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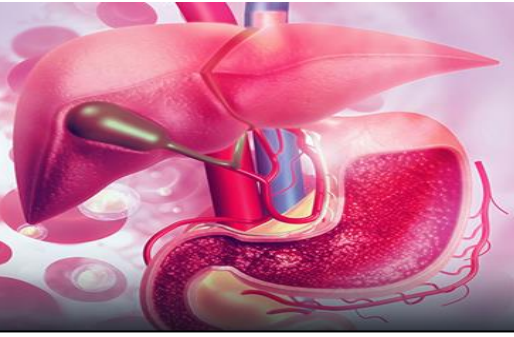

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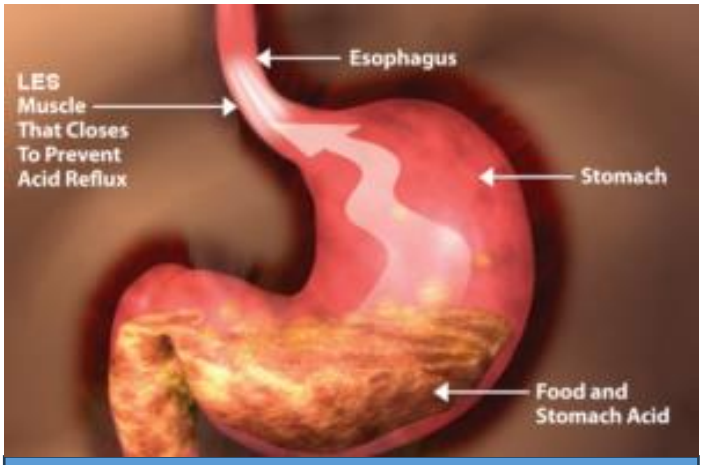
Analysis of the gastroesophageal reflux disease prevalence from infancy through young adulthood

INTRODUCTION

Congenital diaphragmatic hernia (CDH) is a severe, potentially life-threatening defect of the diaphragm, with survival rates ranging from 60-81% in high-volume centers, influenced by the size of the defect and the presence of associated congenital cardiac anomalies.[1] One of the most common long-term complications in CDH survivors is gastroesophageal reflux disease (GERD), particularly within the first year of life. The pathogenesis of GERD in CDH patients is multifactorial, involving intrinsic esophageal motility dysfunction, anatomical changes such as intrathoracic positioning of the esophagus and stomach, and impaired gastroesophageal junction (GEJ) function. Additionally, increased intra-abdominal pressure following CDH repair may exacerbate the risk of GERD by distorting the GEJ and elevating the pressure gradient. While the prevalence of GERD decreases over time, its long-term course remains poorly understood, with some cases progressing to more severe conditions such as Barrett's esophagus or esophageal adenocarcinoma.[2] This study aims to assess the prevalence and long-term trajectory of GERD in CDH patients by analyzing pH-multichannel intraluminal impedance (pH-MII) and endoscopic data collected through a longitudinal follow-up program.



Gastroesophageal Reflux Disease

METHODS

A retrospective study was conducted on CDH survivors who underwent 24-hour pH-MII measurements between January 2013 and December 2020, including infants (5-14 months, corrected for prematurity) and children (7-9 years) as part of routine follow-up care. Additionally, medical records of CDH patients who underwent esophagogastroduodenoscopy (EGD) between June 2015 and June 2022 were reviewed. Baseline data, including sex, gestational age, birth weight, ECMO use, hernia side, type of repair, and associated anomalies, were extracted. Clinical characteristics such as anti-reflux medication use, previous fundoplication, GERD symptoms, and BMI were also collected. The pH-MII protocol involved discontinuing acid suppression therapy before testing, and data were analyzed through automated and manual revision for reflux parameters. For adolescents and adults, endoscopic surveillance followed standard protocols, including biopsy for esophagitis and Barrett's esophagus. Data analysis involved descriptive statistics, non-parametric tests, and continuous and categorical variables comparisons using SPSS version 25.

