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# Complex Constellation of Gastroptosis, Achalasia, and Hypertophic Gastritis: A Case Report

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**Keywords**
*achalasia, case report, gastroptosis, hypertorphic gastritis, hypoglycemia,*

**Abstract**
*The concurrent presentation of multiple upper gastrointestinal (GI) disorders is a rare clinical entity that poses significant diagnostic and therapeutic challenges. We present an unprecedented case of coexisting achalasia, gastroptosis, hypertrophic gastritis, and hyperacidity, which culminated in a life-threatening metabolic emergency. Case presentation: A 63-year-old female with a history of chronic dyspepsia presented with progressive nausea, vomiting, significant weight loss, and an acute episode of severe hypoglycemia (random blood glucose 52 mg/dL). A barium contrast study (Oesophagus-Maag-Duodenum) was the key diagnostic investigation. Radiological findings revealed a dilated esophagus with a distal "rat tail" sign characteristic of achalasia. The same study demonstrated severe inferior displacement of the stomach into the pelvic cavity, confirming gastroptosis, and markedly thickened gastric mucosal folds, indicative of hypertrophic gastritis. Conclusion: This case illustrates a unique constellation of anatomical, motor, and inflammatory GI pathologies. The severe hypoglycemia is attributed to a "double-delay" mechanism, where esophageal stasis from achalasia combined with delayed gastric emptying from gastroptosis led to erratic nutrient delivery and a dysregulated, exaggerated insulin response. This report highlights the critical role of comprehensive radiological evaluation in diagnosing complex, overlapping GI conditions and underscores the importance of considering multi-pathology interplay when faced with atypical clinical manifestations.*

## INTRODUCTION

Gastrointestinal (GI) disorders often present with a diverse clinical spectrum, but the combination of several structural and functional abnormalities in one patient is a rare phenomenon. Patients with overlapping chronic upper GI symptoms have always been a challenge for clinicians concerning diagnosis and therapeutic approaches for management. They differ from a single condition, which rarely requires complex management. This report presents an extremely rare case of the convergence of four different diagnoses in one patient: gastroptosis, achalasia, hypertrophic gastritis, and hyperacidity, which collectively manifested as severe, life-threatening hypoglycemia.

Gastroptosis is a rare condition characterized by an abnormal lowering of the stomach position, which can cause chronic dyspepsia symptoms and is difficult to recognize early (Bestari et al., 2022). Achalasia has a low prevalence (approximately 1 per 100,000 population), characterized by failure of the lower esophageal sphincter to relax and loss of

esophageal peristalsis, which contributes to dysphagia, weight loss, and malnutrition (Katsumata et al., 2023). Meanwhile, hypertrophic gastritis, including Ménétrier's disease, is a very rare premalignant disorder with uncertain incidence, characterized by enlarged gastric mucosal folds, impaired acid secretion, and protein loss (Gomes, 2021). Hyperacidity is a common comorbidity, frequently associated with gastroesophageal reflux disease (GERD) (Kasugai & Ogasawara, 2024).

Pathophysiologically, this combination of conditions reflects the interaction of anatomical, functional, and secretory disorders. Gastroptosis can cause gastric stasis and reflux, which exacerbate dyspeptic symptoms (Staszewska et al., 2023). In achalasia, failure of neuronal inhibition in the myenteric plexus results in aperistalsis and functional obstruction in the esophagus (Savarino et al., 2022). Hypertrophic gastritis involves activation of the epidermal growth factor receptor (EGFR) pathway due to increased TGF-α, which stimulates excessive proliferation of gastric mucosal cells (Huh et al., 2016). Hyperacidity itself exacerbates mucosal irritation and increases the risk of ulcer complications, worsening the patient's malnutrition. Its pathophysiology is complex, involving failure of the anti-reflux barrier at the esophagogastric junction, transient lower esophageal sphincter relaxation, and the presence of a highly acidic post-prandial "acid pocket" near the cardia (Kasugai & Ogasawara, 2024).

