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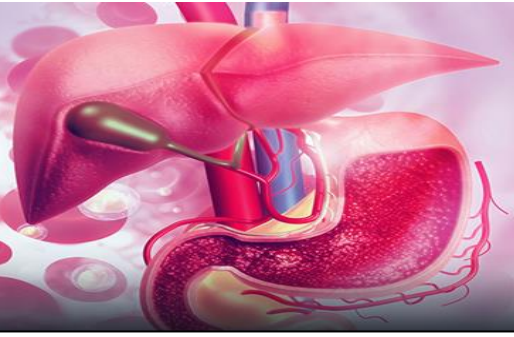

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Best Regards,

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Impact of H. pylori infection on extraintestinal cancer prevalence

INTRODUCTION

H. pylori infection

Helicobacter pylori (H. pylori) is a flagellated gram-negative bacterium that colonizes the mucus layer of the stomach and proximal duodenum. A recent systematic review and meta-regression estimate that approximately 43.9% of the global adult population was infected with this pathogen.[1] The risk of H. pylori infection was influenced by various factors, including geographic and ethnic background, socioeconomic status, age, and hygiene practices. H. pylori is a significant contributor to the pathogenesis of chronic gastritis, peptic ulcers, and several malignancies, particularly gastric adenocarcinoma and mucosa-associated lymphoid tissue (MALT) lymphoma. The development of these conditions was linked to host genetic predispositions to carcinogenesis and specific virulence factors of H. pylori, notably the cytotoxin-associated gene A (CagA) antigen. Conversely, the effects of H. pylori eradication on gastric cancer and its role in the development of extra-gastric non-malignant diseases, such as autoimmune and cardiovascular conditions, as well as malignancies including lung, pancreatic, and colon cancers, remain subjects of considerable debate.[2] Therefore, this study aims to evaluate the association between H. pylori infection and its eradication with the subsequent development of both gastrointestinal (GI) and non-GI cancers.

METHODS- This retrospective cohort study utilized the Disease Analyzer (IQVIA) database, which has drug prescriptions, diagnoses, and basic medical and demographic data obtained directly and in anonymous format from computer systems used in the practices of general practitioners and specialists. This study included adult patients (≥18 years) diagnosed with H. pylori infection, requiring a minimum of 12 months of observation before the index date and at least 6 months of follow-up afterward. Patients with prior or recent cancer diagnoses were excluded. Non-H. pylori patients were matched 1:1 based on demographics and comorbidities. The primary outcomes included initial cancer diagnoses (ICD-10: C00–C97), focusing on gastrointestinal and respiratory cancers. Statistical analyses involved the Wilcoxon signed-rank test, McNemar test, and Stuart-Maxwell test for baseline comparisons; Kaplan-Meier curves for cumulative incidence; and univariable Cox regression to assess the association between H. pylori and cancer outcomes.

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RESULTS- This study examined 25,317 H. pylori-positive individuals alongside a matched control group. Over up to 10 years, 12.8% of the H. pylori cohort developed cancer compared to 11.8% in controls (p=0.002) (Figure 1). A significant association was found between H. pylori and cancer risk (HR: 1.11; 95% CI: 1.04 to 1.18), particularly for stomach (HR: 1.59) and pancreatic cancers (HR: 1.46). Non-eradicated patients had a higher cancer rate (13.2%) versus controls (11.8%, p<0.001), while eradicated patients showed no significant difference (12.5%, p=0.231) (Figure 2). H. pylori eradication was negatively associated with bronchus and lung cancer (HR: 0.60; 95% CI: 0.44 to 0.83), suggesting that H. pylori infection may increase cancer risk, especially for stomach and pancreatic cancers, with eradication potentially lowering the risk for some malignancies.

