haematologica
Journal of the Ferrata Storti Foundation

Histological response dynamics in gastric extranodal marginal zone B-cell lymphoma of the mucosa-associated lymphoid tissue after helicobacter-pylori eradication: a single-center longitudinal analysis

by Vincent Sunder-Plassmann, Barbara Kiesewetter, Ingrid Simonitsch-Klupp, Rosa Brand, Werner Dolak, Marius E Mayerhoefer and Markus Raderer

Received: October 14, 2025.
Accepted: February 25, 2026.

Citation: Vincent Sunder-Plassmann, Barbara Kiesewetter, Ingrid Simonitsch-Klupp, Rosa Brand, Werner Dolak, Marius E Mayerhoefer and Markus Raderer. Histological response dynamics in gastric extranodal marginal zone B-cell lymphoma of the mucosa-associated lymphoid tissue after helicobacter-pylori eradication: a single-center longitudinal analysis.
Haematologica. 2026 Mar 19. doi: 10.3324/haematol.2025.300054 [Epub ahead of print]

Category: Letter to the editor

Title: Histological response dynamics in gastric extranodal marginal zone B-cell lymphoma of the mucosa-associated lymphoid tissue after helicobacter-pylori eradication: a single-center longitudinal analysis

Vincent Sunder-Plassmann,[1,2] Barbara Kiesewetter,[1] Ingrid Simonitsch-Klupp,[3] Rosa Brand,[3] Werner Dolak,[4] Marius E. Mayerhoefer,[5,6] and Markus Raderer[1]

[1]Division of Oncology, Department of Medicine I, Medical University of Vienna, Vienna, Austria

[2]Christian Doppler Laboratory for Personalized Immunotherapy, Department of Medicine I, Medical University of Vienna, Vienna, Austria

[3]Department of Pathology, Medical University of Vienna, Vienna, Austria

[4]Division of Gastroenterology and Hepatology, Department of Medicine III, Medical University of Vienna, Vienna, Austria

[5]Department of Radiology, Division of Oncologic Imaging, NYU Langone Health, NYU Grossman School of Medicine, New York, USA

[6]Department of Biomedical Imaging and Image-Guided Therapy, Medical University of Vienna, Vienna, Austria

Corresponding author: Markus Raderer, Division of Oncology, Department of Medicine I, Medical University of Vienna**,** Waehringer Guertel 18-20, 1090 Vienna, Austria**,** Phone number: +43 1 40 400 44450**,** E-mail: markus.raderer@meduniwien.ac.at

Data-sharing statement: Data may be made available upon reasonable request to the corresponding author, contingent upon approval by the institutional review board and completion of a data sharing agreement.

Acknowledgements: The financial support by the Austrian Federal Ministry for Digital and Economic Affairs, the National Foundation for Research, Technology and Development and the Christian Doppler Research Association is gratefully acknowledged.

Authorship contribution: V.S. and M.R. conceived the conceptual idea, designed and performed the analysis; V.S., B.K., I.S.K., R.B., W.D., M.M. and M.R. contributed to data

collection and interpretation of the results; V.S. and M.R. wrote the manuscript; B.K., I.S.K., R.B., W.D. and M.M. critically reviewed the manuscript.

All named authors meet all four criteria for (co)-authorship provided by the Good Scientific Practice guidelines of the Medical University of Vienna. All authors read and approved the final manuscript.

Conflict-of-interest disclosure: V.S., R.B., and W.D. report no competing financial interests. B.K. received honoraria for lectures or advisory board participation from the following for-profit companies: from AAA, AstraZeneca, BMS, Beigene, Daichii Sankyo, Boehringer Ingelheim, Ipsen, MSD, Novartis, Eli Lilly, Roche, and Janssen Cilag. I.S.K. received honoraria for lectures from Takeda. M.M. received honoraria for lectures from the following for-profit companies: Siemens, General Electric, Bristol Myers Squibb. M.R. received honoraria from AbbVie, BeOne, Eisai, Eli Lilly, Gilead, Ipsen, Johnson and Johnson, Novartis, Pfizer and Roche.

Short title: Histological response dynamics in gastric MALT lymphoma

Gastric extranodal marginal zone B-cell lymphoma (EMZL) of the mucosa-associated lymphoid tissue (MALT) is unique within all B-cell lymphomas due to its strong pathophysiological connection with chronic antigenic stimulation by helicobacter-pylori (HP) infection.[1] Since the seminal discovery that antibiotic eradication of HP can induce durable remissions, HP eradication has become the recommended first-line treatment for gastric EMZL irrespective of stage, achieving long-term disease control and mostly obviating the need for further oncological interventions.[2-5]

One of the central challenges in managing gastric EMZL lies in the interpretation of histological response following HP eradication, as the histopathological evolution often follows a non-linear and patient-specific trajectory.[3, 6] The Groupe d'Étude des Lymphomes de l'Adulte (GELA) histological response criteria provide a structured framework for classifying histopathological post-treatment changes as complete remission (CR), probable minimal residual disease (pMRD), responding residual disease (rRD) and no change (NC).[7] However, little is known about the clinical relevance of fluctuating response depth or transient histological changes over extended follow-up, despite using extensive gastric mapping biopsy protocols.[8] This uncertainty is particularly important, as histological persistence or relapse after one year following HP eradication usually results in oncological therapy.[9]

We therefore conducted a retrospective analysis of patients with primary gastric EMZL treated with first-line HP eradication at a single tertiary center between 1999 and 2024 (Ethics Committee approval No 791/2011).

Histopathological samples of all patients were reviewed by a centralized reference pathologist and response assessed using the GELA classification. Immunohistochemistry for CD20, CD79a, CD5, CD10, CD23, Cyclin D1, Ki-67, evaluation of light chain restriction and molecular analyses to detect t(11;18)(q21;q21) translocation were routinely performed. Clinical characteristics were extracted from medical records. Data on infiltration depth of gastric EMZL was not routinely available and thus not included in our analysis. Patients with absence of HP on histology, absence of gastritis rated as post-HP gastritis and negative serology were rated as HP-negative. Time to next treatment (TTNT) was analyzed as the primary outcome via uni- and multivariable cox-regression analysis to reflect real-world clinical decision-making. Temporal differences between groups were assessed using the log-rank test. Categorical variables were compared using the χ^2 test or Fisher's exact test, as appropriate. A two-sided p-value of <0.05 was considered statistically significant.