The coexistence of these multiple underlying pathologies, particularly involving anatomical, motor, and inflammatory abnormalities, appears as a complicated clinical puzzle. The case highlights the complexity of diagnoses, the interaction of the conditions, and its implications for the therapeutic approach. The purpose of this case report is to describe the clinical manifestations and unusual diagnostic findings, analyze the pathophysiological mechanisms of the interaction between these anatomical, motor, and inflammatory disorders, and identify effective management strategies for complex multi-pathological conditions. In practical terms, this report provides clinical benefits by presenting a comprehensive diagnostic approach and management strategies that can be used as a guide for clinicians in managing complex and atypical gastrointestinal cases. Additionally, theoretically, this case enriches the understanding of pathophysiological interactions that can lead to systemic complications such as hypoglycemia, while serving as a valuable educational resource for medical personnel in recognizing and dealing with the presentation of diseases involving multi-morbidity.

## RESEARCH METHOD

This study employed a descriptive case report design to present and analyze the unique clinical presentation, diagnostic findings, and management of a patient with multiple concurrent upper gastrointestinal disorders. This approach allows for an in-depth exploration of rare or complex clinical conditions that are not commonly encountered in routine practice.

### Case Presentation

A 63-year-old female presented to the emergency department with complaints of upper abdominal pain, nausea, and post-meal vomiting that had been ongoing for several months. Her symptoms had progressively worsened, accompanied by a significant loss of appetite and a

weight loss of approximately 10 kg. Prior to the admission, the patient has vomitted more than ten times in a span of 24 hours. On the day of admission, the patient experienced an episode of acute weakness, dizziness, and cold sweats. A random blood glucose (RBG) test in the emergency department showed a level of 52 mg/dL. The patient had a history of hypertension, diabetes mellitus, and chronic GERD/dyspepsia.

Upon arrival at the emergency department, the patient appeared weak and pale. Her vital signs on admission were: temperature 36.0°C, heart rate 76 beats/minute, respiratory rate 22 breaths/minute, and blood pressure 141/87 mmHg. Physical examination of the abdomen revealed mild tenderness in the epigastric region without any palpable mass or signs of peritonitis. Skin turgor was decreased, indicating mild dehydration. Her height was 150 cm and weight was 45 kg, indicating a low body mass index (BMI) and poor nutritional status.

Initial laboratory tests confirmed severe hypoglycemia (RBG 52 mg/dL). The metabolic panel also showed mild hypoalbuminemia, raising suspicion of a protein-losing gastropathy. Imaging studies included ultrasound of the upper and lower abdomen, and barium contrast study (Oesophago-Maag-Duodenum). Abdominal ultrasound showed a thickened gastric wall, leading to an impression of gastritis. The bladder wall was also noted to be thickened, suggesting cystitis. The liver, gallbladder, pancreas, spleen, and kidneys were all within normal limits. Barium Contrast Study (OMD) confirmed multiple diagnoses simultaneously. The esophagogram showed that the esophageal caliber was enlarged down to the distal end, with a narrowed tip, creating a "rat tail" appearance pathognomonic for achalasia. During the stomach examination with the patient in a standing position, a significant inferior displacement of the stomach into the pelvic cavity was observed, confirming the diagnosis of gastroptosis. The study also revealed thickened mucosal folds, supporting the diagnosis of hypertrophic gastritis.