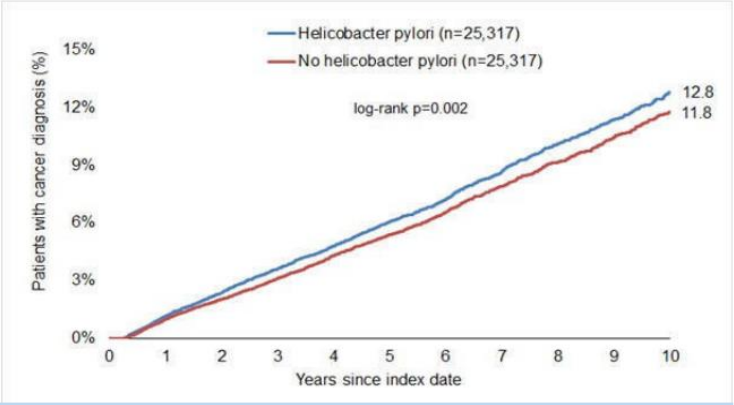

Figure 1. Cumulative cancer incidence in patients with and without H. pylori

DISCUSSION- In this study, 12.8% of individuals with H. pylori were diagnosed with cancer over a decade of follow-up, indicating a significant association with a hazard ratio (HR) of 1.11. While the association with gastric cancer was notable, it did not achieve statistical significance (p = 0.049), contrasting with existing literature that consistently reports a reduced incidence of gastric cancer following H. pylori eradication.[3] The current study's subgroup analyses revealed that H. pylori eradication significantly impacted bronchus and lung cancer, yielding an HR of 0.60, suggesting a lower risk for those who eradicated the infection, along with a trend indicating that non-eradicated H. pylori was associated with an increased risk of these cancers (p = 0.035). In contrast, findings for gastric cancer (GC) showed a decreased HR of 1.48 after eradication, compared to an HR of 1.7 in the non-eradicated group; however, this difference did not reach statistical significance, diverging from prior research indicating a protective effect of H. pylori eradication on GC incidence.[4]

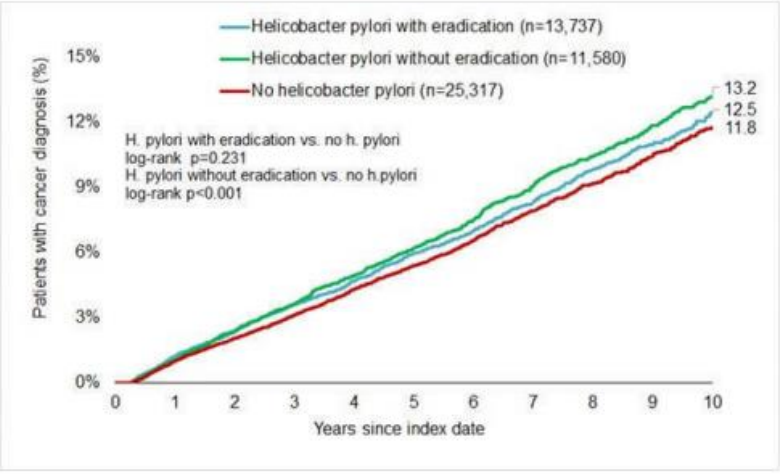

Figure 2. Cumulative cancer incidence in patients with and without H. pylori after receiving eradication therapy

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CONCLUSION-The analysis provided a comprehensive overview of 10-year cancer prevalence related to H. pylori infection. Higher cancer rates were observed among individuals with H. pylori, with a non-eradicated status linked to subsequent cancer diagnoses. Notably, H. pylori eradication was associated with a decreased incidence of bronchial and lung cancer, highlighting its potential protective role in this context.

H. pylori infection was linked to higher cancer prevalence, but its eradication may reduce the risk of bronchial and lung cancers, suggesting a potential protective effect.

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Relationship Between the Triglyceride-Glucose Index and Gallstone Risk

INTRODUCTION- Gallstones are a common gastrointestinal condition, affecting approximately 10–15% of the global population, with variations observed across different countries.[1] In the United States, it was estimated that about 10–20% of individuals have gallstones, and this prevalence is increasing.[2] The formation of gallstones is influenced by both genetic and environmental factors. Notable risk factors include age, gender, race, pregnancy, obesity, diabetes, and elevated cholesterol levels. Despite this, there were no reliable clinical indicators for predicting or preventing the development of gallstones. The TyG index, which combines triglycerides and fasting blood glucose, has been identified as a significant marker associated with various metabolic conditions, including coronary heart disease and diabetes. It serves as a useful alternative for assessing insulin resistance (IR), a key risk factor for several metabolic diseases. Given the established links between the TyG index and various health conditions, it was important to explore its potential association with gallstones. To date, no studies have specifically examined this relationship.[3] Therefore, this study investigated the connection between the TyG index and gallstones.