Among 498 patients with MALT lymphoma identified at the Medical University of Vienna, 172 had primary gastric involvement. 129/172 were treated with upfront HP eradication and 111/129 patients had sufficient clinicopathological data available for longitudinal analysis (**Table 1**). Median follow-up after HP eradication was 28.9 months, with a median of five biopsy timepoints per patient, resulting in a total of 724 reviewed endoscopic biopsies (**Figure 1**). Gastric mapping biopsies were performed in all patients except for 14, who underwent gastroduodenoscopy without known biopsy protocol at external institutions and referred for centralized pathological review to our institution.

Upon HP eradication, 73.9% of patients achieved at least rRD, 56.8% achieved at least pMRD, and 46.8% CR. The median time to first response was 5.6 months for ≥rRD, 6.3 months for ≥pMRD, and 9.9 months for CR, underscoring the gradual deepening of histological response after HP eradication. In contrast, 26.1% of patients showed no histological response (=NC) throughout follow-up (**Figure 2A**).

At the respective last visit, 72/111 patients were still in routine follow-up after HP eradication. In contrast, 39 patients (35.1%) received additional non-antibiotic treatment (**Table 1**). Among these, eleven patients had histopathological signs of EMZL progression or recurrence within the stomach, while four patients had extragastric progression. Eleven patients received further treatment due to lymphoma persistence after HP eradication. One patient underwent diagnostic resection of a suspicious lung nodule, which was histologically confirmed as EMZL. In three patients, no information on the reason for subsequent therapy was available.

Nine further patients were diagnosed with gastric EMZL-associated secondary malignancies during follow-up, highlighting the importance of continued endoscopic surveillance. 7/111 patients (7.8%) developed transformation into a diffuse large B-cell lymphoma (DLBCL), which appears slightly higher than expected from earlier series, where it was judged to be between 2 – 5%.[10] Transformation occurred after a median of 8.7 months after EMZL diagnosis, which is earlier compared to a recently published median time to transformation of 3.67 years in a large retrospective cohort.[11] Gastric EMZL of five of those patients did not respond to HP eradication, while one patient each had rRD or CR as best response before DLBCL transformation. Importantly, patients with DLBCL transforming from EMZL restricted to the stomach appear to respond exceptionally well to chemoimmunotherapy[12] - all six patients with a known follow-up are in persisting CR after chemoimmunotherapy at 24+ to 67+ months. Two additional patients were diagnosed with gastric adenocarcinoma two months and eleven

years after HP eradication; one underwent gastrectomy while the other was treated with systemic chemotherapy, and both had CR with regard to their EMZL in their last specimen.

The depth of histological response was strongly associated with subsequent treatment decisions. Among histological non-responders, 55.2% of patients (16/29) received additional non-antibiotic treatment, whereas significantly less patients that achieved rRD, pMRD or CR were subsequently treated. Specifically, 28.1% of patients (23/82; p = 0.012) with ≥rRD, 23.8% of patients (15/63; p = 0.004) with ≥pMRD and 26.9% of patients (14/52; p = 0.016) with CR received further local or systemic treatment (**Figure 2B**). That effect largely persisted in uni- and multivariable cox regression analysis including stage, age, HP status and the presence of concomitant autoimmune disease; CR (HR = 0.26, 95% CI = 0.11 - 0.59; p = 0.001) and pMRD (HR = 0.12, 95% CI = 0.02 – 0.93, p = 0.042; **Figure 2C**). In addition, patients with Lugano stage II gastric EMZL were significantly more likely to receive subsequent treatment than stage I patients (HR = 2.7, 95% CI = 1.3 – 5.57, p = 0.007), although 10/19 patients with Lugano stage II achieved ≥rRD and nine did not receive subsequent treatment beyond antibiotics.

Longitudinal analysis revealed pronounced intra-patient variability in histological trajectories. Among patients who initially achieved ≥rRD, 25.6% exhibited at least one subsequent biopsy classified as NC that spontaneously reconverted to ≥rRD. Similarly, 30.8% of patients with CR experienced transient histological relapse to pMRD, rRD or NC followed by spontaneous reconversion to CR. These fluctuations were typically not accompanied by clinical or radiological progression. Nevertheless, patients with waning histological responses were significantly more likely to receive subsequent therapy than those with sustained or continuously improving responses (OR = 4.15; 95% CI = 1.02 – 24.70; p = 0.03), suggesting that dynamic patterns influence treatment decisions despite their often-benign clinical course. Importantly, a substantial proportion of patients without CR could be managed without further therapy, supporting the concept that complete histological remission is not required for durable clinical benefit in gastric EMZL. Indeed, recent clinical trials have demonstrated that additional (chemo-)therapy following HP eradication offers no significant clinical benefit in patients with gastric EMZL.[13]

At diagnosis, 68.8% of patients were classified as HP-positive, with a stable incidence over time (**Supplementary Figure 1**). HP-positive patients were significantly more likely to achieve ≥rRD, ≥pMRD, and CR (p = 0.002; p = 0.04 and p = 0.02, respectively) compared with HP-negative

patients (**Figure 2D and Supplementary Table 1**) and showed a trend towards faster response onset and deepening (**Figure 2A**), in agreement with historical data.[14] However, responses were not restricted to HP-positive disease: more than half of HP-negative patients achieved ≥rRD, and nearly one-third achieved CR following HP eradication alone, which appears to be higher than in previous publications.[15] Notably, 44% of HP-negative patients did not require any further lymphoma-directed therapy, supporting HP eradication as an appropriate first-line approach even in HP-negative gastric EMZL. Of major clinical impact is the fact that the time to (best) response in HP-negative patients was longer than for HP-positive patients in our series. This challenges the recommended practice of initiating oncological therapies after a shorter follow-up time of only 3-6 months after antibiotic therapy in case of non-response than in HP-positive patients, as suggested in the current ESMO guidelines.[5]

Despite similar patterns linking deeper histological response to lower treatment rates in both groups, HP-negative patients who achieved CR were more likely to receive subsequent therapy than HP-positive patients with CR (**Figure 2E**). Sustained histological responses were numerically more frequent in HP-positive patients, although differences did not reach statistical significance. Fewer patients with sustained histopathological response received subsequent non-antibiotic treatment than patients with a waning histopathological response in both groups (**Figure 2F**).

