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CASE REPORT OPEN ACCESS

Case report: A young female with epigastric pain, functional dyspepsia, and chronic cholecystitis diagnostic and therapeutic challenges

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Abstract

Young adults face a diagnostic challenge when experiencing epigastric pain because it can stem from organic conditions such as gastritis and cholecystitis or functional disorders including dyspepsia. The case demonstrates the difficulties that occur during diagnosis and treatment of gastrointestinal conditions which overlap with each other. A 19-year-old female patient experienced recurring pain in her epigastric area. The patient received unremarkable lab results but endoscopy showed grade II esophagitis together with mixed gastritis and ultrasound results confirmed chronic cholecystitis. The patient received *H. pylori* eradication treatment with clarithromycin/omeprazole/tinidazole and antispasmodic mebeverine and digestive enzyme pancreatin despite normal *H. pylori* test results. The case demonstrates the requirement for multiple evaluation methods including endoscopy and imaging and symptom evaluation when epigastric pain results from both functional and organic causes.

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Introduction

Patients commonly present with epigastric pain which describes abdominal discomfort that occurs in the upper central part of the abdomen and requires extensive evaluation to determine its source. The symptoms can stem from different sources including gastrointestinal organs as well as hepatobiliary organs pancreatic organs and heart tissue (Miranda, 2022; Zheng *et al.*, 2025). A stepwise diagnostic procedure helps identify serious pathology without performing unnecessary interventions because the symptoms can stem from multiple causes. The evaluation starts with a detailed patient history and physical assessment which leads to basic laboratory tests to detect infections and organ dysfunction as well as inflammatory processes (Chong *et al.*, 2023; Maffei *et al.*, 2024). The results of normal laboratory tests do not automatically eliminate the possibility of important underlying medical conditions. A young female patient with persistent epigastric pain showed normal blood test results according to Youssoufi *et al.* (2025).

The presented case demonstrates a typical diagnostic dilemma because patients with dyspepsia can have both functional and structural gastrointestinal issues which need imaging or endoscopy for detection (Suzuki, 2021; Black *et al.*, 2022). The diagnostic tests involving upper gastrointestinal endoscopy and abdominal ultrasonography revealed important findings which blood tests failed to detect. Laboratory tests should not be the sole basis for assessing persistent or unexplained epigastric pain. The discovery of gastritis and biliary sludge along with gastric motility disorders becomes possible through endoscopy and imaging modalities when these conditions escape initial detection. The clinical approach requires practitioners to keep a strong awareness for potential conditions while creating diagnostic plans based on individual patient circumstances when preliminary tests fail to provide answers.

Case presentation

A 19-year-old female patient experiences recurring epigastric pain which manifests as burning discomfort that intensifies following meals. The patient has no symptoms of weight loss or hematemesis or fever and she has never undergone abdominal surgery. The postprandial nature of her pain together with her lack of surgical history and alarm features makes functional dyspepsia or gastritis a possible diagnosis but further testing is needed to rule out biliary dyskinesia and early peptic ulcer disease (Table 1).

Table 1. Summary of diagnostic findings

Test	Results	Clinical significance
H. pylori breath test	Negative (0.01 y.e.)	Low probability of active *H. pylori* infection
Endoscopy	Grade II esophagitis, mixed gastritis	Confirms mucosal inflammation; rules out ulcers
Ultrasound	Gallbladder wall thickening (2.3 mm)	Supports chronic cholecystitis
CBC/ESR	All parameters normal	Excludes anemia/infection

Endoscopy

The upper gastrointestinal endoscopy showed Grade II esophagitis which involved esophageal inflammation and mucosal changes at 36 cm Z-line distance with an open cardiac sphincter indicating gastroesophageal reflux as a possible cause. The gastric examination revealed widespread mucosal hyperemia which indicated mixed gastritis but no erosions or ulcers were present. The endoscopic findings confirm both esophagitis and gastritis as the causes of the patient's chronic burning postprandial epigastric pain which normal laboratory tests failed to explain (Table 2, Fig. 1&2).