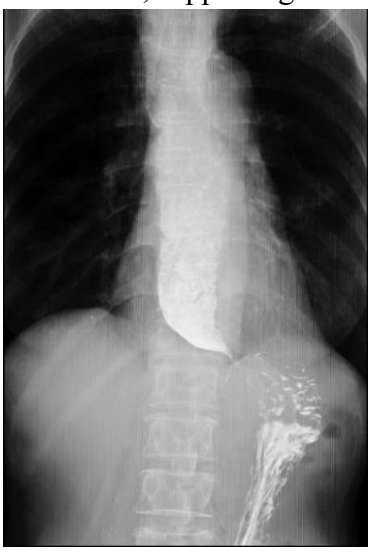

**Figure 1**. **Esophagography, AP erect view**
Source: Archives of Radiology, 2024

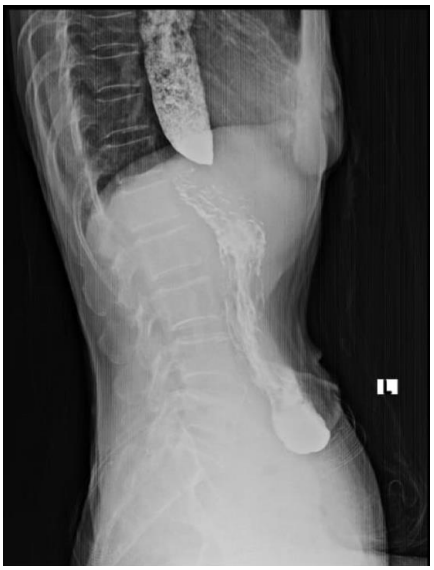

**Figure 2. Esophagography, lateral erect view**
Source: Archives of Radiology, 2024

These images show the severely dilated caliber of the esophagus filled with barium contrast down to the distal end. At the bottom, where the esophagus meets the stomach, there is a smooth, tapered narrowing, classically known as “rat’s tail” or “bird’s beak” sign. The retention of a large column of barium indicates stasis or impaired emptying of the esophagus. These findings are pathogmnomonic for achalasia.

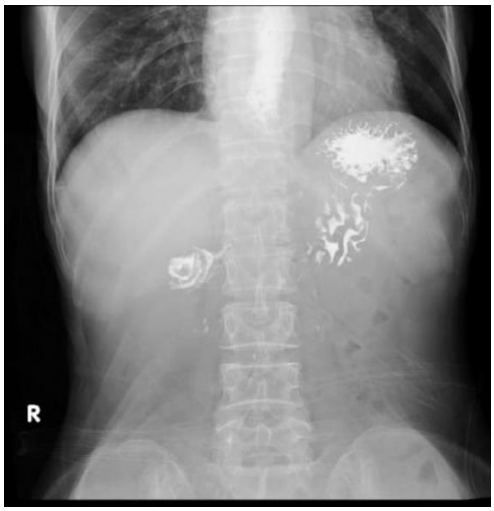


**Figure 3. Gastrography, AP view**
Source: Archives of Radiology, 2024

This image provides a closer look at the stomach's mucosal lining. It highlights markedly thickened, tortuous, and irregular mucosal folds (rugae). The barium contrast coats these prominent folds, creating a pattern that is characteristic of hypertrophic gastritis.

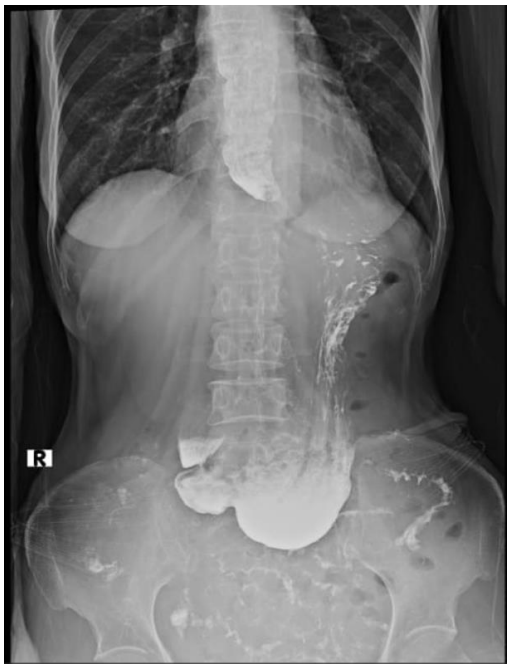


**Figure 4. Gastrography, AP erect view**
Source: Archives of Radiology, 2024

The entire stomach is shown to be severely displaced downward, with its lowest point sitting deep within the pelvic cavity, well below the iliac crests. This is a clear and dramatic depiction of severe gastroptosis. The thickened gastric folds of hypertrophic gastritis are again visible.

