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ADVANCES IN THE MANAGEMENT OF GASTRIC LYMPHOMAS: INTEGRATING GLOBAL EVIDENCE INTO CLINICAL PRACTICE

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ABSTRACT

Introduction and purpose: This narrative review explores gastric lymphomas, focusing on their epidemiology, diagnosis, treatment, and prognosis. It aims to integrate international guidelines with Polish clinical practice to address the diagnostic and therapeutic challenges associated with these rare and histologically diverse malignancies.

Results: Primary gastric lymphomas, particularly MALT lymphoma and DLBCL, require individualized management. MALT lymphoma is highly responsive to Helicobacter pylori eradication, with success rates reaching up to 80% in localized cases. However, the presence of the t(11;18)(q21;q21) translocation indicates a reduced response and necessitates additional treatment, such as radiotherapy or immunochemotherapy. For gastric DLBCL, R-CHOP remains the standard of care, and surgery has been largely replaced by organ-preserving strategies. Polish clinical protocols align with international standards, emphasizing bismuth-based eradication regimens and the growing role of molecular diagnostics.

Conclusion: Effective management of gastric lymphomas requires a multidisciplinary and individualized approach that accounts for histologic subtype, stage, H. pylori status, and molecular features. Non-surgical, evidence-based strategies significantly improve outcomes, with Polish practice reflecting global standards in antibiotic stewardship and precision oncology.

Keywords: Gastric lymphoma; MALT lymphoma; DLBCL; t(11;18)(q21;q21); H. pylori; individualized treatment.

INTRODUCTION

Primary gastric lymphomas represent a rare subset of both gastrointestinal and lymphoid neoplasms, yet they hold particular clinical significance due to their unique pathogenesis, diagnostic complexity, and highly variable prognosis. Unlike gastric adenocarcinomas, which arise from epithelial transformation, lymphomas of the stomach originate from lymphoid tissue that either develops secondarily within the gastric mucosa or involves the stomach as part of systemic disease. Among extranodal non-Hodgkin lymphomas (NHL), the gastrointestinal (GI) tract is the most frequently affected site, and the stomach is involved in over half of such cases. These tumors encompass a broad histologic spectrum, with extranodal marginal zone B-cell lymphoma of mucosa-associated lymphoid tissue (MALT lymphoma) and diffuse large B-cell lymphoma (DLBCL) being the two predominant subtypes [1] . Gastric lymphomas are characterized by distinct biological and clinical behaviors, ranging from indolent, infection-associated MALT lymphomas that may regress following *Helicobacter pylori* eradication, to aggressive DLBCLs that require systemic immunochemotherapy. The clinical presentation is often nonspecific and overlaps with that of benign gastropathies or gastric carcinoma, making early recognition a diagnostic challenge. Moreover, advances in endoscopic techniques, histopathology, and molecular diagnostics have substantially refined the diagnostic algorithm and facilitated individualized treatment planning. This review aims to provide a comprehensive and updated overview of gastric lymphomas, with a focus on their classification, diagnosis, treatment, and prognosis, while also incorporating current Polish clinical practices and epidemiological data. Special attention is given to the pivotal role of *H. pylori* in lymphomagenesis, the importance of molecular markers such as t(11;18)(q21;q21), and the integration of modern staging systems into therapeutic decision-making [2].

METHODS

A comprehensive literature search was conducted using major medical databases including PubMed, Scopus, and Web of Science. Studies published between 1990 and 2024 were reviewed and selected based on clinical relevance and methodological quality. Search terms included "gastric lymphoma," "MALT lymphoma," "DLBCL," "Helicobacter pylori," "t(11;18)," "rituximab," "radiotherapy," and "gastric cancer differential diagnosis." Priority was given to high-level evidence such as randomized controlled trials, consensus guidelines (e.g., ESMO, NCCN), systematic reviews, and national data from the Polish National Cancer Registry (KRN). No formal risk of bias assessment was performed, as this is a narrative review.

LIMITATIONS

This narrative review is subject to certain limitations. As it is not a systematic review, there is a possibility of selection bias in the literature included. Furthermore, the lack of formal quality assessment of individual studies limits the ability to draw graded recommendations. Nonetheless, we have prioritized high-quality evidence such as guidelines, randomized controlled trials, and large cohort studies to ensure a comprehensive and clinically relevant overview.

RESULTS OF SELECTION AND CONTENT OF THE REVIEW

EPIDEMIOLOGY

Primary gastric lymphomas are uncommon neoplasms, accounting for approximately 3% of all gastric malignancies and about 10% of all lymphomas [1,2]. Despite their rarity, they constitute the most frequent site of extranodal involvement in non-Hodgkin lymphoma (NHL), particularly within the gastrointestinal (GI) tract. The GI tract is the site of 30% to 45% of all extranodal NHL cases, and among these, the stomach is affected in 60% to 75% of cases [3].