Table 2. Endoscopy findings key abnormalities

Location	Findings	Grade/Measurement
Esophagus	Mucosal hyperemia	Grade II esophagitis
	Incomplete cardial closure	Z-line at 36 cm
Stomach	Diffuse hyperemia	Mixed gastritis
	No erosions/ulcers	

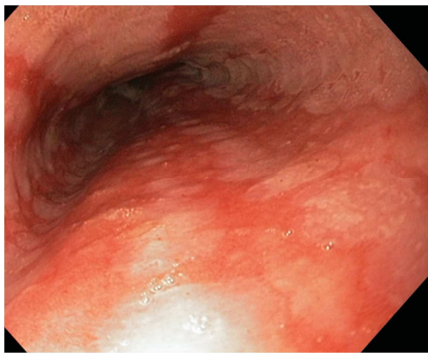

Fig. 1. The endoscopy showed grade II esophagitis which involved esophageal inflammation and mucosal changes at 36 cm Z-line distance with an open cardiac sphincter indicating gastroesophageal reflux as a possible cause

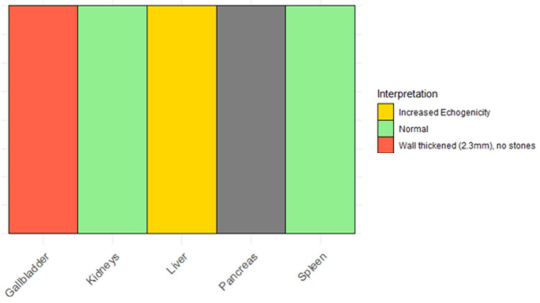

Fig. 2. Upper GI endoscopy findings. Endoscopic examination showed grade II esophagitis, an incompetent cardia, and the presence of white fluid in the gastric lumen, thickened and tortuous gastric folds, and diffuse mucosal hyperemia. These findings support a diagnosis of reflux esophagitis and mixed gastritis

Table 3. Ultrasound findings, key abnormalitie

Organ	Findings	Normal range	Significance
Gallbladder	Wall thickening (2.3 mm)	<3 mm	Chronic cholecystitis
	No stones/sludge	Absent	Rules out acute obstruction
Liver	Mild hyperechogenicity	Homogeneous	Likely fatty changes

Abdominal ultrasound

The abdominal ultrasonography revealed a 2.3 mm thickened gallbladder wall without gallstones which supported a diagnosis of chronic cholecystitis possibly with acalculous characteristics. The liver and pancreas and spleen showed normal findings but the liver displayed mild hyperechogenicity which might indicate early hepatic steatosis. The confirmed esophagitis and gastritis explain the primary symptoms but the patient's postprandial epigastric discomfort may also result from chronic cholecystitis so a multidisciplinary management approach is necessary (Table 3, Fig. 3&4).

Fig. 3. Abdominal ultrasound showed gallbladder wall thickening of 2.3 mm without evidence of gallstones, consistent with a diagnosis of chronic cholecystitis, possibly of an acalculous nature

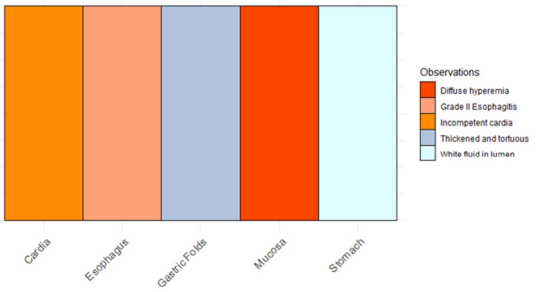

Fig. 4. Abdominal ultrasound findings. Ultrasound imaging revealed a normal-sized liver with increased echogenicity suggestive of fatty infiltration, a gallbladder with thickened walls (2.3 mm) but without gallstones, and a normal pancreas, kidneys, and spleen. Findings are consistent with chronic cholecystitis

Diagnostic workup

Initial laboratory findings for the patient were within normal limits, including a hemoglobin level of 130 g/L, white blood cell count of 5.71 × 10^9/L, and an erythrocyte sedimentation rate (ESR) of 5 mm/hr, indicating no signs of anemia, infection, or systemic inflammation. Additionally, the Helicobacter pylori breath test was negative (0.01 y.e.), suggesting that *H. pylori* infection is unlikely to be the cause of her

symptoms. These unremarkable results, while reassuring, do not rule out functional or structural gastrointestinal disorders, emphasizing the need for further investigation.

