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Clinical Impact of Gastrointestinal Multiplex PCR Panels on Relapse Prediction in Ulcerative Colitis: A Prospective One-Year Follow-up Study

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Abstract

Background: This study aimed to evaluate the effect of the gastrointestinal system multiplex PCR panel (GISP) on the relapse-free survival of ulcerative colitis (UC) both active and in remission patients.

Methods: In this prospective single-center study, adult patients with ulcerative colitis were followed for one year. Baseline disease activity was assessed using the Partial Mayo Score (PMS). Gastrointestinal multiplex PCR panel testing was performed, and the association between PCR positivity and relapse-free survival was analyzed using survival models.

Results: At baseline 74.1% of patients were in remission and 25.9% were in the active period. At least one causative agent was detected in 39.7% of stool samples evaluated by GISP. The most common agents were Enteropathogenic *E. coli*(EPEC) (52.2%), *Campylobacter* spp. (13.8%), and Enteroaggregative *E. coli* (EAEC) (11.6%). There was no difference between patients in baseline remission and relapse groups regarding agent positivity.

A relapse developed in 38.5% of the patients within one year follow-up. There was no difference between relapse-free survival in GISP positive and negative patients. In patients who were active at baseline, the relapse-free period was shorter. As a result of the study, the most critical factor affecting the relapse-free period was the partial Mayo score (PMS), which we used to determine the activity status of the disease at baseline.

Conclusion: According to the results of the study, GISP positivity did not affect relapse-free survival. The role of GISP in UC patients' follow-up and predicting clinical outcomes of patients in relapse or remission was limited.

Introduction

Ulcerative colitis (UC) is a chronic inflammatory bowel disease (IBD) with an increasing global prevalence, particularly in industrialized and developing regions(1). Alterations in the intestinal microbiota, characterized by dysbiosis and increased pathogen burden, are central to UC pathogenesis and may influence disease activation and clinical outcomes. Moreover, infectious gastroenteritis and UC flares often present with overlapping clinical features, complicating diagnostic and therapeutic decision-making. Distinguishing between these entities is critical, as management strategies differ substantially. Therefore, stool-based testing for enteric pathogens is a fundamental component of the initial evaluation of UC patients presenting with symptoms suggestive of disease(2).

As an alternative to standard microbiological methods, the gastrointestinal system multiplex polymerase chain reaction (PCR) panels (GISP) are widely used. These tests enable the simultaneous detection of bacterial, viral, and parasitic pathogens from a single stool sample, typically within a period of one hour. However, the high sensitivity of PCR limits its ability to differentiate between viable and nonviable organisms, thereby preventing accurate distinction between infection and colonization. This represents a

major limitation of GISPs (3). Therefore, despite their widespread use, there is still no consensus on the effects of GİSPs on chronic GI diseases such as IBD (2). While some studies have reported that GISP use in patients with IBD is associated with reduced treatment escalation(2,3),others have demonstrated potential drawbacks, including prolonged hospital stays, unnecessary antibiotic exposure, and delays in appropriate diagnosis and treatment due to misattribution of symptoms to infection (2,4). Notably, most available data has been derived from cohorts of symptomatic patients.

This study included patients with UC in both the active disease and remission at baseline prospectively evaluated the clinical impact of GISP. The primary objective was to investigate the association between GISP positivity and relapse-free survival over a year follow-up period.

Methods

Study Design and Population

This single-center, prospective, observational study with a cross-sectional baseline assessment was conducted between October 2022 and May 2024. Adult patients (³18 years old), with a confirmed diagnosis of UC, including both newly diagnosed and those under routine follow-up, were eligible for inclusion. Pregnancy, prior total colectomy, receipt of active immunosuppressive therapy unrelated to ulcerative colitis management, and the presence of acquired immunodeficiency were exclusion criteria.

Definitions

A partial Mayo score ≥3 at the time of admission was considered active disease (5). Relapse (flare) during follow-up was defined as one of the following:
(i) development of a PMS ≥3 in patients who were in remission at baseline;
(ii) non-response to initial UC therapy or secondary loss of response in patients with active disease (PMS≥3) or newly diagnosed UC; or
(iii) UC-related rehospitalization outside of routine follow-up in patients being monitored for active disease.