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RESULTS

A total of 155 CDH children born between January 2012 and December 2019 were identified, with 85.2% surviving. Among the survivors, 109 infants were eligible, and 30 were included in the study. Prior to testing, 80% were using anti-reflux medication, and gastrointestinal and respiratory symptoms were reported in 16.7% and 3.3%, respectively. pH results were abnormal in 10% of infants, 33.3% had indeterminate results, and 56.7% had normal results. Two-thirds of infants experienced symptoms during testing, but only 30% had a positive symptom index (SI) and 50% had a positive symptom association probability (SAP). In children aged 7-9 years, 36 were included, with 5.6% using anti-reflux medication and 44.4% reporting symptoms prior to testing. pH results were abnormal in 5.6%, and 86.1% had normal results. 26.7% of children experienced symptoms during testing, with 11.1% having positive SI and SAP. Among adolescents (median age 17 years), 47% reported reflux-like symptoms. Endoscopic findings were normal in 57%, with 7% showing macroscopic esophagitis (Table 1). Histological abnormalities were found in 80%, with 16% showing columnar metaplasia, and 7% diagnosed with Barrett's esophagus (BE). No dysplasia was reported, and no correlation was found between alcohol or smoking and GERD or BE. Two patients with BE had prior pH-metry diagnoses and were monitored with PPI treatment. Follow-up endoscopies showed stable or reduced BE without dysplasia.

Group	Infants (5-14 months) (n=30)	Children (7-9 years) (n=36)	Adolescents (17-31 years) (n=30)
Anti-reflux Medication	80%	5.6%	17%
Symptoms Before Testing	16.7% GI, 3.3% Respiratory	44.4% GI, 2.8% Respiratory	47% reflux-like symptoms
Abnormal pH-MII Results	10%	5.6%	N/A
Normal pH Results	56.7%	86.1%	N/A
Reflux Hypersensitivity	10%	5.6%	N/A
Functional Symptoms	6.6%	27.7%	N/A
Endoscopy Findings	N/A	N/A	57% Normal, 7% Esophagitis, 7% BE

Table 1. Demographic variables of participating children

DISCUSSION

This study found that GERD prevalence remained low (~10%) in CDH survivors across all age groups, with many participants experiencing symptoms despite normal pH-MII results, aligning with previous studies such as Caruso et al., who reported high GERD rates in CDH survivors with intrathoracic stomachs.[3] Additionally, a

significant proportion of adolescents and young adults exhibited microscopic esophagitis, which was consistent with Morandi et al.'s findings, but without progression to Barrett's esophagus or dysplasia.[4]

CONCLUSION

This study demonstrated that GERD prevalence in CDH survivors is low across all ages, despite the presence of symptoms in many patients. This finding suggested that standardized PPI therapy is unnecessary. It was recommended for a shift toward a symptom-driven approach in diagnosing and managing GERD in CDH patients, with symptom assessment primarily based on reports from caregivers or patients.

GERD is rare in CDH survivors, so routine PPI therapy is unnecessary; a symptom-driven approach based on patient or caregiver reports is recommended.

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Impact of Second-hand Smoke on Peptic Ulcer Disease and gastroesophageal reflux disease in Non-Smokers

INTRODUCTION

Peptic ulcer disease (PUD) is characterized by damage to the mucosal lining of the stomach and/or duodenum, often extending to the muscle layer due to gastric acid production.[1] The Global Burden of Disease Study estimated approximately 8 million cases of PUD worldwide in 2019.[2] Risk factors for PUD include high body mass index (BMI), smoking, stress, NSAIDs use, and Helicobacter pylori infection, with potential complications such as bleeding, gastric outlet obstruction, and perforation. Gastroesophageal reflux disease (GERD), caused by the reflux of stomach contents into the esophagus, is estimated to affect 5.0 per 1,000 person-years globally. Smoking is a well-established risk factor for both PUD and GERD, contributing to the increased prevalence and severity of these conditions. While the association between active smoking and these diseases is well-documented, the impact of second-hand smoke (SHS) exposure remains underexplored. Despite its association with numerous health issues, the role of SHS in the development of PUD and GERD is not fully understood.[3] So, this study

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investigated the relationship between SHS exposure and the risk of PUD and GERD in a large cohort of never-smokers from the Taiwan Biobank (TWB).