In summary, this large single-center longitudinal analysis demonstrates that histological response after HP eradication in gastric EMZL is highly dynamic and frequently fluctuating. Complete histological remission is not a prerequisite for durable disease control, and transient histological persistence or relapse is common and often clinically inconsequential. HP eradication alone provides long-term disease control in most HP-positive patients and a meaningful subset of HP-negative patients. Our findings support a conservative, observation-based strategy after HP eradication, emphasizing the integration of histological findings into the broader clinical context rather than using isolated biopsy results as triggers for immediate therapy.

References

1. Wotherspoon AC, Ortiz-Hidalgo C, Falzon MR, Isaacson PG. Helicobacter pylori-associated gastritis and primary B-cell gastric lymphoma. Lancet. 1991;338(8776):1175-1176.

2. Raderer M, Kiesewetter B, Ferreri AJM. Clinicopathologic characteristics and treatment of marginal zone lymphoma of mucosa-associated lymphoid tissue (MALT lymphoma). CA Cancer J Clin. 2016;66(2):152-171.

3. Fischbach W, Goebeler ME, Ruskone-Fourmestraux A, et al. Most patients with minimal histological residuals of gastric MALT lymphoma after successful eradication of Helicobacter pylori can be managed safely by a watch and wait strategy: experience from a large international series. Gut. 2007;56(12):1685-1687.

4. Nakamura S, Sugiyama T, Matsumoto T, et al. Long-term clinical outcome of gastric MALT lymphoma after eradication of Helicobacter pylori: a multicentre cohort follow-up study of 420 patients in Japan. Gut. 2012;61(4):507-513.

5. Zucca E, Arcaini L, Buske C, et al. Marginal zone lymphomas: ESMO Clinical Practice Guidelines for diagnosis, treatment and follow-up. Ann Oncol. 2020;31(1):17-29.

6. Kiesewetter B, Simonitsch-Klupp I, Dolak W, Mayerhoefer ME, Raderer M. Depth of Remission Following First-Line Treatment Is an Independent Prognostic Marker for Progression-Free Survival in Gastric Mucosa-Associated Lymphoid Tissue (MALT) Lymphoma. Cancers (Basel). 2020;12(2):492.

7. Copie-Bergman C, Gaulard P, Lavergne-Slove A, et al. Proposal for a new histological grading system for post-treatment evaluation of gastric MALT lymphoma. Gut. 2003;52(11):1656.

8. Ruskoné-Fourmestraux A, Fischbach W, Aleman BMP, et al. EGILS consensus report. Gastric extranodal marginal zone B-cell lymphoma of MALT. Gut. 2011;60(6):747-758.

9. Raderer M, Kiesewetter B. What you always wanted to know about gastric MALT-lymphoma: a focus on recent developments. Ther Adv Med Oncol. 2021;13:17588359211033825.

10. Kiesewetter B, Lamm W, Dolak W, et al. Transformed mucosa-associated lymphoid tissue lymphomas: A single institution retrospective study including polymerase chain reaction-based clonality analysis. Br J Haematol. 2019;186(3):448-459.

11. He Y, Gao C. Risk and clinical implications of histological transformation in extranodal marginal zone B-cell lymphoma of mucosa-associated lymphoid tissue: A population-based analysis. Sci Rep. 2025;15(1):20407.

12. Maeshima AM, Taniguchi H, Toyoda K, et al. Clinicopathological features of histological transformation from extranodal marginal zone B-cell lymphoma of

mucosa-associated lymphoid tissue to diffuse large B-cell lymphoma: an analysis of 467 patients. Br J Haematol. 2016;174(6):923-931.

13. Hancock BW, Qian W, Linch D, Delchier J-C, Smith P, Jakupovic I, Burton C, Souhami R, Wotherspoon A, Copie-Bergman C, Capella C, Traulle C, Levy M, Cortelazzo S, Ferreri AJM, Ambrosetti A, Pinotti G, Martinelli G, Vitolo U, Cavalli F, Gisselbrecht C, Zucca E. Chlorambucil versus observation after anti-Helicobacter therapy in gastric MALT lymphomas: results of the international randomised LY03 trial. Br J Haematol. 2009;144(3):367-375.

14. Zullo A, Hassan C, Ridola L, et al. Eradication Therapy in Helicobacter pylori-negative, Gastric Low-grade Mucosa–associated Lymphoid Tissue Lymphoma Patients: A Systematic Review. J Clin Gastroenterol. 2013;47(10):824-827.

15. Asano N IK, Koike T, Imatani A, Shimosegawa T. Helicobacter pylori-negative gastric mucosa-associated lymphoid tissue lymphomas: A review. World J Gastroenterol. 2015;21(26):8014-8020.