Statistical Analyses

Statistical analyses were performed using Jamovi version 2.5.3. The normality of variables was examined using visual (histograms and probability plots) and analytical methods (Kolmogorov-Smirnov/Shapiro Wilk tests). Non-normally distributed variables are presented as median and interquartile range (IQR), while normally distributed data are presented as mean and standard deviation. Chi-square or Fisher's exact tests were used to compare categorical variables. Continuous variable comparisons were performed using the Student's t-test for normally distributed data and the Mann-Whitney U test for non-normally distributed data. The relationship between GISP positivity, EPEC and Campylobacter spp. positivity, and PMS on day 0 with relapse-free days was evaluated using Kaplan-Meier survival analyses and the log-rank test.

Cox regression models were created to evaluate the factors affecting attack-free days. Variables with $p<0.05$ in univariate analyses and those that could be clinically significant but did not show high correlation ($rho<0.75$) with each other (day 0 disease activity, multiplex PCR result, hemoglobin, CRP, and UC duration) were included in the model. The proportional hazard assumptions for each categorical variable were assessed using graphs. Cases where the Type-1 error value was <0.05 were considered statistically significant results.

Sample Size Calculation

Based on a study by Hanada and et al., the one year relapse rate was 11% in PCR positive patients and 37% in PCR negative patients with IBD(6). Assuming these proportions, a two-sided type I error of 0.05 and a type II error of 0.20 (80% power), a minimum of 42 patients was required in each group. Previous studies have reported PCR positivity rates in patients with IBD ranging from 17% to 26% (7). Assuming an estimated prevalence of 23%, a total sample size of 182 patients (±10%) was planned to ensure the inclusion of at least 42 PCR-positive individuals.

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Results

At the end of the sample collection period a total of 241 patients were included in the study. Stool samples were obtained from 191 of the patients. After excluding repeated samples from the same patient and samples deemed to be invalid after microbiological examination, a total of 174 patients were included in the analyses. Demographic and clinical characteristics of patients included in the study are presented in Table 1.

Table 1. Demographic and clinical characteristics of patients included in the study

Age, median (IQR %25-75)	44 (30-59)
Sex, female, n (%)	74 (41.4)
UC duration(years) median (IQR %25-75)	6 (2-10)
Change in treatment due to attack, n (%)	15 (8.6)
Colectomy, n (%)	6 (3.4)
Antibiotic use in the last 6 months, n (%)	40 (23.0)
History of attacks in the last 6 months, n (%)	63 (36.2)
Treatment group, n (%)	
No treatment	10 (5.7)
Monotherapy	95 (54.6)
Combination therapy	69 (39.7)
UC treatment, n (%)	
5- ASA[2]	137 (78.7)
Azathioprine	36 (20.7)
Anti-TNF [3]	32 (18.4)
Monoclonal antibody[4]	24 (13.8)
Corticosteroid [2]	18 (10.3)
Janus kinase inhibitors	-
Disease status (Day 0), n (%)	
Remission	129 (74.1)
Active	45(25.9)
Clinical findings (Day 0)n (%)	
Abdominal pain	21 (12.1)
Weight loss	13 (7.5)
Disorder of oral intake	8 (4.6)
Vomiting	2 (1.1)
Fever	1 (0.6)
Stool frequency, median (IQR 25-75%)	2 (2-4)
Colitis-related complaints	56 (32.2)

Extraintestinal symptoms	12 (6.9)
Laboratory findings (Day 0), median (IQR 25-75%)	
WBC, mm^3	7250 (6100- 8800)
Hemoglobin, g/dL	13.8 (12.5-14.9)
Platelet, x $10^3/mm^3$	275 (224-338)
AST, U/L	20 (17-25)
ALT, U/L	20 (15-29.5)
Total billirubin, mg/dL	0.53 (0.42-0.76)
Albumin, g/dL	4.4 (4.2-4.7)
Creatinine, mg/dL	0.74 (0.64-0.89)
CRP, mg/dL	4 (2.4 – 9.6)
ESR, mm/h	16 (9-33)
Endoscopic evaluation in the last 6 months,, n (%)	85 (48.9)
Endoscopic finding, n (%) [1]	
Remission	31 (36.5)
Active	54 (63.5)
Endoscopic site, n (%)[1]	
No finding	19 (22.4)
Proctitis (E1)	31 (36.5)
Distal (E2)	24 (28.2)
Pancolitis(E3)	11 (12.9)
EAI, median (IQR%25-75) [1]	8 (2-10)
MES, median (IQR%25-75) [1]	2 (0-3)
Stool frequency (last 3 days) ,n (%)	
Normal	112 (64.4)
1-2 stools/day than normal	27 (15.5)
3-4 stools/day than normal	16 (9.2)
≥5 stools/day than normal	19 (10.9)