METHODS

In this population-based study, a total of 88,297 participants (18,595 males, 69,702 females, mean age 50.1 ± 11.0 years) were included after excluding 33,067 smokers. The study recorded variables including body height, weight, blood pressure (measured after a 1–2-minute break, with three readings averaged), and medical history of hypertension, diabetes, peptic ulcer disease (PUD), and gastroesophageal reflux disease (GERD). Additional health measures included estimated glomerular filtration rate (eGFR), triglycerides, cholesterol (HDL-C/LDL-C), haemoglobin, uric acid, and fasting glucose. Smoking and second-hand smoke (SHS) exposure were assessed through questionnaires, categorizing participants by smoking status (never, ex, or active smokers) and SHS exposure duration. Statistical analyses, including independent t-tests, chi-square tests, and multivariable logistic regression, were performed to identify factors associated with PUD and/or GERD, with a p-value of <0.05 considered statistically significant.

RESULTS

In a cohort of 88,297 participants, 13.5% had peptic ulcer disease (PUD) and 13.3% had gastroesophageal reflux disease (GERD). PUD was associated with older age, male sex, diabetes mellitus (DM), hypertension, alcohol and betel nut use, higher systolic blood pressure, fasting glucose, hemoglobin, triglycerides, total cholesterol, LDL-C, and lower eGFR. GERD was linked to older age, female sex, DM, hypertension, alcohol use, elevated fasting glucose, triglycerides, total cholesterol, LDL-C, lower eGFR, and uric acid. Second-hand smoke (SHS) exposure was independently associated with an increased risk of PUD (OR=1.166, 95% CI=1.084–1.254) and GERD (OR=1.131, 95% CI=1.053–1.216). Exposure to SHS ≥1 hour/week further elevated the risk of PUD (OR=1.232, 95% CI=1.121–1.355) and GERD (OR=1.200, 95% CI=1.093–1.319), with never-smokers exposed to SHS showing significant associations with both conditions (Figure 1).

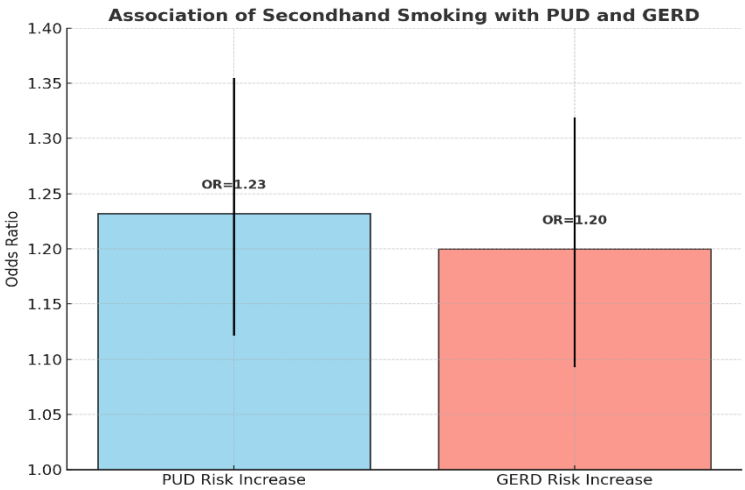

Figure 1. Association Between Second-hand Smoke Exposure and Risk of PUD and GERD

DISCUSSION

This population-based study revealed that second-hand smoke (SHS) exposure significantly increased the risks of peptic ulcer disease (PUD) and gastroesophageal reflux disease (GERD), with ≥1 hour of weekly exposure linked to 1.23-fold and 1.20-fold higher risks, respectively. These findings align with prior studies demonstrating the adverse effects of tobacco exposure, including delayed ulcer healing.[3] Additionally, the association between

SHS and GERD was consistent with studies on active smoking, which highlighted decreased gastroesophageal sphincter pressure, increased acid exposure, and impaired acid clearance as contributing factors.[3]

CONCLUSION

This large population-based study demonstrated significant associations between second-hand smoke (SHS) exposure and increased risks of peptic ulcer disease (PUD) and gastroesophageal reflux disease (GERD), with ≥1 hour per week of SHS exposure linked to 1.23- and 1.20-fold higher risks, respectively.

