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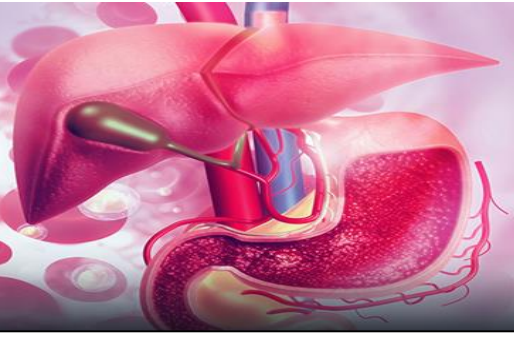

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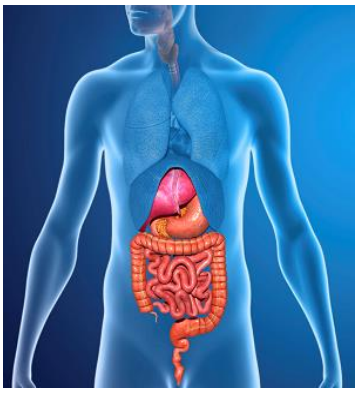
# Correlation and temporal sequence between stroke-associated pneumonia and gastrointestinal bleeding after acute ischemic stroke

**INTRODUCTION-**Ischemic stroke accounts for 80% of all strokes worldwide. Complications, both neurological and non-neurological, have an impact on outcomes in acute ischemic stroke. Non-neurological consequences of acute ischemic stroke include gastrointestinal bleeding and infection. Gastrointestinal bleeding in patients suffering from acute ischemic stroke is typically upper gastrointestinal bleeding. Pneumonia and urinary tract infections are the most common infections in acute ischemic stroke.[1] Stroke-associated pneumonia (SAP) and gastrointestinal bleeding (GIB) are both major medical consequences of stroke. The previous study found a substantial link between SAP and GIB following a stroke. However, little is known regarding SAP and GIB's chronological sequence. So, the current study aimed to confirm the relationship and elucidate the temporal sequence of SAP and GIB following an ischemic stroke.[2]



**Gastrointestinal bleeding**

**METHODS-**This was a prospective cohort study and included 1129 patients with ischemic stroke. Patients were eligible for this study provided they met the following conditions: (1) Age 18 or older; (2) Hospitalized with a diagnosis of acute ischemic stroke (AIS) based on World Health Organization criteria; (3) Confirmed by head computed tomography and/or brain magnetic resonance imaging; and (4) Time from stroke onset to hospitalization within 7 days. Patients with ischemic stroke from the in-hospital Medical Complications after Acute Stroke study were examined. Data on SAP and GIB occurrences throughout hospitalization, as well as time intervals between stroke start and SAP and GIB diagnosis, were gathered. Multiple logistic regression was performed to determine the relationship between SAP and GIB. The time intervals between the beginning of the stroke and the SAP and GIB diagnosis were compared using the Kruskal-Wallis test.

**RESULTS-**This study shown that there were 899 (79.6%) males and a mean age of 58.7±2.5. About 14 days (IQR 11–16) was the median stay in the hospital. Individuals with GIB were found to be more likely than those without it to have a history of atrial fibrillation (17.0% vs. 5.6%, P=0.001), a higher NIHSS score (13 vs. 4, P<0.001), to have been hospitalized for longer (21 days vs. 13 days, P<0.001), and to have experienced higher in-hospital mortality (8.5% vs. 0.2%, P<0.001). During their hospital stay, 47 patients (4.3%; 95% CI, 3.0-5.3%) acquired GIB and 86 patients (7.6%; 95% CI, 6.1-9.2%) developed SAP. The results of the unadjusted regression model showed a significant correlation between SAP and the development of GIB following AIS (OR=17.17; 95% CI, 9.17–32.13; P<0.001). SAP remained substantially linked to the progression of GIB from model 2 to model 4 even after adjusting for age, gender, and other possible confounders. Compared to patients without SAP, the odds ratio in model 4 for the development of GIB in patients with SAP was 5.10 (95% CI, 2.01–12.95). Only 27 (31.4%) of the patients with SAP and 11 (23.4%) of the patients with GIB received their diagnoses during the first 48 hours after stroke onset. Over 79.1% and 61.7% of patients with PNE and GIB had equivalent sequelae identified up to 7 days after the stroke started (Figure 1). Overall, after an acute stroke, the median time from