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METHODS- This cross-sectional study utilized data from the National Health and Nutrition Examination Survey (NHANES) conducted by the National Center for Health Statistics (NCHS). Initially, 15,560 participants were considered; however, individuals lacking gallstone data (n=6,328) and those without triglyceride-glucose (TyG) index values (n=5,286) were excluded, yielding a final study population of 3,870 participants. Gallstone presence was determined via a questionnaire inquiring whether a healthcare provider had ever diagnosed the participant with gallstones. The TyG index was calculated using the formula: Ln [triglycerides (mg/dL) × fasting blood glucose (mg/dL)/2], with measurements obtained through automated biochemical analyzers. The study employed multivariate logistic regression models to assess the association between the TyG index and gallstone risk, adjusting for covariates including age, gender, race, education level, marital status, and relevant health conditions such as hypertension and diabetes.

RESULTS- This study found a 10.4% prevalence rate of gallstones among 3,870 participants, with those affected exhibiting a significantly higher TyG index (8.71 ± 0.67) compared to those without gallstones (8.49 ± 0.69) ($P < 0.001$). Each unit increase in the TyG index was associated with a 53% increased risk of gallstones in the unadjusted model (OR 1.53) and a 41% increase in the fully adjusted model (OR 1.41). Sensitivity analysis further indicated that higher TyG index quartiles correlated with an increased risk of gallstones (OR 1.93) (Table 1). Additionally, subgroup analyses revealed significant interactions based on age, gender, BMI, and cholesterol levels with the strongest associations observed in individuals under 50 years, women, and those with higher BMI or cholesterol (all $P < 0.05$).

TyG index	Non-adjusted	Adjust I	Adjust II
Continuous	1.53 (1.33, 1.76) <0.0001	1.45 (1.24, 1.70) <0.0001	1.41 (1.07, 1.86) 0.0134
Categories			
Quartile 1	1.0	1.0	1.0
Quartile 2	1.97 (1.40, 2.78) 0.0001	1.71 (1.20, 2.43) 0.0030	1.70 (1.19, 2.43) 0.0035
Quartile 3	2.11 (1.50, 2.96) <0.0001	1.73 (1.21, 2.46) 0.0025	1.62 (1.12, 2.35) 0.0104
Quartile 4	2.88 (2.07, 3.99) <0.0001	2.32 (1.63, 3.28) <0.0001	1.93 (1.27, 2.94) 0.0022
p for trend	<0.01	<0.01	<0.01

Table 1. Relationship between the TyG index and the risk of gallstones assessed using logistic regression analysis.

DISCUSSION- This study demonstrated that the TyG (triglyceride-glucose) index significantly predicts gallstone risk, showing a 41% increased risk for each unit rise. This finding aligns with Ahmed et al. which emphasized the role of insulin resistance in gallstone formation.[4] Additionally, results corroborate Guerrero-Romero et al.'s validation of the TyG index as a reliable marker for insulin resistance, underscoring its practical application in clinical settings due to its high sensitivity and cost-effectiveness.[5] Furthermore, the study revealed

a strong positive association between the TyG index and gallstone formation, particularly in individuals with a BMI greater than 25. These findings further reinforce the existing literature that identified insulin resistance as a critical factor in the development of gallstones. Elevated insulin resistance can disrupt cholesterol metabolism and biliary lipid secretion, thereby increasing the likelihood of gallstone formation. Overall, the insights gained from this study highlight the importance of monitoring the TyG index as a valuable tool for assessing gallstone risk, especially in overweight populations.[6]

CONCLUSION- This study demonstrated a significant correlation between a higher TyG index and an increased risk of gallstones. The association was particularly notable in specific subgroups, including individuals under 50 years of age, women, those with total cholesterol levels above 200 mg/dL, individuals with a BMI over 25, and those without diabetes. Consequently, the TyG index could serve as a valuable predictor for assessing the risk of gallstones in these populations.

The TyG index may serve as a novel marker for assessing the risk of gallstones, warranting further investigation into its potential role in predicting gallstone development in individuals.