Second-hand smoke (SHS) exposure of ≥1 hour per week increases the risk of peptic ulcer disease (1.23x) and GERD (1.20x).

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Investigation of the Bidirectional Association between Inflammatory Bowel Disease and Type 1 Diabetes

INTRODUCTION

Inflammatory bowel disease (IBD) is a chronic inflammatory disorder of the gastrointestinal tract with an uncertain etiology. Although IBD predominantly affects young adults, it can occur at any age, with approximately 25% of cases presenting before 20 years of age.[1] The subtypes of IBD include Crohn's disease (CD), ulcerative colitis (UC), and IBD-unclassified (IBD-U), where the subtype remains uncertain. Type 1 diabetes (T1D) is an autoimmune disorder characterized by the destruction of pancreatic β-cells, leading to lifelong insulin deficiency. While T1D affects approximately 0.1% of the global population, its prevalence in Sweden is around 0.5%. Both conditions are thought to result from complex interactions involving immune hyperactivation, metabolic disruption, and gut microbiota imbalances. The co-occurrence of IBD and T1D has been linked to poor clinical outcomes, but evidence on their bidirectional associations remains limited.[2] So, this study aimed to explore the bidirectional relationship between IBD and T1D using Swedish national registers through a nationwide matched cohort and case-control study.

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METHODS

This study leveraged data from the ESPRESSO cohort and the Swedish National Patient Register (NPR) to investigate the relationship between IBD and T1D. IBD patients were identified through ICD codes and biopsy data, with the diagnosis confirmed when both criteria were met. Two comparison groups were used: individuals from the general population and IBD-free full siblings. T1D diagnosis was confirmed through ICD codes, with a focus on patients aged ≤28 years for incident T1D. Cohort and case-control studies were conducted, with hazard ratios (HRs) and odds ratios (ORs) calculated, adjusting for covariates such as socioeconomic status, country of birth, and history of autoimmune diseases. Sensitivity analyses addressed potential biases, including detection bias, surveillance bias, and reverse causation, and examined the impact of IBD treatments. The study also included subgroup analyses based on factors such as sex, age at diagnosis, and IBD phenotype. Data were analyzed using SAS, Stata, and R, with statistical significance set at $p \leq 0.05$.

RESULTS

In this study, 20,314 patients with IBD and 99,200 matched reference individuals were analyzed. The incidence of T1D was higher in IBD patients (0.6%) compared to controls (0.4%), with an adjusted hazard ratio (aHR) of 1.58 (95% CI: 1.27–1.95). This increased risk was driven by UC (aHR = 2.02), while CD showed no significant association. Subgroup analyses revealed a stronger association in males and individuals aged 18-28. In sibling comparison analyses, IBD patients also had a higher hazard of T1D (aHR = 1.44), particularly those with UC (aHR = 1.90) (Figure 1). In the case-control study, 1.2% of IBD patients had a prior diagnosis of T1D, with higher odds of prior T1D in IBD patients compared to controls (aOR = 1.36), particularly in UC (aOR = 1.36) and IBD-U (aOR = 1.63). Sensitivity analyses confirmed the robustness of these findings, with associations observed across different subgroups and IBD subtypes.