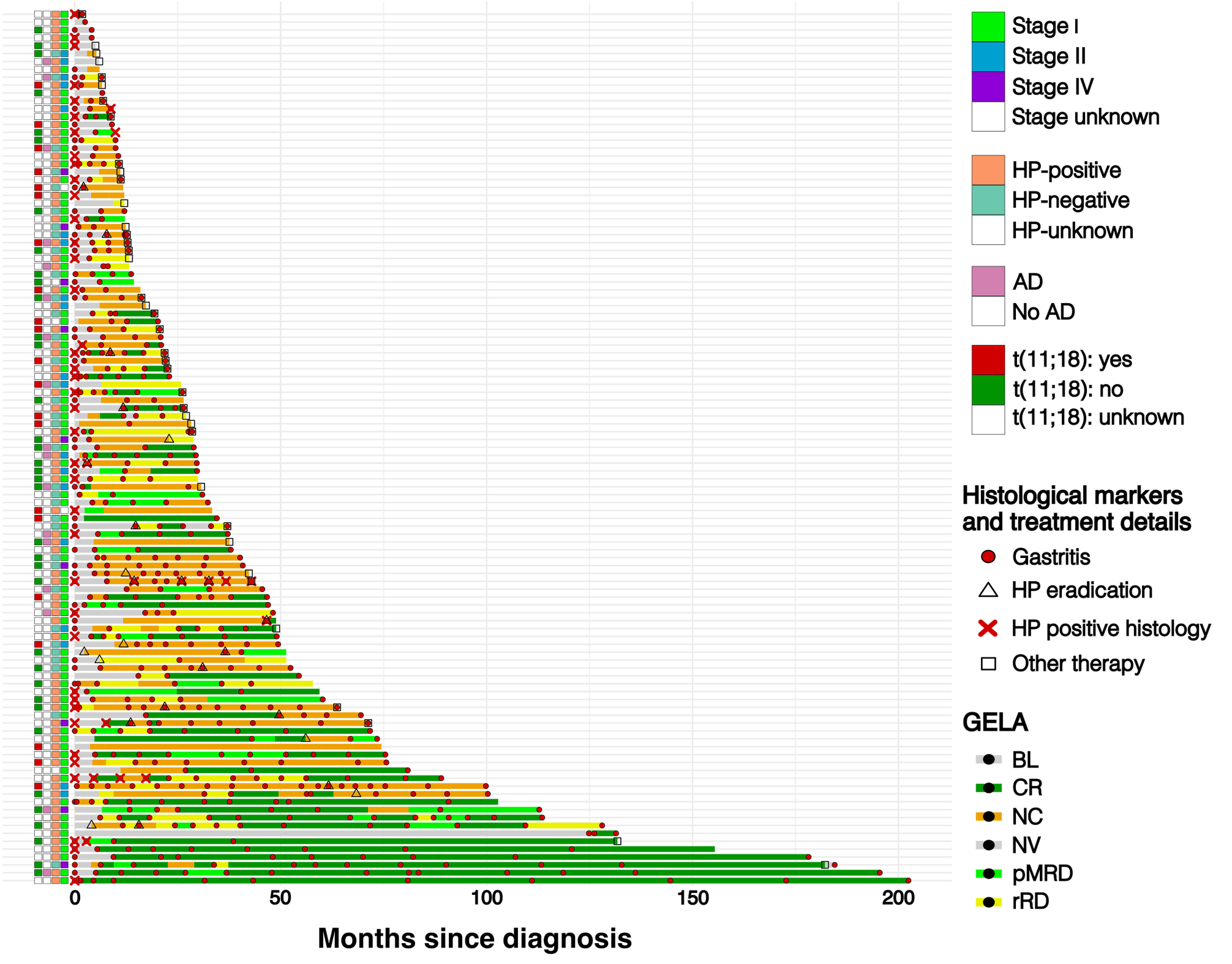
Table 1. Cohort demographics

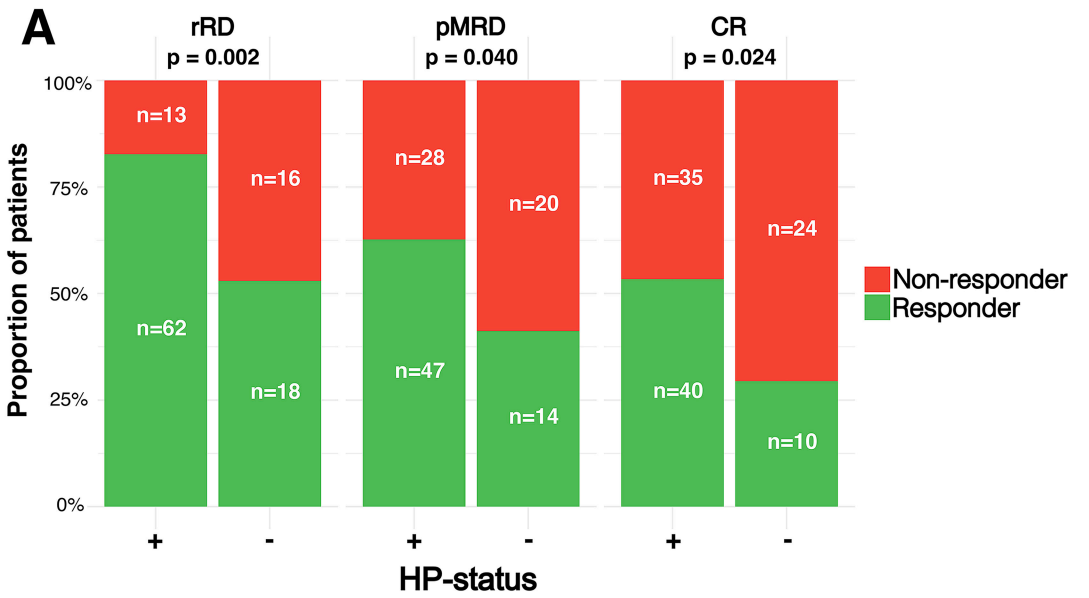
Characteristics	
MALT lymphoma patients, n	498
Primary gastric MALT lymphoma, n (%)	172/498 (34.5)
First-line HP eradication, n (%)	129/172 (75.0)
Excluded from analysis, n (%)	18/129 (13.9)
Insufficient clinicopathological data, n (%)	15/18 (83.3)
Unclear timing of HP eradication, n (%)	2/18 (11.1)
Synchronous breast cancer treatment, n (%)	1/18 (5.6)
Primary gastric MALT lymphoma	
Total study cohort, n (%)	111 (100)
Median age, years (range)	61.8 (31.5 – 84.6)
Sex (n, %)	
Female	55/111 (49.6)
Male	56/111 (50.4)
Stage (Lugano), n (%)	
I	82/109 (75.2)
II	19/109 (17.4)
IV	8/109 (7.3)
NA	2/111 (1.8)
IPI score, n (%)	
0	66/103 (64.1)
1	34/103 (33.0)
2	3/103 (7.3)
NA	8/111 (7.2)
HP-status, n (%)	
HP-positive	75/109 (68.8)
HP-negative	34/109 (31.2)
NA	2/111 (1.8)
First-line HP eradication, n (%)	111/111 (100)
Prior HP eradication, n (%)	8/111 (7.2)
Median number of cycles, n (range)	1 (1-4)
≥ 2 cycles of HP eradication, n (%)	21/111 (18.9)
Autoimmune disease, n (%)	17/111 (15.3)
t(11;18)(q21;q21), n (%)	
positive	21/57 (36.8)
negative	36/57 (63.2)
NA	54/111 (48.7)
Gastritis at diagnosis, n (%)	70/78 (89.7)
NA	33/111 (29.7)
MALT-associated secondary malignancy, n (%)	9/111 (8.1%)
DLBCL transformation	7/9 (77.8%)
Stomach cancer	2/9 (22.2%)
Median follow-up, months (range)	28.9 (1.8 – 202.4)
Median number of follow-ups, n (range)	5 (2 – 22)
Median time between follow-ups, months (range)	5.7 (0.2 – 124.8)
Best histopathological response, n (%)	
NC	29/111 (26.1)
≥ rRD	82/111 (73.9)
≥ pMRD	63/111 (56.8)
CR	52/111 (46.8)
Median time to first response, months (range)	
rRD	5.6 (0 – 124.8)
pMRD	6.3 (0.8 – 124.8)
CR	9.9 (0.8 – 126.2)
Non-antibiotic therapy after HP eradication, n (%)	39/111 (35.1)
Systemic Therapy, n (%)	35/39 (89.7)
Chemoimmunotherapy, n (%)	22/35 (62.9)
Anti-CD20 therapy, n (%)	6/35 (17.1)
Chemotherapy, n (%)	3/35 (8.6)
Proteasome inhibitor, n (%)	2/35 (5.7)
Immunomodulatory agent, n (%)	2/35 (5.7)
Local therapy, n (%)	4/39 (10.3)
Surgery, n (%)	2/4 (50)
Radiotherapy, n (%)	2/4 (50)