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stroke onset to SAP diagnosis was substantially shorter than the duration from GIB (4 days vs. 5 days; P=0.039). Only patients with NIHSS ≥16 showed a significant difference when the patients were categorized by the site of the infarct and the severity of their stroke.

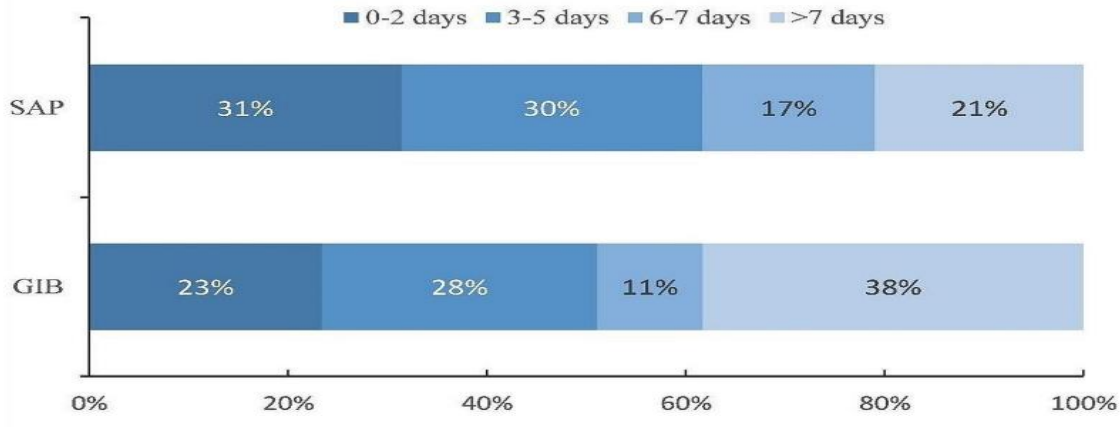


Figure 1. The percentage distribution of days in patients with pneumonia and gastrointestinal bleeding from the time of ischemic stroke to the identification of complications.

**DISCUSSION -**The incidence of GIB and SAP following a stroke was 4.3% and 7.6%, respectively, during the hospital stay. The study also revealed that SAP was linked to GIB following an ischemic stroke and that SAP was diagnosed earlier than GIB. According to this study, SAP in ischemic stroke patients may be a risk factor or risk marker for GIB.[2] Similar to this study, Rumalla K et al. showed that GIB was substantially related to SAP when they looked at its relationship to other in-hospital problems in patients with AIS.[3]

**CONCLUSION-**The current study concluded that after a stroke, GIB was linked to SAP, and the median time between the stroke's onset and SAP diagnosis was earlier than the time between GIB and SAP diagnoses. When stroke patients receive an SAP diagnosis, it should be given more consideration and precautions taken to avoid GIB.

**Stroke-associated pneumonia (SAP) began earlier than gastrointestinal bleeding (GIB), and it was associated with GIB following an ischemic stroke.**

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# Investigation of the association between gastroesophageal reflux disease and Sjogren's syndrome through a bidirectional two-sample Mendelian randomization (MR) study

**INTRODUCTION-** Sjogren's syndrome (SS) is a chronic autoimmune disease that predominantly affects salivary and lacrimal glands, leading to dryness of the mouth and eyes. It can also involve other organs, causing a variety of symptoms across multiple systems.[1] One of the most prevalent gastrointestinal (GI) disorders currently known as gastroesophageal reflux disease (GERD), is characterized by the reflux of acid into the esophagus, which can cause heartburn and regurgitation which further worsen the condition of the patient.[2] GERD is a common complication in SS patients, with a significant percentage of them experiencing reflux and heartburn. The relationship between SS and GERD has been a subject of interest, but observational studies have shown inconsistent results. This study aimed to explore the causal relationship between GERD and SS using a bidirectional Mendelian randomization (MR) approach.[1]