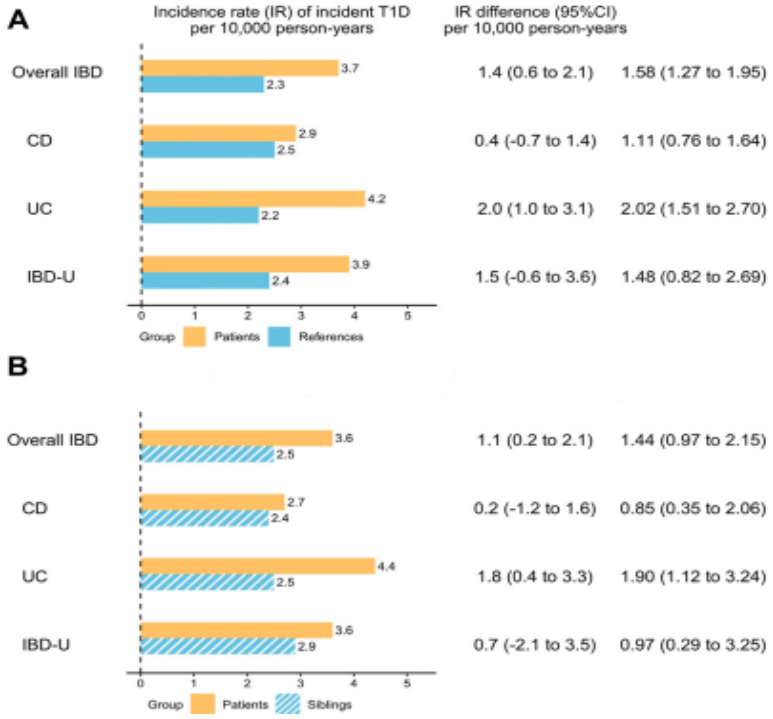

Figure 1: Incidence rates for type 1 diabetes (T1D) in IBD patients compared to matched references (A) and siblings (B), with 95% CI. (Model 2). CD: Crohn's disease; IBD-U: IBD-unclassified; UC: ulcerative colitis.

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DISCUSSION

In this nationwide matched cohort and case–control study, it was found that patients with IBD diagnosed before age 28 had a 58% increased hazard of developing T1D, while IBD patients (without age restrictions) had 36% higher odds of prior T1D. This aligns with previous studies, though the incidence of T1D in this cohort was lower than that of a Danish study, likely due to the younger age of our participants.[3] Additionally, the bidirectional association was more pronounced in UC and PSC patients, which was consistent with prior studies showing stronger association between T1D and UC.[4]

CONCLUSION

Patients with IBD have a modestly increased risk of developing T1D, both before and after IBD diagnosis, with a stronger association in adult-onset IBD, males, those with UC, and patients with PSC. This bidirectional relationship may be partially independent of shared genetics and early environmental factors.

IBD patients, especially those with adult-onset, UC, or PSC, have a slightly higher risk of developing T1D, potentially independent of shared genetics and environmental factors.

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Case Report on Acute Cholecystitis Due to Taeniasis

INTRODUCTION

Taeniasis is a pathological condition caused by the infestation of humans with various species of parasitic tapeworms from the *Taenia* genus, including *Taenia solium*, *Taenia saginata*, and *Taenia asiatica*.[1] Taeniasis is caused by the ingestion of undercooked pork (*T. solium* and *T. asiatica*) or beef (*T. saginata*), containing cysticerci that mature into adult tapeworms in the intestine. It

Figure 1. Formalin-fixed gallbladder specimen demonstrating edema in the gallbladder wall with a tapeworm present within the lumen.

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is more common in rural areas with poor sanitation and close human-animal contact. Many infected individuals are asymptomatic and unaware of the infection until proglottids appear in feces or on clothing. Symptomatic cases can present with gastrointestinal issues like nausea, abdominal pain, and diarrhea. Though it primarily affects the small intestine, taeniasis can occasionally spread to extraintestinal sites, including the appendix, pancreas, liver, and biliary system.[2] This report describes a rare case of acute cholecystitis caused by a *T. saginata* or *T. asiatica* infection in a 47-year-old woman, accompanied by a review of relevant literature.

CASE PRESENTATION

A 47-year-old woman presented to Hospital with severe epigastric pain, nausea, and vomiting lasting 6 hours. She had no fever, diarrhea, or recent history of unusual eating habits or travel. Physical examination revealed tenderness in the epigastric and right upper quadrant regions without signs of peritonitis. Laboratory results showed mild direct hyperbilirubinemia and transaminitis, but normal white blood cell and platelet counts. Abdominal ultrasonography suggested acute cholecystitis, with multiple gallstones and an increased gallbladder wall thickness.

