CASE REPORT

Pseudoachalasia due to hepatic compression in an adolescent: A case of enlarged left and accessory liver lobes

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ABSTRACT

Background: Achalasia cardia is a rare oesophageal motility disorder characterized by impaired lower oesophageal sphincter (LES) relaxation and absent peristalsis. While typically idiopathic, rare cases arise from external compression.

Case Presentation: We report an 18-year-old male with a two-year history of progressive dysphagia to liquids and solids, chest pain, regurgitation, and weight loss. Barium swallow revealed oesophageal dilation with tapering ("bird's beak" sign). Definitive diagnosis was made intraoperatively, which revealed an enlarged left liver lobe and an accessory lobe compressing the distal oesophagus.

Intervention: A modified Heller's oesophagocardiomyotomy was performed via laparotomy.

Conclusion: This case highlights a rare cause of pseudoachalasia due to hepatic compression and underscores the importance of considering extrinsic causes in atypical achalasia, especially in resource-limited settings.

INTRODUCTION

Achalasia cardia is an uncommon oesophageal motor disorder marked by impaired lower oesophageal sphincter (LES) relaxation and absence of peristalsis, resulting in dysphagia, chest pain, regurgitation, and weight loss.[1, 2] It radiologically presents with oesophageal narrowing and a characteristic "bird's beak" appearance at the gastroesophageal junction (GEJ).[3] Aetiologies include idiopathic causes, Chagas disease, and pseudoachalasia. Pseudoachalasia is an achalasia-like syndrome secondary to malignancy or mechanical obstruction.[4, 5] Although rare, benign extrinsic compression by hepatic anomalies such as hypertrophied or accessory liver lobes has been implicated.[6, 7] These are often under-recognized due to limited imaging capabilities in low-resource settings.

This report presents a rare case of pseudoachalasia from dual hepatic compression, emphasizing the importance of considering hepatic causes in young patients with atypical achalasia presentations.

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CASE PRESENTATION

An 18-year-old male first year college student, presented with a two-year history of progressive dysphagia, initially to liquids, then solids. He reported chest pain, odynophagia, regurgitation of undigested meals, postprandial vomiting, chronic cough, low-grade fever, generalized weakness, and unintentional weight loss (approximately 10 kg over 12 months). His schooling was interrupted due to feeding difficulties.

Physical examination revealed a thin young man with tachypnoea (22 breaths/min), a BMI of 19 kg/m², and a scaphoid abdomen. Abdominal palpation showed no organomegaly, and no lymphadenopathy was detected.

Timeline of Events

Date	Clinical Event
~2 years before presentation	Onset of dysphagia to liquids
~18 months before	Dysphagia progressed to solids
~6 months before	Onset of chest pain, regurgitation, weight loss
Presentation	Barium swallow performed, suggestive of achalasia
Surgery (April 2025)	Heller's oesophagocardiomyotomy performed via laparotomy
Follow-up (May–July 2025)	Symptom resolution and improved feeding

Diagnostic Assessment

Barium swallow showed widened mediastinum, absent gastric bubble, and "bird's beak" appearance with proximal oesophageal dilation; classic for achalasia cardia (Figure 1) [3].

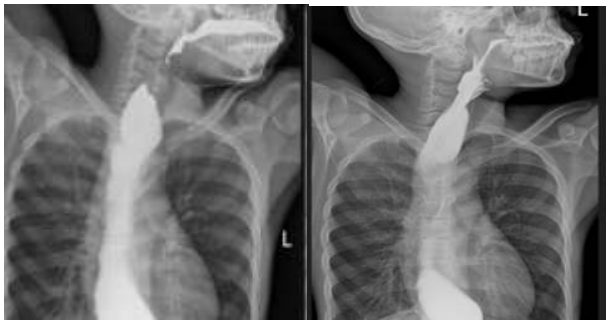

Figure 1: Barium swallow showing classic "bird's beak" sign (arrow), absent gastric bubble, and oesophageal dilation

Preoperative CT or MRI was unavailable due to resource constraints. Serologic and stool testing excluded parasitic infections (including *T. cruzi*), and no clinical or historical evidence of malignancy was identified.