**METHODS-**This was a bidirectional two-sample Mendelian randomization study and used genetic variants as instrumental variables to estimate the causal effect of GERD on SS and vice versa. Genetic data were obtained from two large genome-wide association studies (GWAS). GERD-related data were sourced from the IEU Open GWAS project, involving 602,604 participants (129,080 patients and 473,524 controls). SS-related data were acquired from the FinnGen biobank, comprising 392,423 samples (2,495 patients and 389,928 controls). Single nucleotide polymorphisms (SNPs) significantly associated with GERD and SS were selected as instrumental variables. The SNPs were filtered to ensure they met the MR assumptions of relevance, independence, and exclusion restriction. The F-statistic was calculated to assess the strength of the instrumental variables. The inverse-variance weighted (IVW) method was the primary statistical approach used, supplemented by MR–Egger regression, weighted median, simple mode, and weighted mode methods. Heterogeneity and sensitivity analyses were performed using Cochran's Q statistic, MR-PRESSO, and the Steiger test to validate the direction of causality.

**RESULTS-**The MR analysis demonstrated a significant positive association between genetically predicted GERD and the risk of SS. Specifically, the inverse-variance weighted (IVW) method showed that GERD increased the odds of developing SS with an odds ratio (OR) of 1.3279 (95% confidence interval [CI] 1.0312–1.7099, P = 0.0280). This suggested that individuals with genetic predispositions for GERD were more likely to develop SS. Additional sensitivity analyses, including MR–Egger regression, weighted median, simple mode, and weighted mode methods, consistently supported this finding. These methods accounted for potential biases due to pleiotropy, where a single genetic variant influences multiple traits. The heterogeneity analysis using Cochran's Q statistic indicated no significant heterogeneity (P>0.05), suggesting that the genetic instruments used were appropriate and the results were reliable. MR-PRESSO did not detect any significant outliers, further validating the robustness of the association. The Steiger Test confirmed the correct direction of causality, reinforcing the conclusion that GERD increases the risk of SS. Conversely, the analysis did not reveal a significant causal effect of SS on GERD. The IVW method showed an OR of 1.0024 (95% CI 0.9651–1.0412, P = 0.8995), indicating no increased risk of GERD in individuals with SS (Table 1). Sensitivity analyses using MR–

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Egger, weighted median, simple mode, and weighted mode methods consistently produced non-significant results, suggesting that SS does not have a significant impact on the development of GERD. Heterogeneity analysis showed no significant heterogeneity ($P > 0.05$), and MR-PRESSO did not detect any outliers. The Steiger test confirmed the direction of causality, ensuring that the lack of association was correctly interpreted.

| Exposure | Outcome | nSNP | Method | Beta | *P*-value | OR | 95%CL |
|---|---|---|---|---|---|---|---|
| Sjogren syndrome | GERD | 3 | Inverse variance weighted | 0.0024 | 0.8995 | 1.0024 | (0.9651, 1.0412) |
| Sjogren syndrome | GERD | 3 | MR Egger | 0.0565 | 0.8023 | 1.0582 | (0.7492, 1.4945) |
| Sjogren syndrome | GERD | 3 | Weighted median | 0.0097 | 0.6828 | 1.0097 | (0.9639, 1.0577) |
| Sjogren syndrome | GERD | 3 | Simple mode | 0.0150 | 0.6589 | 1.0151 | (0.9586, 1.0749) |
| Sjogren syndrome | GERD | 3 | Weighted mode | 0.0150 | 0.6556 | 1.0151 | (0.9592, 1.0743) |

Table 1. The MR analysis showing the causal effects of SS on GERD

**DISCUSSION-**The findings from this bidirectional MR study provide evidence of a unidirectional causal relationship, where GERD was a risk factor for SS, but SS does not affect the development of GERD.[1] Similar to this study, Chang C et al. showed that GERD was 2.41 times more likely to occur in individuals with SS.[3]

**CONCLUSION-**The current study confirmed GERD increases the risk of developing SS, highlighting the importance of managing GERD symptoms in patients at risk for or diagnosed with SS. However, SS does not appear to have a significant impact on the risk of GERD. These insights can inform clinical practices and guide future research on the interplay between these two conditions.