Rectal bleeding (last 3 days), n (%)	
None	138 (79.3)
Visible blood with stool less than half time	13 (7.5)
Visible blood with stool half of the time or more	21 (12.1)
Passing blood alone	2 (1.19)
Physician rating of disease activity, n (%)	
Normal	126 (72.4)
Mild	22 (12.6)
Moderate	16 (9.2)
Severe	10 (5.7)
PMS, n (%)	
Remission (<3)	129 (74.1)
Mild (3-4)	22 (12.6)
Moderate (5-6)	11 (6.3)
Severe (≥7)	12 (6.9)

1. 85 patients were evaluated.
2. Local or systemic
3. Infliximab, adalimumab, sertolizumab
4. Vedolizumab, ustekinumab, guselkimab

Abbreviations: ESR;eritrosit sedimentation rate, EAI;endoscopic activity index, MES; Mayo endoscopic score,PMS;partial Mayo score

All patients underwent pathogen testing of stool samples via GISP. At least one pathogen was detected in 69 of the 174 stool samples (39.7%). At the time of the evaluation, at least one pathogen was identified by GISP in 40.3% (n=52/129) of patients in remission and 37.8% (n=17/45) of active patients (p=0.860).

Patients with and without identified pathogens were compared, and the results are presented in Table 2.

Table 2. Comparison of patients according to GISP results

	GISP positive, n=69	GISP negative, n=105	P value
Age, median (IQR %25-75)	41.0 (29.0-58.0)	47.0 (33.0-59.0)	0.209
Sex, female, n (%)	22 (31.9)	50 (47.6)	0.042
UC duration(years) median (IQR %25-75)	6 (2.0-10.0)	7 (3.0-10.0)	0.318
History of attacks in the last 6 months, n (%)	27 (39.1)	36 (34.3)	0.524
Antibiotic use in the last 6 months, n (%)	16 (23.2)	24 (22.9)	1.00
Disease status (Day 0), n (%)			
Remission	52 (75.4)	77 (73.3)	0.860
Active	17 (24.6)	28 (26.7)	
PMS, median (IQR%25-75)	0 (0-2)	0 (0-3)	0.724
EAI, median (IQR%25-75) [1]	8.0 (2-10)	7.5 (1.5-10.0)	0.928
Endoscopic site, n (%)[1]			
No finding	8 (21.6)	11(22.9)	**>0.999 ***
Proctitis	13(35.1)	18 (37.5)	
Distal	10 (27.0)	14 (29.2)	
Pancolitis	6 (16.2)	5 (10.4)	
UC treatment, n (%)			
5- ASA[2]	50 (72.5)	87 (82.9)	0.130
Azathioprine	14 (20.3)	22 (21)	1.00
Anti-TNF[3]	16 (23.2)	16 (15.2)	0.231
Monoclonal antibody[4]	10 (14.5)	14 (13.3)	0.826
Corticosteroid[2]	10 (14.5)	8 (7.6)	0.203
Treatment group, n (%)			
No therapy	4 (5.8)	6 (5.7)	>0.999 *
Monotherapy	36 (52.2)	59 (56.2)	
Combination therapy	29 (42.0)	40 (38.1)	

Flare(within 1 year)	30 (35.2)	37 (43.5)	0.275
Hospitalization (within 1 year), n (%)	3 (4.3)	11 (10.3)	0.167
Colectomy (within 1 year), n (%)	1 (1.4)	1 (1.0)	1.00
Mortality (within 1 year), n (%)	1 (2.6)	1 (1.4)	1.00

1. 85 patients were evaluated.
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*Corrected by Bonferroni adjustment

Abbreviations: EAI;endoscopic activity index, PMS;partial Mayo score

The pathogens detected in patients with positive GISP and their distribution according to disease activity at the time of testing were evaluated. **(Figure 1)**.

The most frequently detected pathogens were EPEC at 52.2% (n=36), *Campylobacter* spp. at 13.8% (n=24), and EAEC at 11.6% (n=8). **(Table 3)**.