In Poland, national epidemiological data indicate a steady burden of NHL. According to the Krajowy Rejestr Nowotworów (National Cancer Registry), 4, 343 new cases of NHL were diagnosed in 2019, with projections estimating an increase to 4, 456 cases in 2025 [4]. These values are consistent with global trends, highlighting a gradual rise in lymphoproliferative diseases, likely related to population aging and improved diagnostic capabilities. Diffuse large B-cell lymphoma (DLBCL) remains the most prevalent histological subtype of NHL in Poland, accounting for approximately 30% to 40% of all cases [5]. Follicular lymphoma (FL), typically an indolent form of NHL, constitutes around 15% to 20% of NHL diagnoses, with about 533 new cases expected annually by 2025 [4]. In the pediatric population, NHL represents the third most common malignancy in Poland. It is diagnosed at a frequency of approximately 11 cases per million children per year [6], aligning with global pediatric cancer registries and emphasizing the importance of early identification of extranodal presentations in young patients [5, 6].

CLINICAL PRESENTATION

Patients with gastric lymphoma commonly present with non-specific gastrointestinal symptoms. Epigastric pain is the most frequent symptom, occurring in approximately 78% of patients. Other presenting features include appetite loss (47%), unintentional weight loss (25%), upper GI bleeding (19%), and vomiting (18%). Constitutional B-symptoms (fever, night sweats, weight loss) are rare in gastric lymphomas and occur in only about 12% of cases [1]. Physical examination is often unremarkable, especially in early-stage disease. However, in advanced cases, palpable abdominal masses or peripheral lymphadenopathy may be present [2].

CLASSIFICATION

Histologically, gastric lymphomas are overwhelmingly dominated by two major subtypes: Extranodal marginal zone B-cell lymphoma of mucosa-associated lymphoid tissue (MALT lymphoma) and Diffuse large B-cell lymphoma (DLBCL). Together, these subtypes account for over 90% of all primary gastric lymphomas [1, 2]. MALT lymphoma is considered a low-grade, indolent neoplasm that arises from acquired lymphoid tissue in the gastric mucosa, often as a result of chronic *Helicobacter pylori* infection. It can remain localized for long periods and, in many cases, regress upon bacterial eradication. In contrast, DLBCL is a high-grade, aggressive lymphoma that may present de novo or as a transformation from a preexisting MALT lymphoma [1, 2, 3].

The classification of lymphomas has undergone several refinements to improve diagnostic precision and therapeutic planning. The Revised European-American Lymphoma (REAL) classification introduced in 1994 provided the first widely accepted framework based on immunophenotypic, genotypic, and clinical features [1, 2, 3]. It was later incorporated into the WHO classification, which continues to guide modern hematopathologic diagnosis. This unification has been critical for clinical staging and selection of optimal therapeutic strategies. In gastric lymphoma, histologic subtype remains a major determinant of both prognosis and first-line treatment [3].

DIAGNOSTICS

The diagnosis of gastric lymphomas is a multistep process that integrates clinical presentation, endoscopic evaluation, histopathology, immunophenotyping, molecular studies, and advanced imaging. Precise histologic diagnosis is essential to guide staging and to select the appropriate therapeutic approach, particularly because of the fundamentally different behavior of indolent MALT lymphoma versus aggressive DLBCL [1].

Upper gastrointestinal endoscopy is the primary diagnostic tool for direct visualization and biopsy of gastric lesions. Endoscopic findings in gastric lymphoma are variable and non-pathognomonic. Common appearances include mucosal erythema, polypoid or nodular lesions (with or without ulceration), thickened folds, and frank ulceration [1]. Given the potential for multifocal disease and submucosal infiltration, multiple large biopsies—both from visibly abnormal and normal-appearing mucosa—are recommended. Endoscopic mucosal resection (EMR) may enhance diagnostic yield in select cases [7, 8]. Histologic confirmation is required to establish the diagnosis. Immunohistochemistry and flow cytometry can distinguish B-cell from T-cell neoplasms and assess markers such as CD20, CD10, BCL2, and Ki-67. Molecular techniques (PCR, FISH) can be used to detect the t(11;18)(q21;q21) translocation, especially in cases of MALT lymphoma [9, 10, 11, 12].

Accurate staging is essential, as disease extent strongly influences prognosis and treatment choice. Imaging modalities include:

- CT of the chest, abdomen, and pelvis, used to assess lymph node involvement and organ infiltration.
- Endoscopic ultrasound (EUS), helpful in evaluating depth of invasion in the gastric wall and regional lymphadenopathy [7, 13].
- PET-CT, increasingly used to detect metabolically active disease and distant involvement. It is particularly valuable in aggressive subtypes such as DLBCL 15[14, 16].