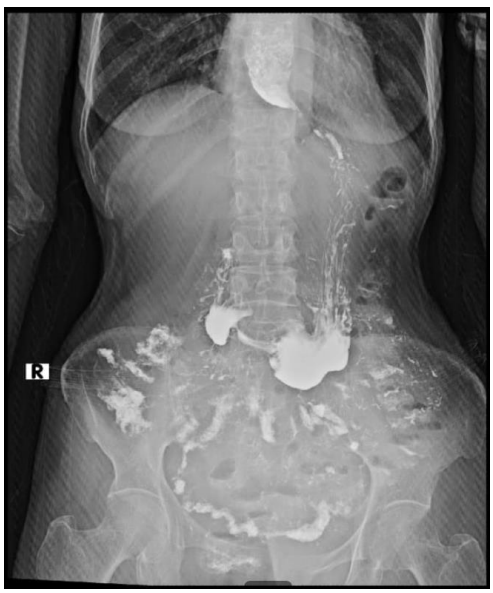


**Figure 5**. **Duodenography, AP supine view**
Source: Archives of Radiology, 2024

There was minimal contrast filling observed in the duodenum. The overall radiological impression was suggestive of a combination of achalasia, gastroptosis, hypertrophic gastritis, and hyperacidity. The initial management of the patient focused on stabilizing her acute condition. The hypoglycemia was immediately treated in the emergency department with an intravenous bolus of 40% dextrose, followed by a 10% dextrose infusion to maintain euglycemia. Pharmacological therapy during hospitalization included intravenous Lansoprazole and Pantoprazole (proton pump inhibitors), Ondansetron and Metoclopramide (antiemetics), Sucralfate syrup and Farmacrol Forte syrup (antacids), and Cisapride (a prokinetic agent). From a nutritional standpoint, the patient was given a diet of small, frequent liquid meals to facilitate passage through the obstructed esophagus and to avoid excessive distention of the ptotic stomach. The patient showed partial clinical improvement during her hospital stay, with better glycemic control and tolerance of a soft diet. She was discharged in good general condition with stable vital signs (BP 111/54 mmHg, pulse 58 beas per minute). Her discharge medications included esomeprazole, cisapride tablets, sucralfate syrup, and farmacrol forte syrup, with a plan for outpatient follow-up in one week.

## RESULTH AND DISCUSSION

This case highlights the significant challenges in attributing symptoms when multiple upper GI pathologies overlap. Cardinal symptoms such as epigastric pain, nausea, and vomiting may arise from any of the four existing conditions. Nausea and vomiting can result from food stasis due to functional obstruction in achalasia, delayed gastric emptying secondary to mechanical and functional dysmotility in gastroptosis, or significant mucosal inflammation in hypertrophic gastritis. Early satiety may manifest in achalasia, gastroptosis, or hypertrophic gastritis (Staszewska et al., 2023). These overlapping symptoms challenge standard diagnostic algorithms and underscore the need for comprehensive investigation and holistic clinical reasoning to achieve an accurate diagnosis in patients with refractory symptoms (Barros et al., 2025).

The life-threatening clinical manifestation in this patient was severe hypoglycemia. This condition was not merely the result of poor nutritional intake. Instead, it was the result of a dangerous dynamic interaction between esophageal stasis and delayed gastric emptying (Marathe et al., 2019). Achalasia causes food accumulation in the esophagus, creating a reservoir that prevents the regular and predictable delivery of nutrients to the stomach (Vaezi et al., 2020). Simultaneously, gastroptosis, with its abnormal gastric position and potential associated dysmotility, leads to slow and inconsistent gastric emptying, similar to functional gastroparesis (Staszewska et al., 2023). This combination produces a unique “double-delay” system, where the patient may effectively remain in a fasting state for hours after eating.