Medications prescribed

The patient was initiated on empirical *H. pylori* triple therapy (Typorex), comprising clarithromycin 250 mg, omeprazole 20 mg, and tinidazole 500 mg, despite a negative breath test, possibly due to the endoscopic evidence of mixed gastritis and the potential for false negatives in non-invasive testing. Mebeverine (Duspatalin®) 200 mg three times daily was prescribed to address possible visceral hypersensitivity or gastrointestinal spasm, common in functional gastrointestinal disorders. Additionally, pancreatin (Creon®) 10,000 IU was included to support digestive enzyme function, targeting potential biliary-pancreatic insufficiency suggested by chronic cholecystitis and mild hepatic changes. This multifaceted regimen reflects a pragmatic approach to overlapping GI symptoms where multiple mechanisms may coexist.

Discussion

This case highlights the diagnostic complexity of chronic epigastric pain in young adults, particularly when initial laboratory findings are unremarkable. In our 19-year-old female patient, normal complete blood count (CBC), inflammatory markers (ESR), and a negative *Helicobacter pylori* breath test initially suggested a functional etiology rather than an organic one (Schneider and McKegney, 2022). However, endoscopic and ultrasonographic evaluations revealed clinically significant abnormalities, including Grade II esophagitis, mixed gastritis, and gallbladder wall thickening indicative of chronic cholecystitis (Mencarini *et al.*, 2024). This clear discrepancy between laboratory and imaging findings underscores the limitations of relying solely on non-invasive tests, particularly in gastrointestinal symptomatology (Alshammari *et al.*, 2021). The possibility of a false-negative *H. pylori* result also warrants attention, as no endoscopic biopsy was performed a diagnostic step that might have yielded greater sensitivity (Bordin *et al.*, 2021). Another key insight is the coexistence of functional and organic gastrointestinal disorders.

According to the Rome IV criteria, functional dyspepsia remains a valid consideration, particularly given the patient's postprandial burning pain and lack of alarming features such as weight loss or gastrointestinal bleeding (Wauters *et al.*, 2021; Savarino *et al.*, 2022; Aye *et al.*, 2025). However, the identification of organic pathology namely esophagitis, gastritis, and chronic cholecystitis points to a dual-pathology scenario where functional and structural processes overlap (Gallaher and Charles, 2022) . This emphasizes the need for clinicians to consider both dimensions in patients with persistent symptoms, even in the absence of definitive lab abnormalities.

Therapeutically, a pragmatic, multimodal approach was employed. The use of a proton pump inhibitor (omeprazole) is appropriate for acid suppression in esophagitis and gastritis (Sezgin and Yardımcı, 2025). Despite the negative *H. pylori* test, empirical triple therapy (Typorex) was initiated, accounting for potential false negatives and the prevalence of *H. pylori*-related gastritis in this demographic (Seisenbekova *et al.*, 2025). Mebeverine, an antispasmodic, was prescribed to address suspected visceral hypersensitivity or functional bowel spasm (Black and Ford, 2025).

Additionally, pancreatin (Creon®) was introduced to support digestion, potentially mitigating subclinical biliary-pancreatic dysfunction associated with chronic cholecystitis (Chu *et al.*, 2025). Overall, this case underscores the value of a holistic, individualized treatment plan informed by both functional and structural assessments, particularly in young patients with unexplained chronic gastrointestinal symptoms.

Conclusion

This case underscores the importance of comprehensive evaluation in young patients presenting with chronic epigastric pain. First, it illustrates that normal laboratory tests—including

inflammatory markers and *H. pylori* breathe testing—do not exclude significant underlying pathology; endoscopic and imaging assessments are essential for accurate diagnosis. Second, chronic cholecystitis, particularly in its acalculous form, can closely mimic functional dyspepsia, highlighting the diagnostic value of abdominal ultrasonography. Third, in regions with high *H. pylori* prevalence, empirical eradication therapy may be justified even in the setting of a negative breath test, especially when endoscopic findings are suggestive.

Recommendations

To enhance diagnostic accuracy and optimize patient outcomes, we recommend repeat *H. pylori* testing using stool antigen if symptoms persist, as it may improve detection sensitivity. Additionally, given the ultrasonographic evidence of chronic cholecystitis, surgical consultation for possible cholecystectomy should be considered if the patient's symptoms intensify or fail to respond to medical management.

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