Table 3. Distribution of pathogens in remission and active patient groups

Pathogen, n (%)	Total	Remission, n=52	Active, n=17	p value
Sapovirus	-	-	-	-
Adenovirus	1	1(0.8)	-	n/a
Astrovirus	-	-	-	-
Norovirus	4	2 (1.6)	2 (4.4)	0.275
Rotavirus	-	-	-	-
EPEC	36	30 (23.3)	6 (13.3)	0.157
EAEC	8	8(6.2)	-	n/a
ETEC	3	3(2.3)	-	n/a
STEC	6	4(3.1)	2(4.4)	0.649
EIEC	-	-	-	-
Yersinia enterocolitica	-	-	-	-
Campylobacter spp.	24	18(14.0)	6(13.3)	0.917
Salmonella spp.	-	-	-	-
Vibrio parahaemolyticus	-	-	-	-
Vibrio cholerae	-	-	-	-
Plseiomonas spp.	-	-	-	-
C. difficile toksin A	-	-	-	-
C. difficile toksin B	-	-	-	-
C. difficile toksin AB	2	1 (0.8)	1(2.2)	0.451
Giardia lamblia	2	1 (0.8)	1 (2.2)	0.451
Entamoeba histolytica	2	1 (0.8)	1 (2.2)	0.451
Cryptosporidium spp.	2	2 (1.6)	-	n/a
Cyclospora spp.	-	-	-	-
Enteroinvasive pathogens[1]	33	24 (18.6)	9 (20.0)	0.837
Polymicrobial	19	18(14.0)	1(2.2)	0.028

[1] *Campylobacter* spp., *Salmonella* spp., EAEC, STEC, EIEC, *Yersinia enterocolitica*, *Vibrio parahaemolyticus, Entamoeba histolytica*, *C. difficile* toxin AB.

In our study, multiple pathogens were identified at once in 19 among the 174 patients (11%). EPEC in 41.7%, *Campylobacter* spp. in 41.7%, EAEC in 50%, STEC in 50% ve ETEC in 66.7% were detected as part of polymicrobial positivity. All parasitic positivity was polymicrobial. *C. difficile* and viral pathogens were, on the other hand, detected as single pathogens in all cases.

The median cycle treshold(CT) values for the entirely polymicrobial parasitic agents were as follows: *Giardia lamblia* 25.3 (IQR 24.9-25.7), *Entamoeba histolytica* 25.7 (IQR 25.4-26), and *Cryptosporidium* spp. 23.3 (IQR 22.7-24).

The CT values of EPEC, *Campylobacter* spp., and EAEC, the three main pathogens in our study, were assessed for each pathogen in the remission and active groups on day 0, revealing no significant difference.When these pathogens were categorized as high positive (CT ≤26) and low positive (CT >26), also no significant difference was found in the remission and active patient groups on the day 0. (Supplementary).

Direct stool microscopy and culture examination were performed in 72 (41.4%) patients. In the direct microscopic examination, leukocytes and erythrocytes weren't present in 41 (56.9%) of the patients. Leukocytes were detected in all fields in 14 (19.4%) patients, leukocytes and erythrocytes were detected in 16 (22.2%) patients, and only erythrocytes were detected in 1 (1.4%) patient. *Salmonella* spp. and *Shigella* spp. were not detected in the culture examination of the patients.

In prospective follow-ups flare occured in 67 patients (38.5%) within the course of one year. Patients with and without flare were compared and presented in Table 4.

Table 4. Comparision of patiets with and without flare within 6 months

	With flare, n=67	Without flare, n=107	P value
Age, median (IQR 25-75%)	38.0 (29.0-55.0)	48.0 (33.5-60.0)	0.036
Sex, female, n (%)	48.0 (33.5-60.0)	45 (42.1)	0.819
UC duration , (IQR 25-75%)	4.0 (2.0-9.0)	7.0 (3.0-10.0)	0.027
Active disease (Day0), n (%)	28 (41.8)	17 (15.9)	0.001
History of attacks in the last 6 months, n (%)	39 (58.2)	24 (22.4)	<0.001
Treatment, n (%)			
5- ASA[1]	52 (77.6)	85 (79.4)	0.774
Azathioprine	14 (20.9)	22 (20.6)	0.958
Anti-TNF[2]	13 (19.4)	19 (17.8)	0.785
Monoclonal antibody[3]	14 (20.9)	10 (9.3)	0.032
Corticosteroid[1]	13 (19.4)	5 (4.7)	0.002
Treatment group, n (%)			
No therapy	1(2.7)	9 (6.6)	>0.999*
Monotherapy	18 (48.6)	77(56.2)	
Combination therapy	18 (48.6)	51(38.2)	
EAI, median (IQR 25-75%) [4]	9 (7.5-10.0)	5 (0-10.0)	0.004
Endoscopic site, n (%)[4]			
No finding	-	19 (29.7)	0.042*
E-1	8 (38.1)	23 (35.9)	
E-2	8 (38.1)	16 (25.0)	
E-3	5 (23.8)	6 (9.4)	
Pancolitis, n (%)	5 (23.8)	6 (9.4)	0.087
PMS (Day0), median (IQR, %25-75)	1 (0.0-4.0)	0 (0.0-1.0)	<0.001
WBC, mm^3 (Day0)	7600 (6600-10000)	7100 (6000-8400)	0.077