However, when the hydrostatic pressure of the esophageal food bolus eventually overcomes the lower esophageal sphincter resistance—or when body position changes occur—a large quantity of undigested food may suddenly and unpredictably enter the stomach and subsequently the small intestine. The rapid delivery of a large carbohydrate bolus to the proximal intestine is known to trigger an excessive release of incretin hormones, particularly

glucagon-like peptide-1 (GLP-1) (Karimi & Kohandel Gargari, 2024). This surge in GLP-1, in turn, provokes an exaggerated and disproportionate pancreatic insulin response (Millstein & Lawler, 2017). The resulting massive postprandial hyperinsulinemia leads to rapid glucose uptake by peripheral tissues, causing a drastic drop in blood glucose levels and severe reactive hypoglycemia.

Consequently, the hypoglycemia observed in this patient illustrates a significant temporal discordance between nutrient absorption and insulin response. This condition is notably intensified by the consecutive occurrence of achalasia and gastroptosis, creating what can be termed a "perfect metabolic storm."

The conditions diagnosed in this patient are individually rare, making their concurrent presentation an exceptional finding. The simultaneous occurrence of these four distinct pathologies has not been documented in the literature, underlining the unique nature of this case.

Gastroptosis is considered a rare and underdiagnosed condition with an unknown actual prevalence. There's currently a scarcity of data on the prevalance of gastroptosis (Tangul & Senayli, 2025). Some data suggest it primarily affects women between 20 and 50 years old and is often associated with low body weight. One study from Japan reported a prevalence of 12% in men and 43% in women, most of whom were underweight (Bestari et al., 2022).

Achalasia is also a rare esophageal motility disorder, with an incidence of approximately 1 in 100,000 individuals and a prevalence of 10 in 100,000 (Savarino et al., 2022). It typically affects adults between the ages of 30 and 60, with no gender predilection (Ramchandani & Pal, 2020). While gastritis is extremely common, a subtype of this condition called hypertrophic gastritis is an extremely rare disorder, with fewer than 1,000 cases reported in the literature to date. It predominantly affects middle-aged males, typically between 30 and 60 years old (Keener et al., 2023).

In contrast to the other conditions, hyperacidity associated with GERD is extremely common, affecting up to 20% of the population in the United States and 13% of people worldwide (Kasugai & Ogasawara, 2024). Its global prevalence has been steadily increasing, making it a significant public health issue.

Each condition has a distinct gold standard for diagnosis, highlighting the need for a multimodal diagnostic approach as was used in this case. Gastroptosis is established through radiological imaging of the abdomen performed while the patient is in an upright position. An upper GI series using barium contrast is the definitive method (Staszewska et al., 2023). High-resolution esophageal manometry (HRM) is considered the gold standard for diagnosing achalasia and its subtypes (Vaezi et al., 2020). However, a barium esophagram is also a highly sensitive and specific initial diagnostic tool.

The gold standard to diagnosing hypertrophic gastritis, as applied to all case of gastritis, is an esophagogastroduodenoscopy (EGD) with a deep or full-thickness mucosal biopsy of the affected gastric tissue to confirm the characteristic histological features (Azer et al., 2025). Ambulatory 24-hour esophageal pH monitoring is considered the gold standard for quantifying acid reflux (Lawenko & Lee, 2016). However, an upper endoscopy that reveals conclusive

evidence of reflux-induced damage, such as erosive esophagitis (Los Angeles grade B or higher), peptic stricture, or Barrett's esophagus, is also considered definitive for a GERD diagnosis (Simadibrata et al., 2023).