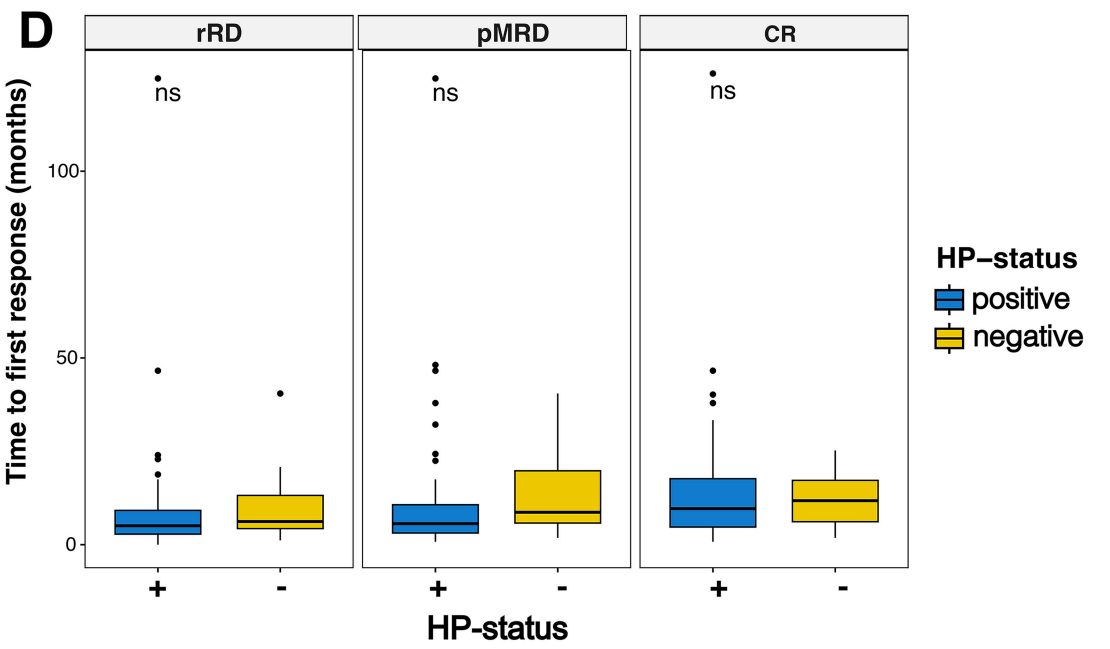
CR, complete remission; DLBCL, diffuse large B-cell lymphoma; HP, helicobacter pylori; n, number; NA, not applicable; NC, no change; PMRD, probable minimal residual disease; rRD, responding residual disease.

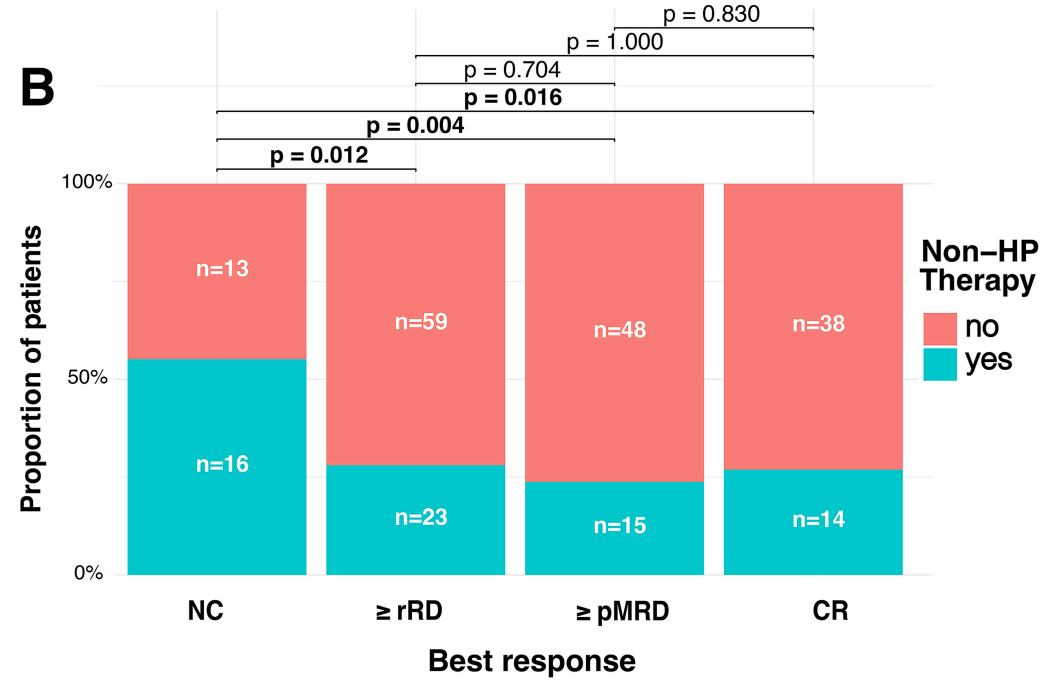
Figure 1: The histopathological response trajectory after first-line HP eradication treatment in patients with primary gastric MALT lymphoma. Each bar represents one patient in the study. The plot depicts relevant clinical covariates including disease stage (Lugano classification), HP-status, the presence of concomitant autoimmune diseases or t(11;18) translocation. Furthermore, the presence of gastritis and HP in each histopathological specimen is noted, where available. We furthermore highlighted the treatment course including consecutive HP-eradications and non-antibiotic treatment per patient. BL, baseline; CR, complete remission; GELA, Groupe d'Étude des Lymphomes de l'Adulte; HP, helicobacter pylori; NC, no change; NV, not available; pMRD, probable minimal residual disease; rRD, responding residual disease.