Therapeutic Intervention

The patient underwent a modified Heller's oesophagocardiomyotomy via laparotomy. This was preferred over laparoscopy due to limited resources. Intraoperative findings included:

- An enlarged left hepatic lobe
- A well-defined accessory lobe compressing the abdominal oesophagus (Figure 2)
- An anatomically normal-appearing abdominal oesophagus

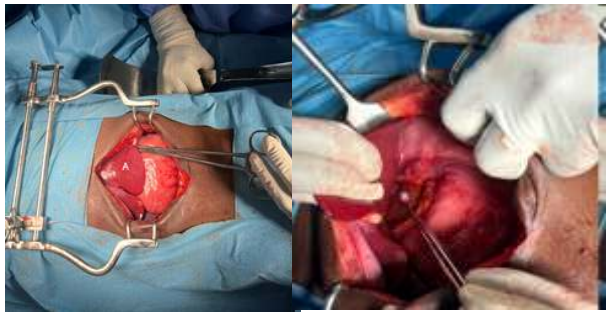

Figure 2: Intraoperative view showing: A – Enlarged left hepatic lobe, B – Accessory hepatic lobe compressing oesophagus

No liver resection was done as the lobes were non-pathologic and decompression achieved symptom relief. He was discharged on postoperative day 6 and placed on a soft diet.

Follow-Up and Outcomes

At 4-week follow-up, the patient reported full symptom resolution, resumed normal feeding and social activities, and had gained 5 kg. He resumed schooling and reported improved psychosocial well-being. Postoperative imaging and manometry were not done due to resource constraints.

Patient Perspective

"I was embarrassed to eat in public because I would regurgitate food. Since the surgery, I can now enjoy meals with my friends without fear. I'm back in school and happy again."

DISCUSSION

Pseudoachalasia, an important yet underrecognized mimic of idiopathic achalasia, typically arises from malignancy or benign extrinsic compression.[4, 5, 8] In this case, dual hepatic anomalies (an enlarged left lobe and accessory lobe) compressed the distal oesophagus, creating functional obstruction indistinguishable from primary achalasia. While benign liver anomalies are rarely implicated in pseudoachalasia,[6, 7, 9] this represents the first adolescent case involving simultaneous compression by two distinct hepatic lobes.

Diagnostic challenges persist, as barium swallow and manometry often fail to differentiate idiopathic from secondary causes.[10, 11] Although cross-sectional imaging (CT/MRI) or endoscopic ultrasound is essential for detecting compressive lesions.[12, 13] These modalities remain frequently inaccessible in resource-limited settings. Our experience demonstrates that intraoperative exploration can guide diagnosis when advanced imaging is unavailable. Crucially, while surgery remains definitive therapy, outcomes depend on aetiology: extrinsic decompression often achieves faster resolution than idiopathic disease, where irreversible neural damage portends prolonged recovery.

This case demonstrates two notable strengths: first, it represents the novel presentation of dual hepatic compression causing pseudoachalasia in a young patient; second, complete symptom resolution was achieved through decompression alone, avoiding hepatic resection. Important limitations include the absence of preoperative cross-sectional imaging (CT/MRI), lack of histopathological confirmation of the accessory lobe, and unavailable postoperative manometry to assess functional recovery. These constraints underscore key clinical implications: clinicians should consider pseudoachalasia in young patients with atypical achalasia presentations, include hepatic anomalies in the differential diagnosis, and maintain high intraoperative vigilance when advanced imaging is inaccessible in resource-limited settings.

CONCLUSION

This case illustrates a rare but significant aetiology of pseudoachalasia due to hepatic compression by both an enlarged left and an accessory liver lobe. It reinforces the need for comprehensive evaluation, including imaging and intraoperative assessment in cases of atypical achalasia, particularly in young patients. Early recognition of non-neurogenic causes allows for timely and effective surgical management.

INFORMED CONSENT

Written informed consent was obtained from the patient for publication of this case report and accompanying images.

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COMPETING INTERESTS

No competing interests declared.

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Data Availability

Radiographs and operative details are available upon reasonable request from the Cardiothoracic Surgery Unit, Federal Medical Centre Ebute Metta, Lagos, Nigeria.

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