Hemoglobin(Day0), g/dL,	13.6 (12.4-14.4)	13.8 (12.9-15.0)	0.076
CRP (Day0), mg/dL,	6.3 (3.0-18.4)	3.41 (2.43-6.14)	0.002
ESR (Day0), mm/hours	22.0 (11.0-44.5)	15.0 (9.0-27.0)	0.022
GISP positive, n (%)	30 (44.8)	39 (36.4)	0.275
Polymicrobial,n (%)	9 (13.4)	10 (9.3)	0.400
Enteroinvasive pathogen, n (%)	16 (23.9)	17 (15.9)	0.191
EPEC	14 (20.9)	22 (20.6)	0.958
*Camplybacte*r spp.	13 (19.4)	11 (10.3)	0.089
EAEC	4 (6.0)	4 (3.7)	0.494

1. Local or systemic
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4. 85 patients were evaluated.

*Corrected by Bonferroni adjustment.

Abbreviations: ESR;eritrosit sedimentation rate, EAI;endoscopic activity index,PMS;partial Mayo score

The median time to development of an attack or re-attack during the one year follow-up period was determined to be 174 days, with a range of 19 to 364 days. The time to onset of attacks did not differ between GISP positive and GISP negative patients, with durations of 193 days (91-313) and 168 days (141-266) respectively (p=0.860, log-rank test). (Figure 2)

No relationship was identified between EPEC [(97 days (25-177) vs. 73.5 days (19-162), Log-rank, p=0.497)], *Camplybacter* spp. [(77 days (28-175) vs. 93,5 days (19-177), Log-rank, p=0,966)] and EAEC attacks [(84 days (19-177) vs. 131days (116-146) Log-rank, p=0.844)] positivity and time to onset of attacks.

No relationship was found between the detection of multiple pathogens and the time of attack development [(154 days (28-180) vs. 164 days (19-180) Log-rank, p=0.517)].

Patients with active disease at baseline had a shorter duration of attack-free days. [(82,5 days (25-176) vs. 108 days (19-177)), Log-rank, p<0,001)].

A model created to assess factors affecting attack-free days during follow-up included disease activity on day 0 (HR: 2.02 (1.17-3.49), p=0.011), GISP positivity (HR: 1.24 (0.76-2.02), p=0.395), age (HR: 0.99 (0.93-1.02), p=0.163), UC duration (HR: 0.98, 0.93-1.02, p=359), and CRP (HR: 1.02 (1.01-1.02), p=0.001). R^2 = 0.487, likelihood ratio, p < 0.001 (Figure 3).

Discussion

Ulcerative colitis is a relapsing–remitting disease, with reported 1-year relapse rates ranging from 13.7% to 58%(8–11). In the present study, the 1-year relapse rate was 38.5%, consistent with previous reports. A key strength of this study is the inclusion of patients in both remission and active disease at baseline. Notably, approximately one-third of patients who were in clinical remission experienced a relapse within one year, suggesting that clinical remission alone may not fully reflect underlying disease activity or long-term prognosis.

The most significant finding of this study is that simple clinical tools, specifically the Partial Mayo Score (PMS), remain far superior to costly molecular diagnostic panels (GISP) in predicting one-year relapse-free survival in Ulcerative Colitis (UC). Baseline disease activity (PMS ≥3) more than doubled the risk of relapse (HR:2.02). This reinforces the principle that "symptom control" is not merely a quality-of-life goal but the most reliable indicator of long-term prognosis in UC management.

Ulcerative colitis (UC) and gastrointestinal infections exhibit a bidirectional relationship. While mucosal barrier dysfunction and immunosuppressive therapy increase infection susceptibility in UC, enteric pathogens may also influence disease activity and prognosis (12). Despite this two-way relationship, our study found no significant association between GISP positivity and relapse duration. The fact that detection rates for the most common pathogens (EPEC and *Campylobacter spp*.) were nearly identical between patients in remission and those with active disease is a critical observation. These findings suggest that the microorganisms detected are likely "pathobionts" (opportunistic resident flora) rather than "true pathogens" (acute infection). The similarity in Cycle Threshold (Ct) values across both groups serves as strong evidence for this hypothesis. High Ct values, indicating a low bacterial load, suggest that PCR panels often detect subclinical "colonization" that does not necessarily drive the underlying inflammatory process.