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INTRODUCTION- The diagnosis of small bowel diseases poses significant challenges in clinical practice, as conventional endoscopes are limited to specific segments of the small intestine. The introduction of capsule endoscopy (CE)[1] and balloon-assisted enteroscopy, including double-balloon enteroscopy (DBE) and single-balloon enteroscopy (SBE), has enhanced the capacity for both diagnosis and therapeutic intervention. SBE, characterized by a single balloon located at the tip of the over tube, offers advantages over DBE, including simplified setup and reduced procedure time. Despite these benefits, there was a lack of studies investigating the efficacy and safety of single-operator SBE, particularly in larger sample sizes.[2] This study aimed to evaluate the efficacy and safety of single-operator SBE in diagnosing small intestinal diseases, to promote its adoption in clinical settings.

METHODS-

This single-center retrospective study evaluated the medical records of patients who underwent single-operator small bowel enteroscopy (SBE) for suspected small intestinal disorders. Prior to the procedure, patients were required to fast for 12 hours and follow a low-residue diet, supplemented by 4 liters of polyethylene glycol for bowel preparation. The SBE procedures were performed using the Olympus SBE endoscope system. Oral SBE was conducted under general anesthesia, while anal SBE utilized conscious sedation.

During the procedure, the operator slid the overtube to the tip of the endoscope, inflated the balloon upon reaching the Treitz ligament, and employed the jiggling technique to pull back both instruments for effectively shortening and straightening the intestine while manipulating the control unit with the left hand. The choice of intubation route was based on clinical symptoms and imaging findings, with the oral approach preferred for lesions suspected in the proximal small bowel.

The primary outcome was the diagnostic yield of SBE, defined as the proportion of significant pathological lesions detected during the procedures. Secondary outcomes included the total enteroscopy rate, procedure time, intubation depth, therapeutic interventions performed, and any complications that arose.

RESULTS- During the study period, 1,422 enteroscopic examinations were performed on 922 patients, with 250 undergoing the oral approach, 172 the anal approach, and 500 a combined approach. Total enteroscopy was achieved in 253 patients (27.44%). Diagnostic yield improved from 60% to 83.33% across the subgroups, and total enteroscopy rates increased from 34.21% to 67.86%. The mean procedure times were 69.28 minutes for the oral approach and 64.95 minutes for the anal approach, with times decreasing as trainees gained experience showed in Figure 1. Intubation depths averaged 389.95 cm for the oral approach and 191.81 cm for the anal approach. Positive findings were detected in 565 patients (61.30%), with common diagnoses including Crohn's disease (7.05%), small intestinal lymphoma (6.83%), and small intestinal carcinoma (6.29%). Despite a combined approach, total enteroscopy failed in 198 patients, and 159 patients had no abnormalities detected during the procedure.

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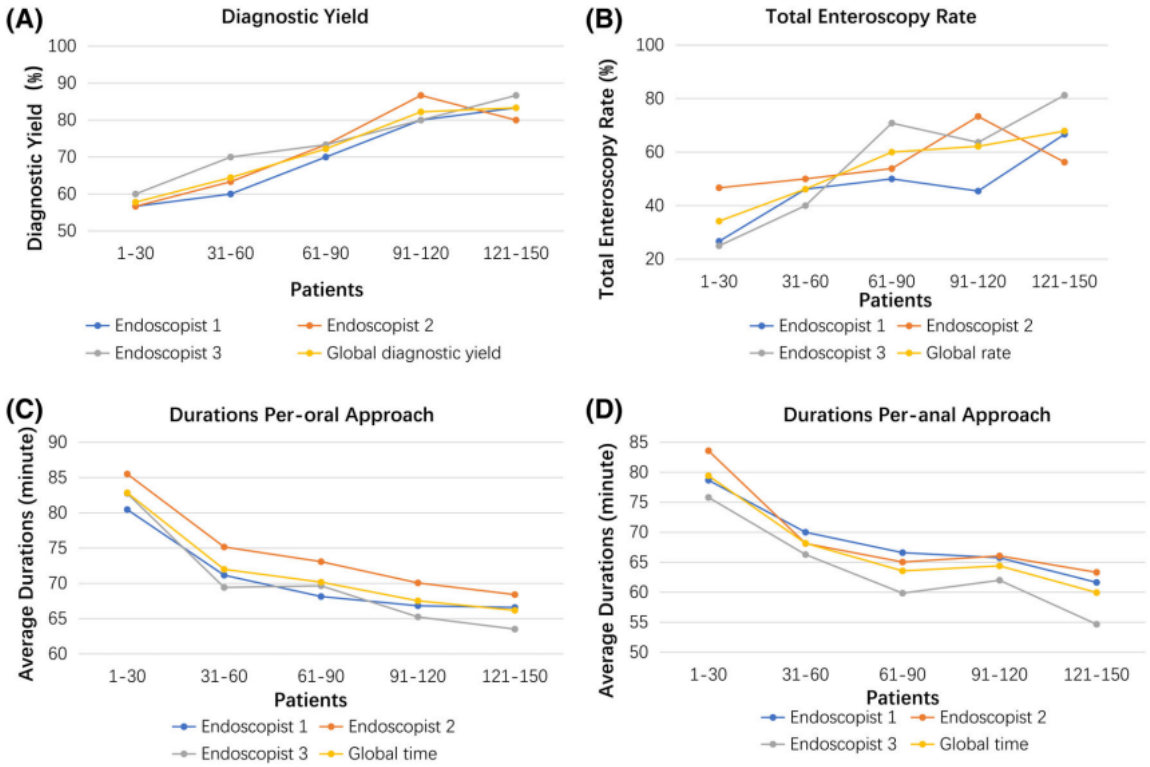