**This bidirectional two-sample Mendelian randomization study demonstrated that Sjogren's syndrome (SS) does not cause GERD, but rather that GERD was a risk factor for SS. It also confirmed the existence of a causal link between SS and GERD.**

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# Assessment of the Impedance of Esophageal Mucosa in the Diagnosis of Gastroesophageal Reflux Disease

**INTRODUCTION-**Gastroesophageal reflux disease (GERD) is a condition that occurs when stomach contents are refluxed, causing unpleasant symptoms and/or consequences. Globally, the prevalence of GERD is around 14%, however, it varies by location and country.[1] The diagnosis of GERD was challenging due to nonspecific symptoms, heterogeneous clinical presentations, and overlap with other gastrointestinal disorders. Traditional diagnostic methods include clinical presentation, therapeutic response, endoscopic evaluation, and prolonged monitoring of gastroesophageal reflux. A new metric, mean nocturnal baseline impedance (MNBI), assessed during multichannel intraluminal impedance-pH monitoring (MII-pH), offered insights into mucosal integrity. Previous studies have indicated that basal impedance correlated with esophageal mucosal health, decreasing with acid exposure. However, MII-pH requires prolonged use of an uncomfortable transnasal catheter. This study evaluated the effectiveness of a new catheter for direct mucosal impedance (MI) measurement during upper gastrointestinal endoscopy (UGE) in diagnosing GERD.[2]

**METHODS-**This study comprised 60 patients with typical GERD manifestations (heartburn and/or regurgitation). Patients were classified into two groups based on their acid exposure time (AET): group A (AET <4%, GERD diagnosis excluded) and group B (AET ≥4%, probable GERD diagnosis, inconclusive or confirmed). The patient received high-resolution esophageal manometry, 24-hour multichannel intraluminal impedance-pH monitoring, an upper gastrointestinal endoscopy, and mucosal impedance measurements. During gastrointestinal endoscopy, mucosal impedance was measured at 2, 5, 10, and 18 cm above the esophagogastric junction (EGJ) using a catheter designed based on devices described in the literature during the last decade. The chi-squared test was used to determine if there was a significant relationship between the qualitative variables and AET (<4% × ≥4%). Wilcoxon or Kruskal-Wallis tests were used to assess the relationship between quantitative and qualitative factors.

**RESULTS-**Sixty patients were included, with 28 in group A (AET <4%) and 32 in group B (AET ≥4%). No significant differences were found between groups regarding age, gender distribution, body mass index, comorbidities, smoking status, and most symptoms. However, eructation was significantly more common in group B. No significant differences were observed in EGJ contractile integral, integrated relaxation pressure, and distal contractile integral values between groups. Figure 1 showed the MI measurements evaluated during UGE. Although EGJ hypotonia and ineffective esophageal motility were more common in group B, these differences were not statistically significant. Mucosal impedance was significantly lower in group B at 2 cm (2264.4 Ω ± 1099.0 vs 4575.0 Ω ± 1407.6 in group A) and 5 cm above the EGJ (4221.2 Ω ± 2623.7 vs 5888.2 Ω ± 2529.4 in group A). No significant differences were found at 10 cm and 18 cm above the EGJ. MI at 2 cm >2970 Ω demonstrated a sensitivity of 96.4% and specificity of 87.5% for excluding GERD.