The widespread use of GISP panels in UC patients introduces a significant risk of misattribution. Detecting *Campylobacter* or *E. coli* species may lead clinicians toward unnecessary antibiotic therapy rather than the essential optimization of UC-specific immunosuppressive or biologic treatments. In our cohort, almost all patients with polymicrobial positivity (multiple pathogens) were in clinical remission. This highlights that a positive GISP result, in the absence of a correlating clinical syndrome, should be viewed as asymptomatic carriage rather than an actionable infection.

The primary limitation of this study is the lack of routine endoscopic confirmation (Mayo Endoscopic Score) for every identified relapse. However, from the perspectives of real-world evidence and cost-effectiveness, non-invasive markers such as PMS and CRP remain the cornerstone of clinical practice. Future research should shift from qualitative (Positive/Negative) to quantitative (Ct-based load analysis) reporting of GISP results. This distinction could help clinicians differentiate between a transient "hitchhiker" and a pathogen that truly threatens mucosal stability.

Conclusion

Baseline clinical activity (PMS) remains the strongest predictor of relapse in UC, while pathogen detection via GISP shows no significant impact on disease course. GISP positivity should be interpreted with caution and primarily regarded as colonization unless strongly supported by the clinical context.

Declarations

Conflict of interest:

The authors declare no conflict of interest.

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Ethics approval and consent to participate: The study protocol was approved by the Gazi University Clinical Research Ethics Committee (approval date: May 16, 2022; approval number 357). Informed consent to participate was obtained from all of the participants in the study. This study complies with the Helsinki Declaration.

Consent for publication: Not applicable.

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Authors Contributions

Methodology, M.B.,O.G.T.,M.D.,M.A.; paitent selection, M.B.,A.K.,T.K.;anaylsis of samples, S.Y.,G.B.;statistical analysis, M.B.; writing, M.B.,O.G.T,S.Y,G.B.

Data Availability

The data underlying this article are available in the article.