The hallmark finding on an upright upper GI series in gastroptosis is the downward displacement of the stomach, where the greater curvature is positioned below the level of the iliac crests (Staszewska et al., 2023). The stomach often appears severely elongated, J-shaped, and may extend down to the umbilicus or into the pelvic cavity (Xu & Garcia, 2024). Associated findings include delayed gastric emptying and retention of contrast material. CT scans can also confirm the abnormal inferior position of the stomach (Youssoufi et al., 2025).

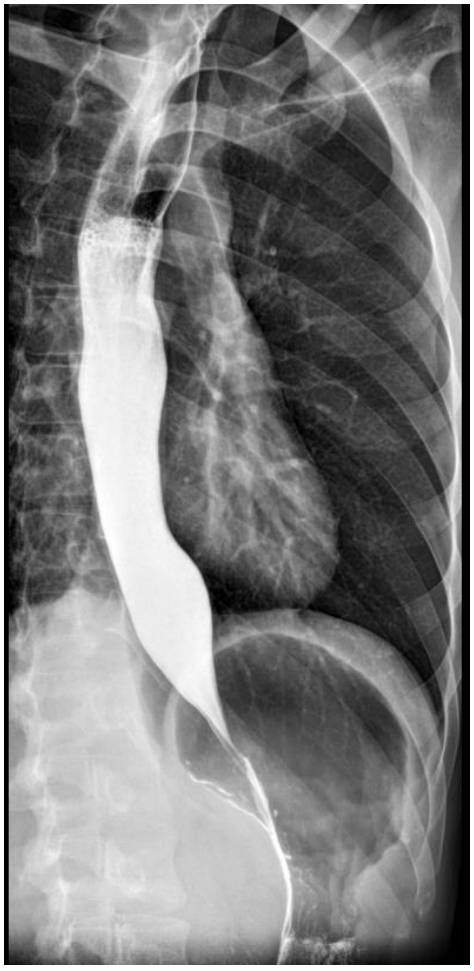

**Figure 6**. **"Rat's tail" sign**
Source: Archives of Radiology, 2024

The classic radiological sign of achalasia on a barium esophagogram is the "bird's beak" or "rat's tail" sign, which represents the smooth, tapered narrowing of the distal esophagus at the non-relaxing LES (Momodu & Wallen, 2025). Proximal to this narrowing, the esophagus is typically dilated and may contain retained food and secretions, leading to a visible air-fluid level on a chest X-ray. The chest radiograph may also show a widened mediastinum due to the

dilated esophagus. Barium studies will also demonstrate aperistalsis, or an absence of coordinated, propulsive esophageal contractions (Ramchandani & Pal, 2020).

While endoscopy is primary, radiological studies can show suggestive features in hypertrophic gastritis. A barium study may reveal markedly thickened, tortuous, and lobulated gastric folds (rugae), creating a "cerebriform" (brain-like) or cobblestone appearance (Keener et al., 2023). These changes are typically most prominent in the gastric body and fundus, with relative sparing of the antrum. On CT, hypertrophic gastritis presents as diffuse or segmental gastric wall thickening, often with associated submucosal edema (Gore et al., 2025).

Barium esophagography can directly visualize the reflux of contrast from the stomach into the esophagus and assess its volume and proximal extent (Katzka, 2018). It can also identify associated abnormalities such as esophageal dysmotility (impaired peristalsis), hiatal hernia, and complications like esophageal strictures. Real-time MRI is an emerging modality that can visualize anatomical factors contributing to reflux, such as a widened Angle of His (Kulinna-Cosentini et al., 2019).