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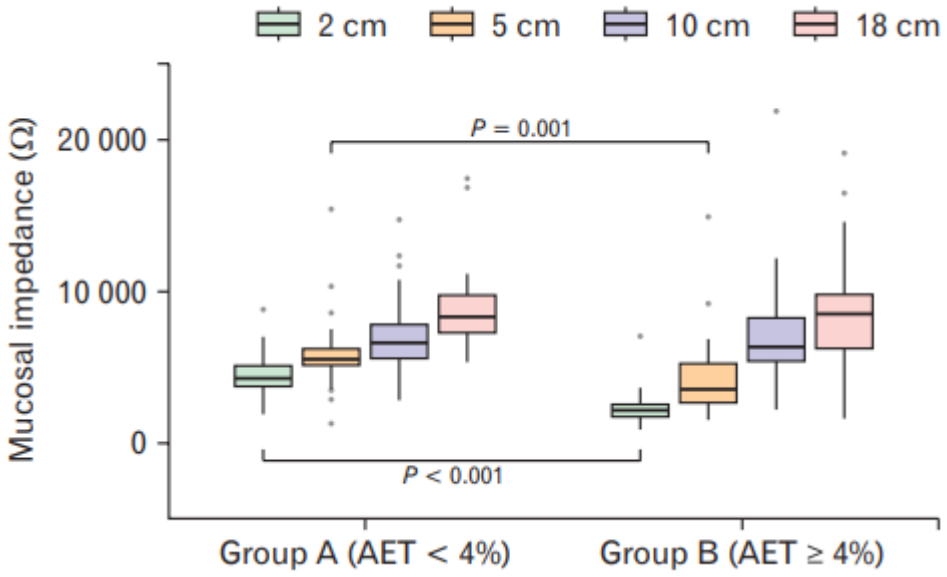


Figure 1. Box plot of esophageal mucosal impedance measured at various points above the esophagogastric junction based on acid exposure time (AET; <4% vs ≥4%).

**DISCUSSION-**This study shown that during endoscopy, mucosal impedance may be directly measured, which was a simple and promising way to diagnose GERD. Mucosal impedance readings were lower in people with aberrant acid exposure times than in those with normal acid exposure times.[2] Similar to this study, Lei WY et al. showed that endoscopic MI testing measurement was a useful technique for the rapid differentiation of esophageal problems during endoscopy. It might be used as a surrogate to anticipate treatment response and guide therapy in clinically challenging settings, in addition to being valuable tool for the diagnosis of GERD.[3]

**CONCLUSION-**The current study concluded that direct measurement of esophageal mucosal impedance during UGE was a promising diagnostic tool for GERD. It effectively distinguishes between patients with normal and abnormal acid exposure times, providing a less invasive and more comfortable alternative to traditional methods. Further research and development of this technique could enhance GERD diagnosis and patient care.

**During endoscopy, mucosal impedance may be directly measured, which was a simple and promising way to diagnose GERD.**

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# Case report on gastrointestinal basidiobolomycosis- an unexpected instance

**INTRODUCTION-**The environmental saprophytic fungus Basidiobolus ranarum, which is found around the world in soil, decomposing organic debris, and the digestive tracts of fish, amphibians, reptiles, and insectivorous bats, is the source of the infection known as gastrointestinal basidiobolomycosis (GIB). The main routes of transmission seem to be minor trauma, local injection, and insect bites; nonetheless, human gut infections are uncommon for Basidiobolus spp.[1] Since GIB is an extremely uncommon fungal infection of the gastrointestinal tract, individuals exhibiting symptoms and indicators of gastrointestinal illness are unlikely to have it suspected. GIB can develop aggressively and result in serious lifetime morbidities if it was not identified and treated on time. This case described an unexpected instance of gastrointestinal basidiobolomycosis (GIB), which was misdiagnosed and treated as acute appendicitis at first. Later, it was believed to be a case of either gastrointestinal lymphoma or fulminant inflammatory bowel disease (IBD).[2]