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Figures

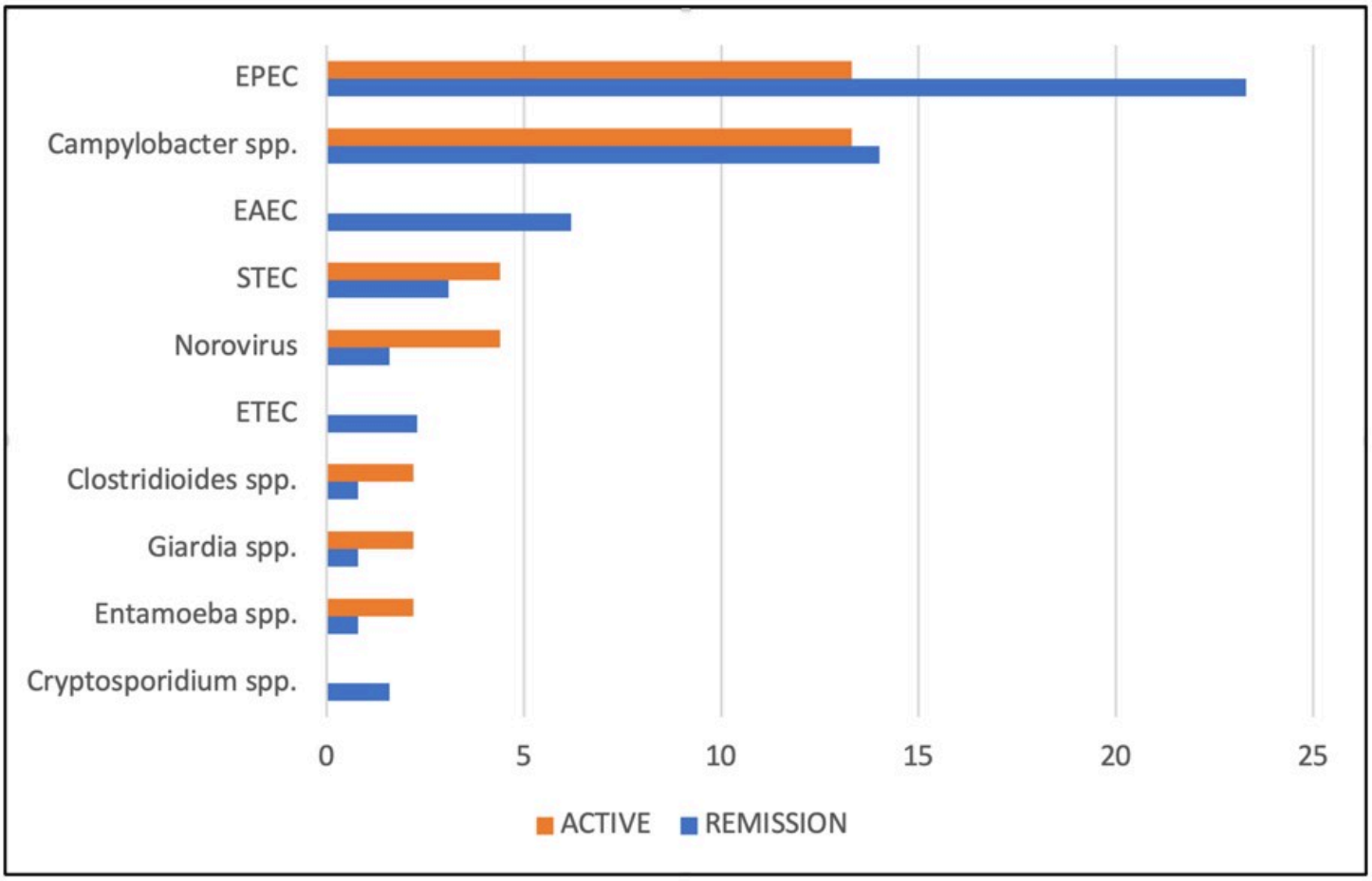


Figure 1

Distrubution of pathogens

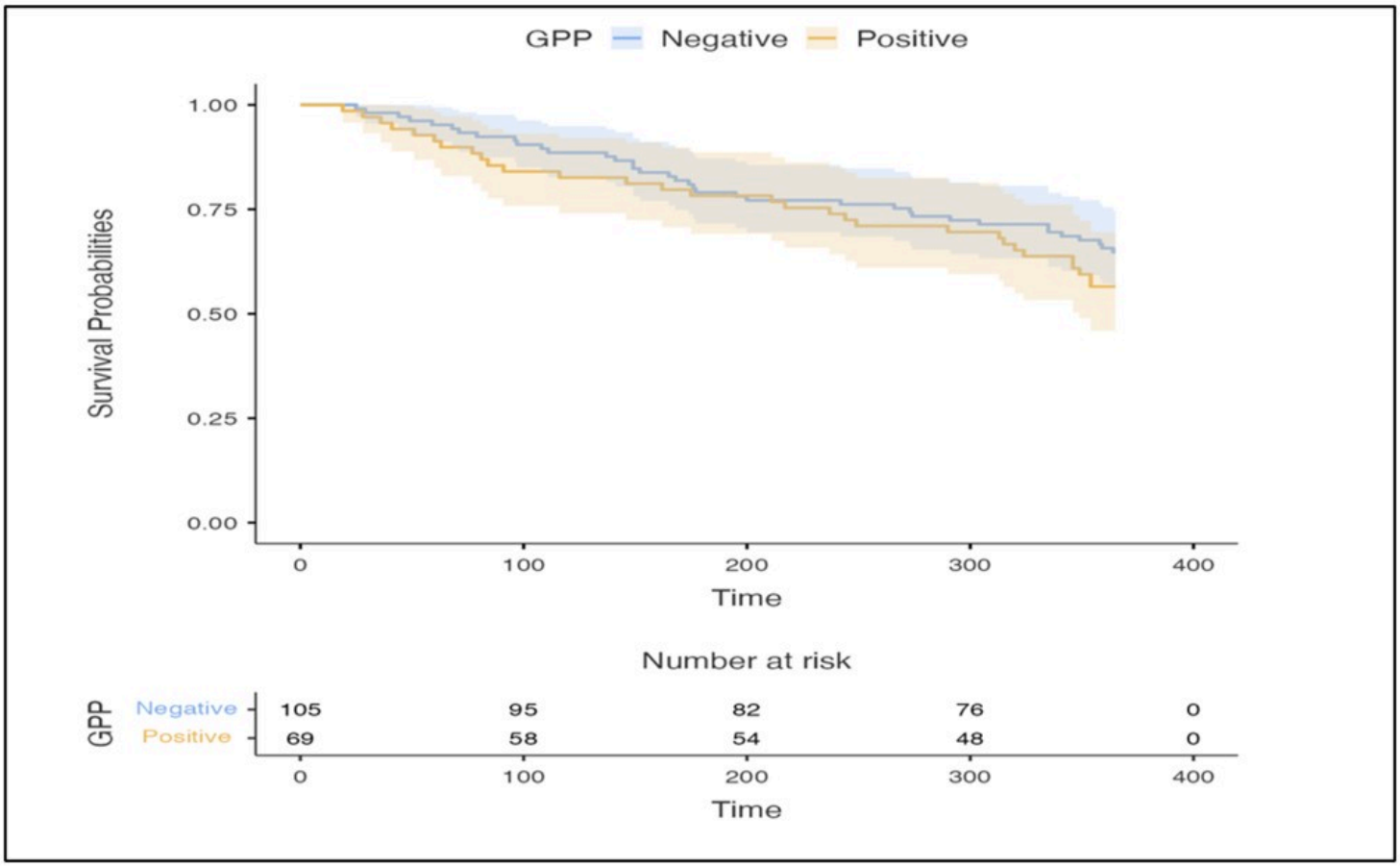


Figure 2