The coexistence of two different motility disorders (achalasia and gastric dysmotility related to gastroptosis) with a significant inflammatory/hyperplastic condition (hypertrophic gastritis) raises questions about a possible unifying pathophysiological mechanism. A plausible hypothesis is an underlying neuro-immune dysregulation. The enteric nervous system (ENS) is the primary regulator of motility throughout the GI tract (Sharkey & Mawe, 2023). Damage to the myenteric plexus, particularly the loss of inhibitory neurons, is the known core pathology in achalasia (Camilleri, 2023). Similar mechanisms, including the loss of interstitial cells of Cajal (ICC) and neuronal dysfunction, are also proposed as the basis for idiopathic gastroparesis (Wang et al., 2021).

Existing literature indicates a strong bidirectional interaction between the mucosal immune system and the ENS (Wallrapp & Chiu, 2024). Chronic inflammation can trigger neuroplastic changes, neuronal damage, and motility dysfunction. In the context of this case, the chronic hypertrophic gastritis might not just be a standalone diagnosis but could be a trigger or an exacerbating factor for ENS dysfunction. The chronic inflammatory process in the gastric mucosa could create a pro-inflammatory cytokine environment that causes or worsens immune-mediated damage to the myenteric plexus (Wallrapp & Chiu, 2024). This damage then manifests proximally as achalasia (failure of inhibitory neurons in the LES) and distally as gastric dysmotility, which is worsened by the anatomical anomaly of gastroptosis (Zhai et al., 2025). This hypothesis is supported by studies showing a significant statistical association between H. pylori infection (a common cause of gastritis) and idiopathic achalasia, with the proposed mechanism being autoimmune damage to enteric neurons through molecular mimicry (Yaseri & Yaseri, 2023). Thus, the gastritis in this patient may have acted as an active driver in the pathophysiological cascade linking the inflammatory disorder with the motility disorders.

The constellation of diagnoses in this patient creates a significant management dificulty, where standard treatment strategies for one condition can directly conflict with the management of another.

1. Prokinetic Conflict: Prokinetic agents, such as metoclopramide or domperidone, are often considered for the management of gastroparesis or gastric dysmotility to accelerate gastric emptying (Isola et al., 2025). However, in this patient, the use of prokinetics would be strongly contraindicated before the distal functional obstruction at the LES (achalasia) is resolved. Forcing gastric contents forward against a non-relaxing LES would dramatically increase intragastric pressure, worsening pain, regurgitation, and increasing the risk of pulmonary aspiration (Lewis et al., 2016).
2. Sequence of Intervention: A critical question arises regarding the sequence of treatment. Addressing the achalasia first—for example, with POEM or pneumatic dilation—would logically eliminate the proximal obstruction. However, this could lead to new problems. The now-free flow of food from the esophagus would accumulate directly in the ptotic and atonic stomach, potentially worsening gastroparesis symptoms, early satiety, and increasing the volume of gastroesophageal reflux (Khashab, 2020).
3. Medical Management Challenges: Medical management for achalasia, such as the use of nitrates or calcium channel blockers, aims to relax the smooth muscle of the LES. However, these drugs are non-specific and could worsen atony in the gastric body, thereby further inhibiting gastric emptying (Idrizaj et al., 2021).

This situation represents a therapeutic paradox. Treating one problem risks exacerbating another. This underlines the limitation of single-disease-based clinical guidelines when faced with complex multi-morbidity cases. Management must be personalized, staged, and closely monitored. The most logical approach is to address the most significant and proximal obstruction first (achalasia), followed by very intensive medical and dietary management to address the gastric dysmotility and hypertrophic gastropathy.

The nonspecific nature of the patient's initial symptoms necessitates a broad differential, which can be narrowed by specific radiological findings. The clinical symptoms of epigastric pain and nausea overlap significantly with more common conditions such as gastritis, pancreatitis, and peptic ulcer disease (Staszewska et al., 2023). While the radiological finding of a displaced stomach is definitive for gastroptosis, these other conditions have distinct radiological features (e.g., ulcer craters for PUD, inflammatory changes on CT for pancreatitis) that differentiate them as the cause of symptoms (Xu & Garcia, 2024).