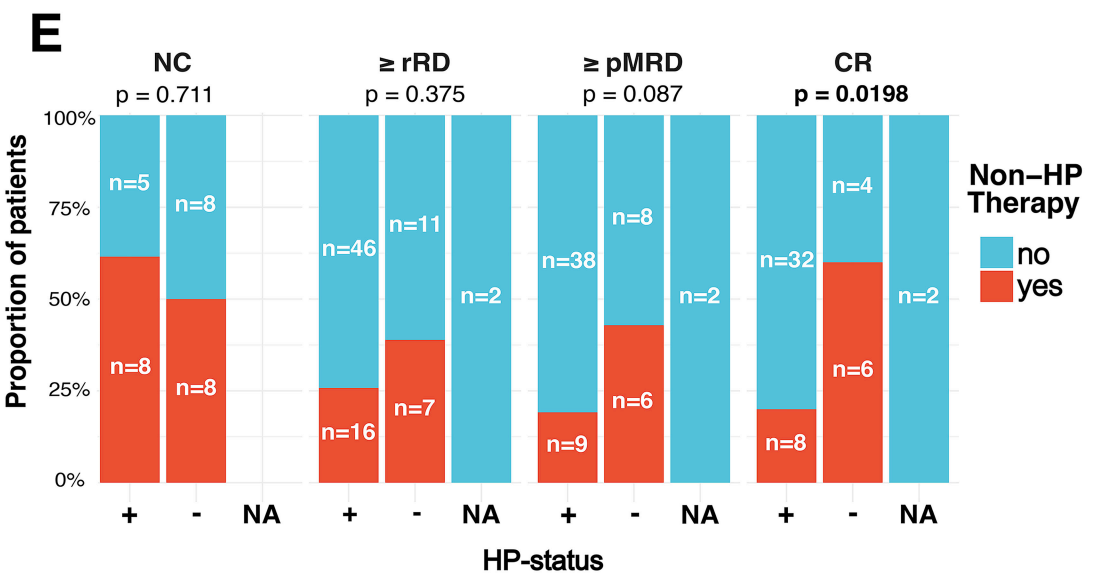
Figure 2: Histopathological response patterns, HP status, and their impact on clinical outcomes in gastric MALT lymphoma patients. (**A**) The median time to first rRD, pMRD or CR after HP eradication increases with response depth and is shorter in HP-positive compared to HP-negative patients, although not significantly ($p > 0.05$). (**B**) Patients with rRD, pMRD or CR as best response were significantly less likely to receive subsequent treatment for their MALT lymphoma. Notably, two patients with rRD or CR and subsequent NC received local radiotherapy. (**C**) A best histological response of pMRD (HR = 0.12, 95% CI = 0.02 – 0.93, $p = 0.042$) or CR (HR = 0.26, 95% CI = 0.11 – 0.59; $p = 0.001$) is associated with a decreased likelihood of receiving subsequent treatment lines in multivariable cox-regression analysis. No significant association of rRD as best response, disease stage, the presence of autoimmune disease, age or HP-status was observed. (**D**) Significantly more HP-positive patients had ≥ rRD, ≥ pMRD or CR as best response after HP eradication compared to HP-negative patients. (**E**) Significantly more HP-negative patients with CR as best histopathological response received subsequent treatments compared to HP-positive patients. No significant differences comparing pMRD, rRD or NC with subsequent treatments were observed between HP-positive and HP-negative patients. (**F**) Fewer patients with a sustained histopathological response received subsequent treatment irrespective of HP-status, although no significant difference was observed for both HP-positive and HP-negative patients ($p > 0.05$ each). AD, autoimmune disease; CR, complete remission; GELA, Groupe d'Étude des Lymphomes de l'Adulte; HP, helicobacter pylori; NA, not available; NC, no change; pMRD, probable minimal residual disease; rRD, responding residual disease.

