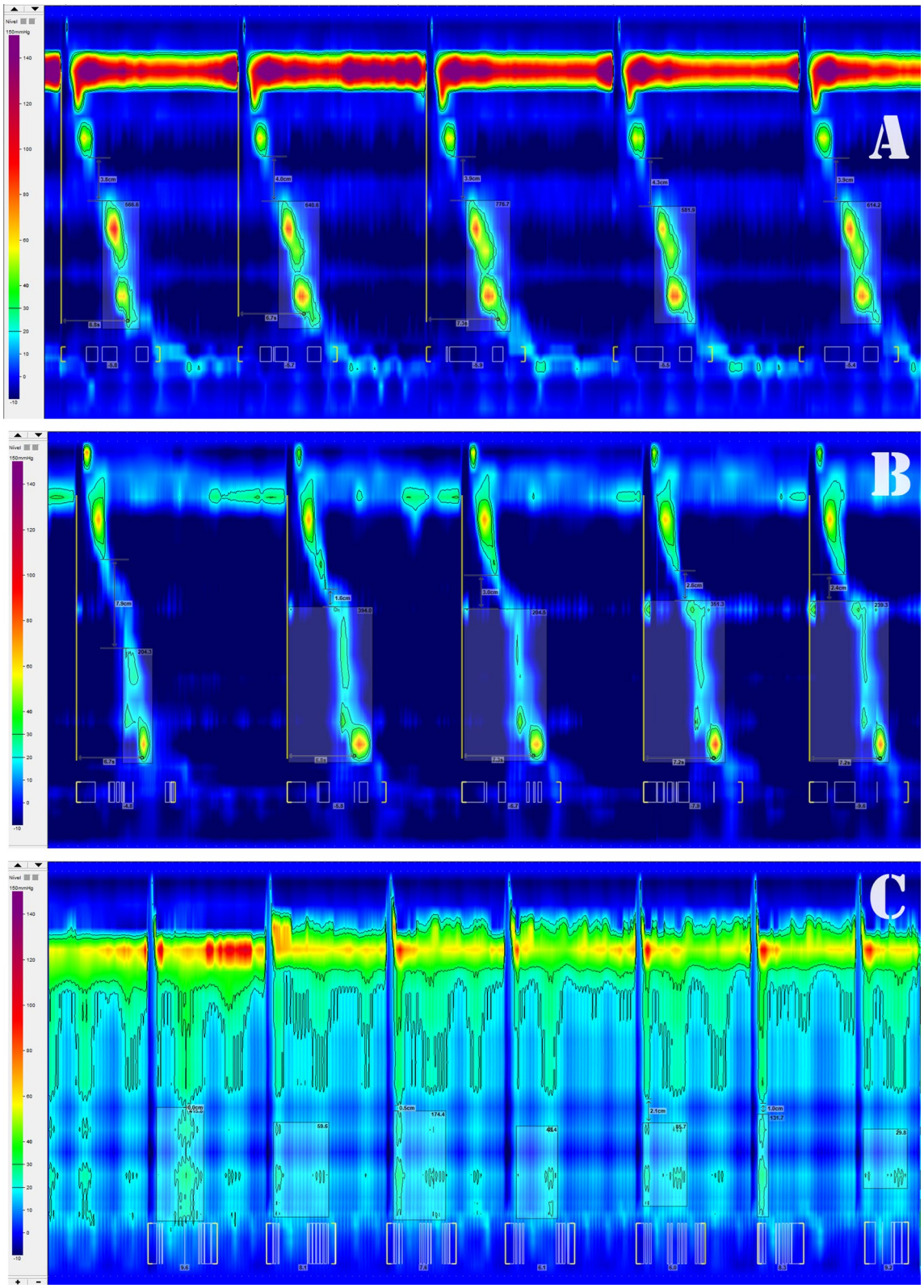

Fig. 1 High-resolution manometry tracings for pseudoachalasia after Nissen fundoplication. Esophageal motility is normal before operation (**A**), deteriorated to distal esophageal spams in 30 days after the operation (**B**), and finally aperistalsis and lack of relaxation of the lower esophageal sphincter 4 months after the operation (**C**)

Table 1 Pseudoachalasia outcomes according to etiology and treatment

Etiology	Treatment	Outcome (*n*/total, % excellent/good results)	References
Post-Nissen fundoplication	Heller's myotomy and partial fundoplication	7/7; 100%	[10, 24, 68, 69]; 2 personal cases
Post-Nissen fundoplication	Endoscopic dilatation	6/13; 46%	[10, 24, 68, 70]; 2 personal cases
Post-Nissen fundoplication	Conversion to partial fundoplication	0/2; 0%	[24, 71]
Post-Nissen fundoplication	Botulinum toxin injection	2/3; 67%	[24]
Post magnetic sphincter augmentation	Endoscopic dilatation	1/1; 100%	[27]
Post Roux-en-Y gastric bypass	Heller's myotomy and partial fundoplication	2/3; 67%	[11, 72, 73]
Post Roux-en-Y gastric bypass	Peroral endoscopic myotomy	2/3; 67%	[74]
Post sleeve gastrectomy	Peroral endoscopic myotomy	2/3; 67%	[74]
Post sleeve gastrectomy	Heller's myotomy and partial fundoplication	1/1; 100%	[75]
Malignant	Botulinum toxin injection	2/7; 29%	[66, 76, 77]

Peroral endoscopic myotomy (POEM) has been used after bariatric operations in a few cases, usually after the failure of other options (Table 1).

Conclusion

Pseudoachalasia is a rare condition. Most available evidence regarding this condition is based on case reports or small retrospective series. There are different causes but all culminating in outflow obstruction. Clinical presentation and image and functional tests overlap with primary achalasia or are inaccurate, thus the identification of secondary achalasia can be delayed. Inadequate diagnosis leads to futile therapies and could worsen prognosis, especially in neoplastic disease. Routine screening is not justifiable; good clinical judgment still remains the best tool [18]. Therapy should be aimed at etiology. Even though Heller's myotomy brings the best results in non-malignant cases, again, good clinical judgment still remains the best tool.

Authors' contributions LYKZ: acquisition of data, analysis and interpretation of data, drafting the article, and final approval of the version to be published. FAMH: conception and design, acquisition of data, analysis and interpretation of data, drafting the article, and final approval of the version to be published. VV: conception and design, review for intellectual content, and final approval of the version to be published. MGP: conception and design, review for intellectual content, and final approval of the version to be published.

Data availability No datasets were generated or analysed during the current study.

Declarations

Competing interests The authors declare no competing interests.

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