The effect of GISP positivity on attack free survival

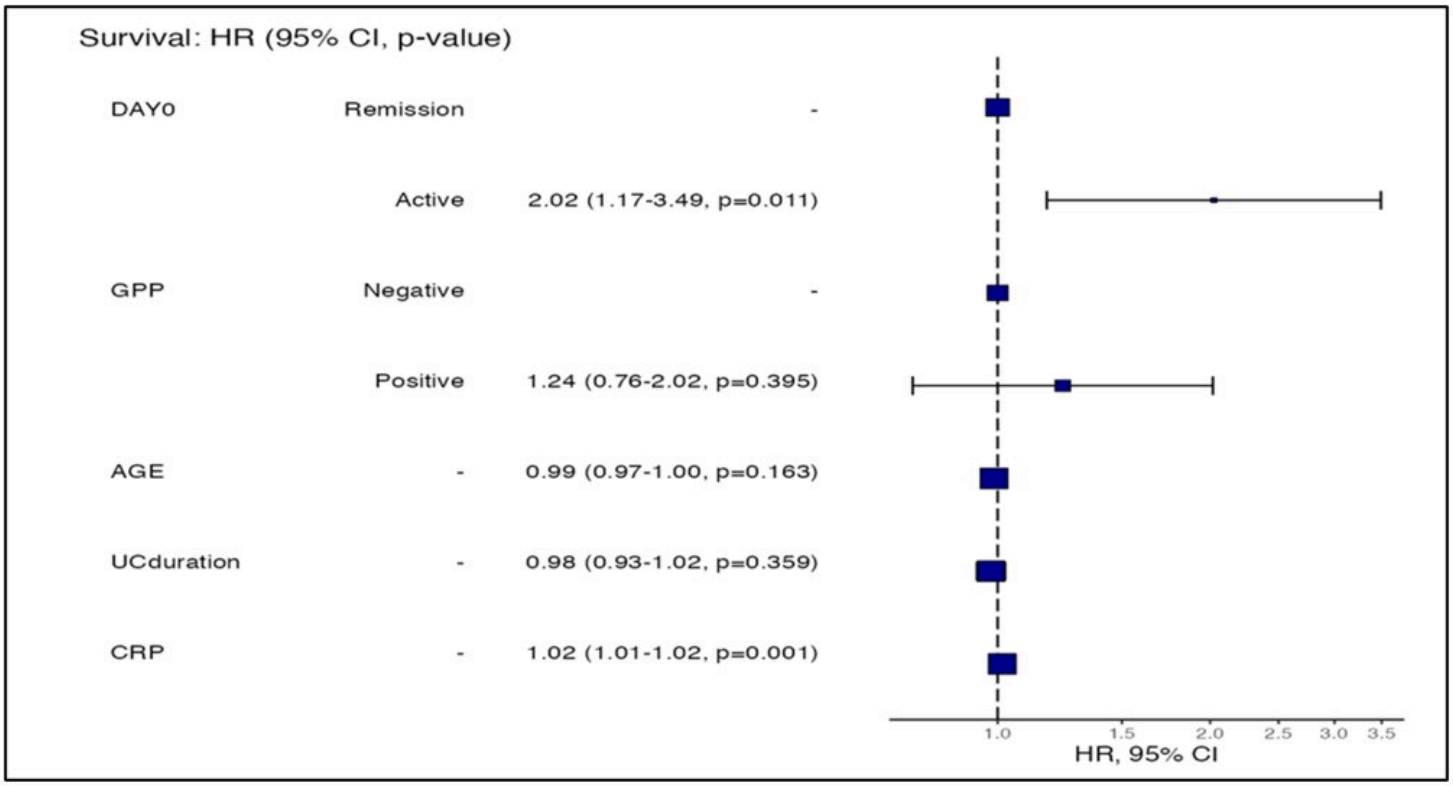


Figure 3

Attack free survival forest plot

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