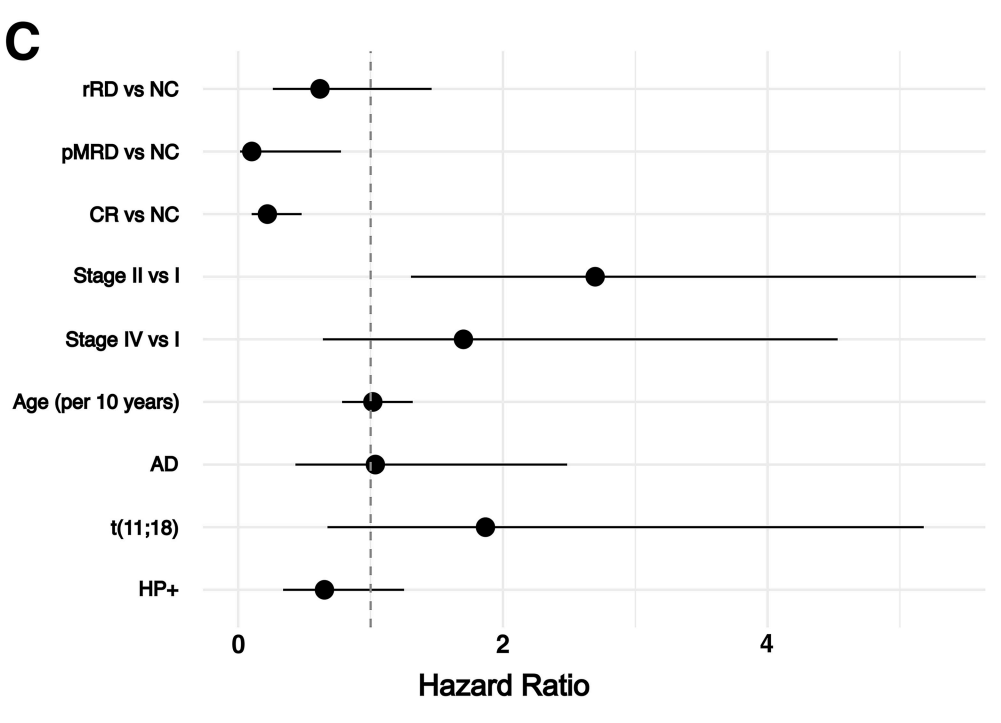

Patient Chacacteristics
Stage I
Stage II
Stage IV
Stage unknown
HP-positive
HP-negative
HP-unknown
AD
No AD
t(11;18): yes
t(11;18): no
t(11;18): unknown
Histological markers and treatment details
Gastritis
HP eradication
HP positive histology
Other therapy
GELA
BL
CR
NC
NV
pMRD
rRD
0
50
100
150
200
Months since diagnosis

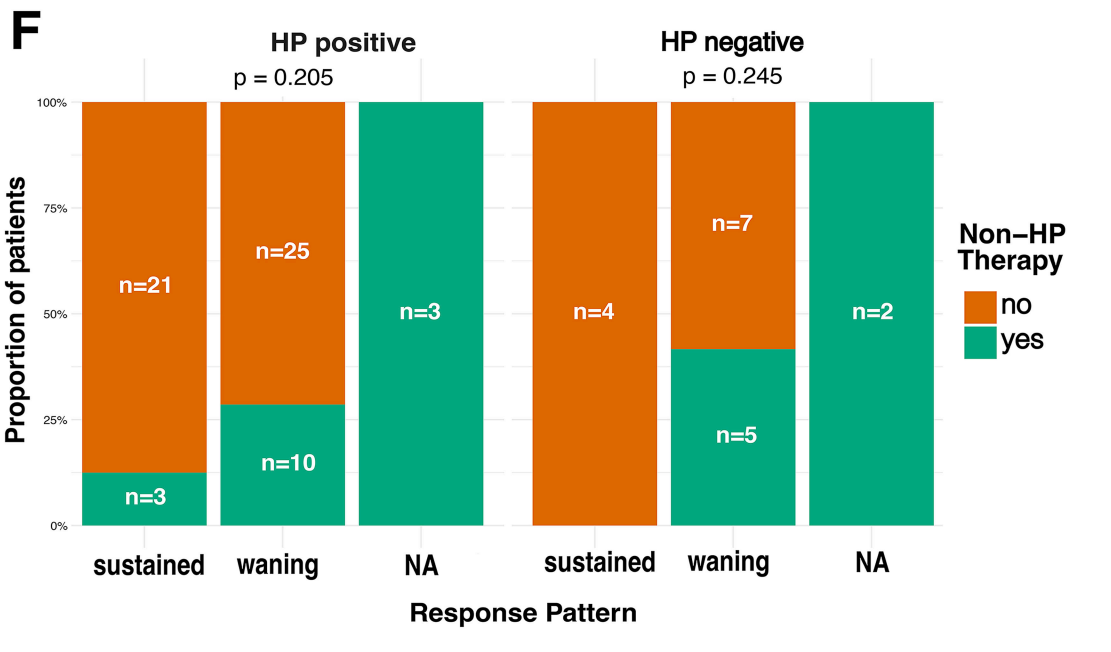

A
rRD
p = 0.002
pMRD
p = 0.040
CR
p = 0.024
100%
75%
50%
25%
0%
n=13
n=62
n=16
n=18
n=28
n=47
n=20
n=14
n=35
n=40
n=24
n=10
+
-
+
-
+
-
HP-status
Proportion of patients
Non-responder
Responder


D
rRD
pMRD
CR
ns
ns
ns
100
50
0
+
-
+
-
+
-
HP-status
Time to first response (months)
HP–status
positive
negative


B
p = 0.830
p = 1.000
p = 0.704
p = 0.016
p = 0.004
p = 0.012
100%
50%
0%
n=13
n=16
n=59
n=23
n=48
n=15
n=38
n=14
NC
≥ rRD
≥ pMRD
CR
Best response
Proportion of patients
Non–HP Therapy
no
yes


E
NC
p = 0.711
≥ rRD
p = 0.375
≥ pMRD
p = 0.087
CR
p = 0.0198
100%
75%
50%
25%
0%
n=5
n=8
n=8
n=8
n=46
n=16
n=11
n=7
n=2
n=38
n=9
n=8
n=6
n=2
n=32
n=8
n=4
n=6
n=2
+
-
NA
+
-
NA
+
-
NA
+
-
NA
HP-status
Proportion of patients
Non–HP Therapy
no
yes


C
rRD vs NC
pMRD vs NC
CR vs NC
Stage II vs I
Stage IV vs I
Age (per 10 years)
AD
t(11;18)
HP+
0
2
4
Hazard Ratio