**CASE PRESENTATION-**A 15-year-old girl presented to the emergency department with acute abdominal pain. Her medical history revealed a three-month duration of progressively worsening symptoms, including sharp, colicky abdominal pain primarily in the right and left lower quadrants, radiating to the epigastric area. The pain was accompanied by persistent vomiting, non-bloody watery diarrhea, fever with night sweats, significant loss of appetite, and a weight loss of 15 kilograms over the three months. Initially, at another hospital, she was diagnosed with acute appendicitis and underwent a laparoscopic appendectomy. However, this intervention did not alleviate her symptoms, which continued to worsen post-surgery. The lack of improvement prompted her family to seek further medical care at our facility. Upon arrival, her physical examination revealed a pale, thin adolescent with a Body Mass Index (BMI) of 12.5, indicating severe malnutrition. Her vital signs were stable at that time. Abdominal examination showed a soft, non-distended abdomen with mild tenderness in the left lower quadrant but no palpable organomegaly. Other systemic examinations did not reveal any abnormalities. Laboratory investigations demonstrated leukocytosis with a white blood cell (WBC) count of 19.90 x 10^9/L, marked eosinophilia (15.4%, absolute count 2.50 x 10^9/L), and microcytic hypochromic anemia with a hemoglobin level of 69 g/L. Additionally, she had hypoalbuminemia (12.77 g/L) and elevated inflammatory markers, including an erythrocyte sedimentation rate (ESR) of 107 mm/h and a C-reactive protein (CRP) level of 252.00 mg/L. Liver function tests revealed mildly elevated alkaline phosphatase (ALP) and gamma-glutamyl transferase (GGT). A computerized tomography (CT) scan of the abdomen revealed non-obstructive wall thickening with aneurysmal lumen dilatation in the sigmoid colon, descending colon, cecum, and terminal ileum, accompanied by retroperitoneal lymphadenopathy and a moderate amount of free fluid in the abdomen and pelvis. Colonoscopy showed patchy, non-obstructing mass-like thickening along the colon, especially at the splenic flexure, with areas of haemorrhage and focal necrosis (Figure 1a). Given these findings, the differential diagnosis included inflammatory bowel disease (IBD), tuberculosis (TB), and lymphoma. Colonoscopic biopsies revealed mucosal ulceration, granulation tissue, and necrotizing granulomatous inflammation. However, Quantiferon tests for TB, blood, urine, and stool cultures, and stool tests for parasites and ova were all negative, complicating the diagnostic process. Despite initial treatments, her condition rapidly deteriorated. She developed severe abdominal pain, spiking fevers up to 39.2°C, and tachycardia. Her abdomen became distended, and tympanic, with diffuse

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tenderness, rebound tenderness, and rigidity. Repeat laboratory tests showed an increased WBC count of 27.80 x 10^9/L with a predominance of neutrophils (77.9%). An abdominal X-ray revealed multiple air-fluid levels, indicating possible intestinal obstruction or perforation. An urgent exploratory laparotomy was performed, revealing a perforated transverse colon with peritonitis and a markedly inflamed right colon and cecum. A subtotal colectomy with end ileostomy was carried out. The excised colon showed diffuse edema, thickening, multiple polypoidal elevations, extensive mucosal ulceration, and a perforation in the transverse colon on gross examination. Microscopic examination of the resected colon revealed necrotizing granulomatous inflammation throughout the cecum, ascending, transverse, and descending colon, with almost full-thickness necrosis in the most affected areas. The histopathological evaluation identified broad, septate fungal hyphae consistent with basidiobolomycosis, surrounded by eosinophilic condensation known as the Splendore-Hoeppli phenomenon. These fungal elements were embedded in necrotic debris and infiltrated the blood vessel walls and pericolic fat. Special stains, including Periodic Acid-Schiff (PAS) and Gomori methenamine silver (GMS), highlighted the fungal hyphae, confirming the diagnosis (Figure 1b to 1d). Given the histopathological findings, a diagnosis of perforating intestinal fungal infection due to basidiobolomycosis with associated necrotizing granulomatous inflammation was made. Immediate postoperative antifungal therapy with voriconazole was initiated and continued for 12 months. Eighteen months' post-colectomy, the patient underwent a successful laparoscopic ileostomy closure. Currently, she remains asymptomatic and continues regular outpatient follow-up with periodic abdominal CT scans to monitor for recurrence.