Figure 1. Stratification analysis of patient quartiles highlighting (A) diagnostic yields, (B) total enteroscopy rates, (C) procedure times for the per-oral approach, and (D) procedure times for the per-anal approach.

DISCUSSION- This study demonstrated that single-operator small bowel enteroscopy (SBE) was highly effective, achieving a diagnostic yield of 78.52% and a total enteroscopy rate of 56.10%. These findings surpass the diagnostic yields reported by Efthimios et al. and Takano et al. for dual-operator SBE, which were lower.[3,4] Additionally, total enteroscopy rate exceeds those reported by Tsujikawa et al. (25%) and May et al. (22%). The procedure also exhibited a low complication rate, with only one instance of perforation, underscoring its efficacy and safety.[5,6]

CONCLUSION- The single-operator small bowel enteroscopy (SBE) demonstrated significant effectiveness, safety, and operational simplicity. Consequently, it holds promise as a viable alternative to the conventional dual-operator SBE approach and has the potential to establish itself as the preferred method for diagnosing and managing small intestinal disorders.

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Single-operator small bowel enteroscopy (SBE) is an effective and safe alternative to dual-operator techniques, offering a promising option for diagnosing and managing small intestinal disorders.

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Case Report on Duodenal Hematoma After Endoscopic Duodenal Biopsy

INTRODUCTION- Intramural duodenal hematoma is a rare complication of upper endoscopy, with limited information available primarily from case reports, leaving a gap in incidence data for adults. This condition often arises from tissue sampling during the procedure and can lead to significant sequelae, including prolonged nutritional support due to duodenal obstruction.[1] Early recognition is crucial for initiating conservative management, which typically involves nasogastric decompression and parenteral nutrition. In severe cases, surgical decompression may be necessary, and recent reports suggest the potential for minimally invasive treatment options.

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CASE PRESENTATION- A 21-year-old man with eosinophilic esophagitis was admitted for severe abdominal pain that started in the lower abdomen and progressed to the right upper quadrant and epigastrium. Four days prior, he had an upper endoscopy with duodenal biopsies for suspected celiac disease, which showed vascular congestion but no eosinophilic infiltration.

Upon admission, he exhibited tenderness in the right upper quadrant and epigastrium, and lab investigations revealed hypokalaemia. A CT scan showed a duodenal hematoma measuring 4.7 x 3.0 x 3.5 cm, causing luminal narrowing. Over 24 hours, his condition worsened, leading to nausea and bilious vomiting, prompting a repeat CT scan that revealed hematoma enlargement (3.9 x 8.3 x 4.5 cm). A nasogastric tube was placed with copious output.

On day 3, CT angiography showed active extravasation and urgent gastroduodenal arterial embolization was performed. Despite nasogastric decompression and parenteral feeding, the patient did not improve. On day 7, laparoscopic evacuation of the hematoma was carried out, during which a submucosal clot was evacuated, and a drain was placed.