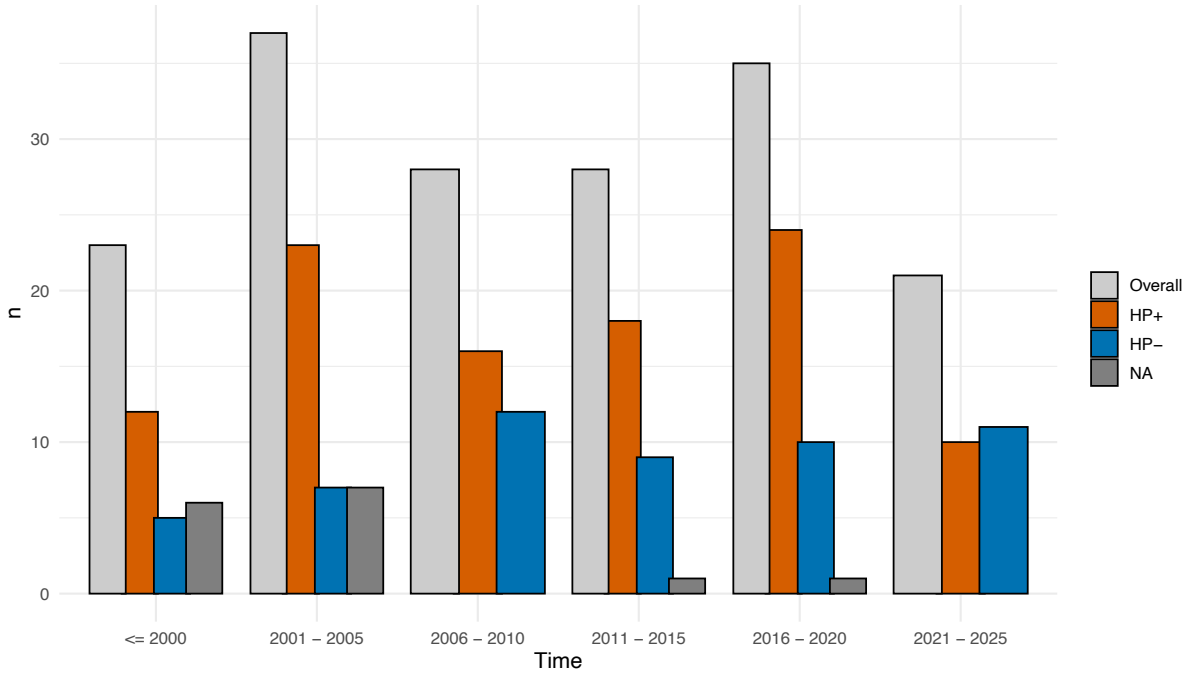

F
HP positive
p = 0.205
HP negative
p = 0.245
100%
75%
50%
25%
0%
n=21
n=3
n=25
n=10
n=3
n=4
n=7
n=5
n=2
sustained
waning
NA
sustained
waning
NA
Response Pattern
Proportion of patients
Non–HP Therapy
no
yes

Supplementary Files

Supplementary Table 1. HP-positive vs HP-negative patients

Characteristics	All patients	HP+	HP-	HP+ vs. HP-
Total cohort, n (%)	111 (100)	75/109 (68.8)	34/109 (31.2)	NA
Median age, years (range)	61.8 (31.5 – 84.6)	63.2 (31.5 – 84.6)	61.0 (35.2 – 84.3)	p > 0.05
Sex, n (%)				
Female	55/111 (49.6)	37/75 (49.3)	16/34 (47.1)	p > 0.05
Male	56/111 (50.4)	38/75 (50.7)	18/34 (52.9)	
Stage (Lugano), n (%)				
I	82/109 (75.2)	58/75 (77.3)	21/34 (61.8)	
II	19/109 (17.4)	11/75 (14.7)	8/34 (23.5)	p > 0.05
IV	8/109 (7.3)	5/75 (6.7)	4/34 (11.8)	
NA	2/111 (1.8)	1/75 (1.3)	1/34 (2.9)	
First-line HP eradication, n (%)	111/111 (100)	75/75 (100)	34/34 (100)	
Median number of cycles, n (range)	1 (1-4)	1 (1-4)	1 (1-4)	p > 0.05
≥ 2 cycles of HP eradication, n (%)	21/111 (18.9)	13/75 (17.3)	8/34 (23.5)	
Autoimmune disease, n (%)	17/111 (15.3)	9/75 (12.0)	8/34 (23.5)	p > 0.05
t11;18)(Q21;q21), n (%)				
positive	21/57 (36.8)	12/34 (35.3)	9/23 (39.1)	
negative	36/57 (63.2)	22/34 (64.7)	14/23 (60.9)	
NA	54/111 (48.7)	41/75 (54.7)	11/34 (32.4)	
Gastritis at diagnosis, n (%)	70/78 (89.7)	51/55 (92.7)	19/23 (82.6)	p > 0.05
NA	33/111 (29.7)	20/75 (26.7)	15/34 (44.1)	
MALT-associated secondary malignancy, n (%)	9/111 (8.1)	7/75 (9.3)	2/34 (5.9)	p > 0.05
DLBCL transformation	7/9 (77.8)	5/75 (6.7)	2/34 (5.9)	p > 0.05
Stomach cancer	2/9 (22.2)	2/75 (2.7)	0/34 (0.0)	p > 0.05
Median follow-up, months (range)	28.9 (1.8 – 202.4)	29.6 (1.81 – 202.4)	27.6 (5.3 – 184.5)	p > 0.05
Median number of follow-ups, n (range)	5 (2 – 22)	5 (2 – 20)	5 (3 – 22)	p > 0.05
Median time between follow-ups, months (range)	5.7 (0.2 – 124.8)	5.9 (0.5 – 124.8)	5.4 (0.2 – 34.2)	p > 0.05
Received further oncological therapy, n (%)	39/111 (35.1)	24/75 (32.0)	15/34 (44.1)	p > 0.05
Best histopathological response, n (%)				
≥ rRD	82/111 (73.9)	62/75 (82.7)	18/34 (52.9)	**p = 0.002**
≥ pMRD	63/111 (56.8)	47/75 (62.7)	14/34 (41.2)	**p = 0.04**
CR	52/111 (46.8)	40/75 (53.3)	10/34 (29.4)	**p = 0.002**
Median time to first response, months (range)				
rRD	5.6 (0 – 124.8)	5.1 (0 – 124.8)	6.2 (1.2 – 40.5)	p > 0.05
pMRD	6.3 (0.8 – 124.8)	5.6 (0.8 – 124.8)	8.7 (1.8 – 40.5)	p > 0.05
CR	9.9 (0.8 – 126.2)	9.6 (0.8 – 126.1)	11.8 (1.8 – 25.2)	p > 0.05

CR, complete remission; DLBCL, diffuse large B-cell lymphoma; HP, helicobacter pylori; n, number; NA, not applicable; NC, no change; pMRD, probable minimal residual disease; rRD, responding residual disease.



Supplementary Figure 1: Temporal trends in gastric MALT lymphoma patients. HP, helicobacter pylori; n, number; NA, not available.