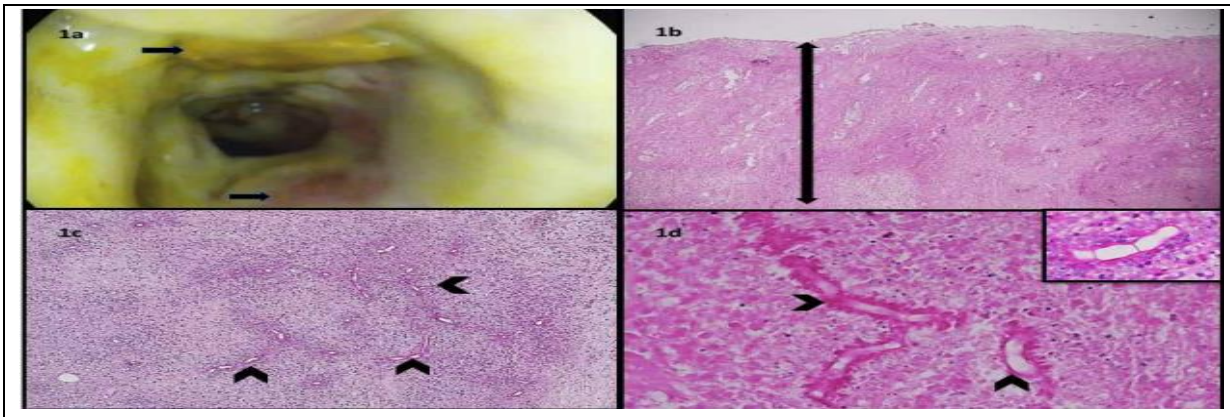


Figure 1. An instance of necrotizing colitis caused by a fungal infection in the intestine called Basidiobolomycosis. (1a) A colonoscopic image revealing elevated polypoidal masses, as well as regions of bleeding and ulceration (arrows). (1b to 1d) A light microscopy photomicrograph of the colectomy revealed severe mucosal ulcers and entire thickness necrosis of the colon wall (double-headed arrow). Basidiobolus ranarum fungal hyphae infiltrate the necrotic colon wall (arrowheads) and form sheath-like eosinophilic condensation, known as the Splendore-Hoeppli phenomenon. The inset displays a greater magnification of the fungal hyphae with septations (original magnifications: 1b, x20; 1c, x100; 1d, x400; inset, x600).

**DISCUSSION-**This case involved a 15-year-old girl who was first diagnosed with acute appendicitis. She was suspected of having inflammatory bowel disease (IBD) or gastrointestinal lymphoma. Her condition quickly deteriorated, resulting in an intestinal perforation requiring subtotal colectomy. After histological investigation, she was diagnosed with GIB. Due to its rarity, the clinical appearance of this condition can be misconstrued, resulting in mismanagement, delayed diagnosis, and secondary consequences.[2] According to the Alsaeed M et

al., a 69-year-old man who had been experiencing fever, weight loss, and abdominal pain for a year presented to the emergency room. The patient's condition was later determined to be gastrointestinal basidiobolomycosis.[3]

**CONCLUSION-** This case of gastrointestinal basidiobolomycosis illustrated the difficulties in diagnosing this rare infection due to its nonspecific presentation and resemblance to other gastrointestinal conditions. Clinicians should consider GIB in differential diagnoses of acute abdomen, particularly in endemic regions. Early histopathological diagnosis and timely antifungal treatment were essential to manage this potentially lethal infection effectively. This report emphasized the need for heightened awareness and clinical suspicion to improve diagnostic accuracy and patient outcomes in cases of GIB.

**GIB should be taken into consideration for patients, particularly those with unpredictable GIT presentations, since prompt diagnosis can have a major impact on the course and related morbidity in these patients by lowering the need for surgery and, consequently, its complications.**

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