Post-surgery, the nasogastric output decreased significantly, leading to its removal on postoperative day 2. The drain was removed on day 5, and the patient's diet was advanced successfully. He was discharged after 13 days in the hospital.

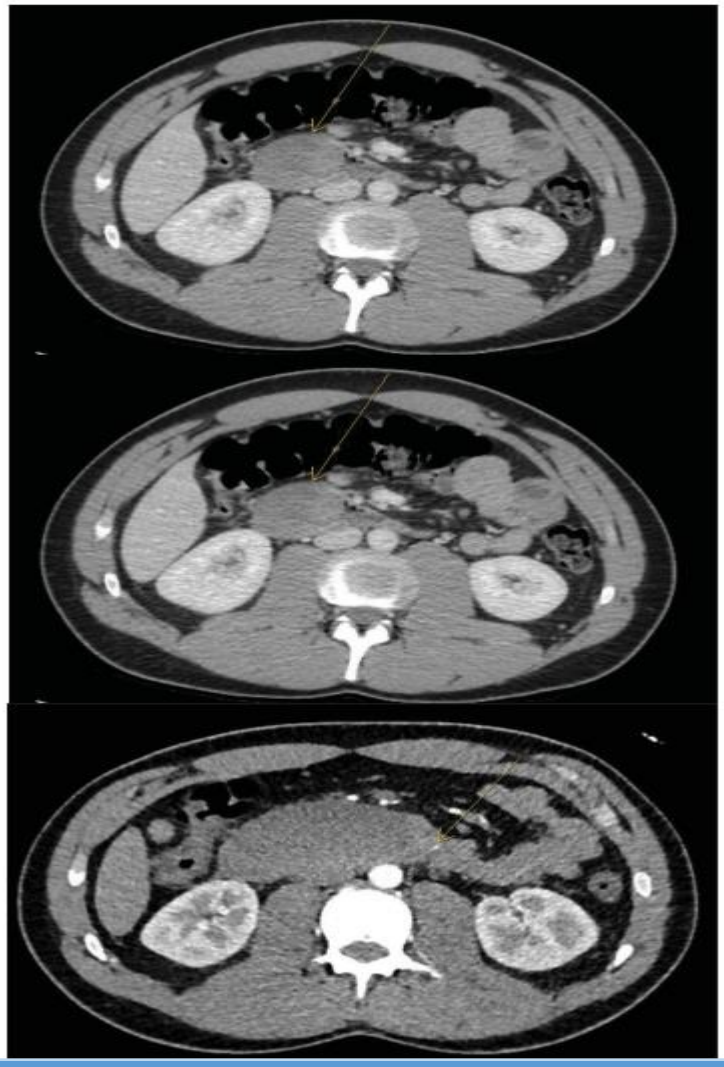

Figure 1. CT abdominal/pelvic images on days 1 and 3 showed increased size of the duodenal hematoma, while a CT angiogram on day 5 revealed a focal area of active extravasation.

DISCUSSION- The present case demonstrated that the patient had no identifiable risk factors, underscoring the unpredictable nature of this complication. This finding contrasts with pediatric populations, where incidence rates are approximately 1 in 2000.[3] While standard management has often focused on conservative approaches—like nasogastric decompression and parenteral nutrition—the present case incorporated arterial embolization to limit hematoma expansion, followed by surgical intervention due to the patient's worsening condition. This contrasts with existing literature, where most adult cases were managed conservatively, with only a few requiring more invasive interventions.[4]

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CONCLUSION- This case report described an unusual case of duodenal hematoma in a 21-year-old man following routine endoscopic duodenal biopsies, without clear risk factors. The patient presented over 72 hours post-procedure, and the hematoma enlarged more than three-fold within the first 24 hours of hospitalization, resulting in persistent duodenal obstruction. Management included arterial embolization to prevent further hematoma expansion, followed by laparoscopic evacuation. Future research on minimally invasive techniques for duodenal hematoma management may help reduce complications and recovery time.

Duodenal hematoma can arise unexpectedly after endoscopy; prompt arterial embolization and laparoscopic evacuation are effective treatments.

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