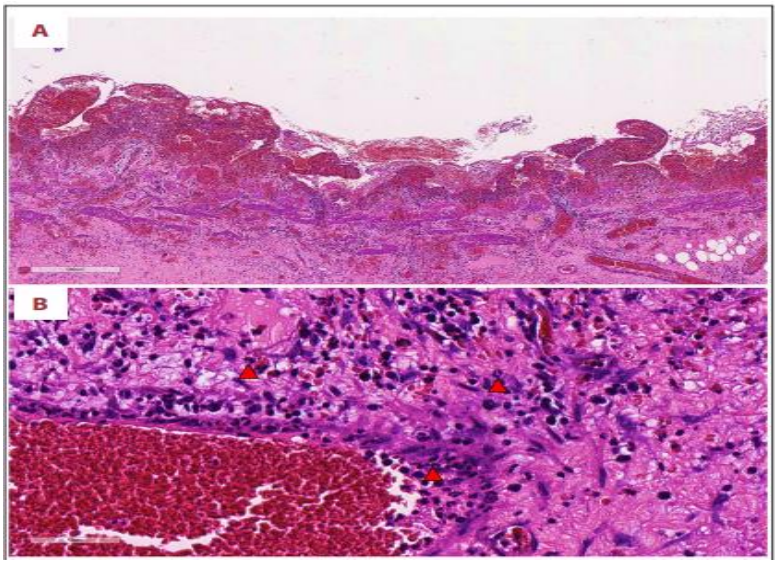

Figure 2. (A) Low power view of gallbladder mucosal ulceration with fibrin exudate (H&E, 40×). (B) High power view showing neutrophil infiltration (H&E, 400×)

The patient was prescribed intravenous omeprazole, buscopan, and ceftriaxone as a preoperative regimen, and elective cholecystectomy was scheduled. The resected gallbladder, measuring 9×3.5×2.5 cm, had a smooth serosal surface, irregular brown mucosa, and a wall thickness of 0.5 cm. Surprisingly, a 70 cm-long tapeworm was found within the gallbladder lumen (Figure 1**).** Histopathological examination of the gallbladder revealed edematous wall changes and necrosis, with acute inflammation (Figure 2). Further examination of the tapeworm showed a thick tegument, smooth muscle layers, and calcareous corpuscles. The specimen was preserved in formalin and sent for further analysis, revealing the potential species as *Taenia saginata* or *Taenia asiatica* based on morphological characteristics, such as uterine branch and twig ratios. Microscopic Examination of Tapeworm with Uterine Branches Following the cholecystectomy, the patient reported relief from abdominal pain. She was prescribed 500 mg of niclosamide (4 tablets) for helminth treatment and discharged after 7 days of postoperative care.

DISCUSSION

Gallbladder taeniasis caused by *T. saginata* was identified in this case, with pathology revealing mucosal necrosis and acute cholecystitis, where the findings align with the observations of Yu et al., who reported ulceration and inflammation in the gallbladder.[3] The species identification was conducted using morphological methods, specifically Semichon's acetic carmine staining, which provided distinct features despite challenges posed by

formalin fixation. This approach contrasts with PCR-based molecular methods, which offer higher species identification accuracy, as demonstrated in studies from Tibet, where PCR revealed higher homology rates for *T. saginata* isolates.[4]

CONCLUSION

This case highlights acute cholecystitis in a Thai female patient caused by *T. saginata* or *T. asiatica*. The findings emphasize the clinical significance of symptomatic infections due to adult *Taenia* parasites, which, although rare, can be challenging to diagnose based solely on imaging. Clinicians should maintain a high index of suspicion for *Taenia* spp. infections in the biliary system, especially in endemic areas where these infections are more prevalent.

Taenia infections should be considered in acute cholecystitis cases, especially in endemic areas, as they are difficult to diagnose with imaging.

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