The primary radiological differential for achalasia is pseudoachalasia, which mimics primary achalasia but is caused by an underlying pathology, most commonly a malignancy at the gastroesophageal junction. Radiologically, primary achalasia typically shows a smooth, tapered "bird's beak" with a narrowed segment less than 3.5 cm long (Barnett et al., 2020). In contrast, pseudoachalasia may present with a longer narrowed segment (>3.5 cm), mucosal irregularity, nodularity, or abrupt shouldering (Schizas et al., 2020). CT is crucial in these cases, as asymmetric or marked wall thickening (>10 mm) is highly suggestive of an underlying malignancy. Another important differential is an esophageal stricture, which is an abnormal, fixed narrowing of the lumen. The most common cause is a benign peptic stricture from chronic GERD, which classically appears as a smooth, tapered, and concentric narrowing (Desai &

Moustarah, 2025). In contrast, the narrowing in achalasia is functional, not a fixed fibrotic structure. Malignant strictures can also cause pseudoachalasia and may appear radiologically as an abrupt, asymmetric narrowing with an irregular contour and shelf-like margins (Yang et al., 2024). Finally, scleroderma esophagus can be distinguished on barium swallow by a patulous or open LES, in contrast to the non-relaxing sphincter of achalasia (Voulgaris & Karamanolis, 2021).

Radiological finding of hypertrophic gastritis has a wide differential diagnosis that primarily includes infiltrative malignancies such as gastric lymphoma and scirrhous adenocarcinoma (linitis plastica), as well as other conditions like Zollinger-Ellison syndrome. CT imaging is valuable for differentiation; malignancies often exhibit more pronounced wall thickening (especially lymphoma) and a loss of normal gastric wall stratification, which is a key predictor of malignancy. Furthermore, the presence of ulcerations and involvement of the gastric antrum are significantly more common in advanced gastric cancer than in benign hypertrophic gastritis, which typically spares the antrum (Seo et al., 2024).

Symptoms of heartburn can mimic other causes of chest pain, including cardiac issues and other esophageal motility disorders. From a radiological standpoint, a barium study helps differentiate GERD from its mimics. It can directly visualize reflux and identify associated findings like a hiatal hernia or impaired esophageal clearance (Katz et al., 2022). This helps distinguish it from peptic ulcer disease, where an ulcer crater may be seen, or from achalasia, which has its own classic "bird's beak" appearance (Levine et al., 2016). A barium study can also characterize esophageal strictures, helping to differentiate benign peptic strictures (typically smooth and tapered) from those caused by malignancy (often irregular with shouldered margins) (Yang et al., 2024).

Although there are case reports on the partial coexistence of these conditions, such as achalasia and gastroparesis or case reports of Ménétrier's disease, no reports were found that documented all four diagnoses simultaneously in a single patient. The uniqueness of this case lies in the combination of an anatomical (gastroptosis), primary motor (achalasia), inflammatory/hyperplastic (hypertrophic gastropathy), and functional (hyperacidity) abnormality, which collectively produced a severe and unexpected metabolic manifestation of hypoglycemia. Therefore, this report provides a new and important contribution to the medical literature.

## CONCLUSION

This case highlights the significant diagnostic and therapeutic challenges arising from the rare coexistence of gastroptosis, achalasia, and hypertrophic gastritis, illustrating how their complex pathophysiological interplay can cause severe systemic effects like life-threatening hypoglycemia via neuro-hormonal dysregulation. It underscores the critical need for clinicians to maintain a broad differential diagnosis and consider multiple overlapping conditions in patients with refractory or atypical upper GI symptoms. Effective management requires a thorough diagnostic process, a nuanced understanding of these interacting disorders, and a personalized, multidisciplinary treatment strategy. Future research should focus on elucidating

the detailed neuro-hormonal mechanisms underlying these multi-pathological interactions and developing standardized protocols for their integrated diagnosis and management.

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