Peripheral blood smear and bone marrow biopsy are integral for detecting systemic spread, especially in high-grade lymphomas.

Testing for *H. pylori* is mandatory in all cases of gastric lymphoma, particularly in suspected or confirmed MALT lymphoma, due to its therapeutic and prognostic implications. In approximately 92% of MALT lymphoma cases, *H. pylori* infection is present [17]. Detection can be performed via urea breath test, stool antigen, serology, rapid urease test (RUT), or histologic staining. The t(11;18)(q21;q21) translocation, present in ~30% of MALT lymphomas, predicts poor response to *H. pylori* eradication therapy and greater likelihood of systemic spread [9, 10, 12, 11]. Molecular testing should therefore be performed, especially in *H. pylori*-negative cases or in tumors unresponsive to eradication therapy. Patients who are candidates for rituximab-based therapies should undergo screening for hepatitis B virus (HBV) due to the risk of viral reactivation during treatment [1, 54].

MALT LYMPHOMA OF THE STOMACH

Extranodal marginal zone B-cell lymphoma of mucosa-associated lymphoid tissue (MALT lymphoma) is a low-grade, indolent form of non-Hodgkin lymphoma that typically arises in the stomach. It is strongly associated with chronic *Helicobacter pylori* infection, which induces lymphoid tissue formation in the gastric mucosa and contributes to lymphomagenesis through persistent antigenic stimulation[1, 17, 18, 19] .

The association between *H. pylori* and gastric MALT lymphoma has been well-established. Up to 92% of patients with gastric MALT lymphoma are infected with *H. pylori* . The chronic inflammation stimulates T-cell–mediated B-cell proliferation, which may evolve into clonal expansion and malignancy [17, 19, 20, 21, 22]. Approximately 30% of cases harbor the t(11;18)(q21;q21) chromosomal translocation, which is linked to resistance to H. pylori eradication

and increased risk of dissemination [9, 10, 12, 11, 23].

H.PYLORI ERADICATION THERAPY

Eradication of *H. pylori* is the recommended first-line treatment for all patients with gastric MALT lymphoma, even in those with stage II or disseminated disease [24, 25]. Complete histological remission is achieved in 50% to 80% of H. *pylori-positive*, localized cases [26, 27, 23, 28, 29], with a median time to response of 15.5 months [28]. The standard regimen combines a proton pump inhibitor (PPI), clarithromycin, and either amoxicillin or metronidazole for 10–14 days [30]. In cases of eradication failure, alternative or sequential therapies are employed [31, 52, 55]. In Poland, increasing resistance to clarithromycin and metronidazole has shifted recommendations toward bismuth-based quadruple therapy (BQT) as the preferred first-line regimen. This includes a PPI, bismuth, tetracycline, and metronidazole administered for 14 days [32]. This regimen is considered highly effective, achieving eradication rates >90%, and is supported by national guidelines. Where bismuth is not available, non-bismuth quadruple therapy (PPI, amoxicillin, clarithromycin, metronidazole) is used. Levofloxacin-based triple therapy is employed in second-line treatment after failure of initial regimens [32, 33]. Confirmation of eradication is recommended ≥4 weeks post-treatment using urea breath or stool antigen testing [32].

RADIATION THERAPY

For patients who are *H. pylori-negative*, refractory to eradication, or positive for t(11;18), radiation therapy (RT) is a well-established and highly effective second-line option. Involved-field RT (24–30 Gy over 3–4 weeks) provides complete response rates nearing 100% and is associated with minimal toxicity [34, 35, 36, 37, 38, 39]. In Polish practice, RT is also recommended for stage IE–II1E disease unresponsive to antibiotics. A dose of 30 Gy in 15 fractions is standard, mirroring international protocols. Surgery is now reserved for complications (e.g., bleeding, perforation), not as primary treatment [40].

CHEMOTHERAPY AND IMMUNOTHERAPY

Systemic therapy is considered in patients with advanced-stage disease, nodal involvement, or resistance to local therapy. Alkylating agents such as chlorambucil and cyclophosphamide are commonly used. Cladribine and fludarabine are additional options with demonstrated efficacy in inducing remission [41, 42]. Rituximab, a CD20-targeted monoclonal antibody, can be used as monotherapy or in combination with chemotherapy, particularly in patients with low tumor burden or poor performance status. In Poland, rituximab is typically administered weekly for 4 weeks, with possible maintenance every 2–3 months over two years [43]. The IELSG-19 trial demonstrated improved event-free survival with rituximab plus chlorambucil compared to chlorambucil alone (68% vs. 50%), although overall survival at 5 years was equal in both groups (89%) [44, 54].

RELAPSED OR REFRACTORY DISEASE

For patients with relapse or refractory disease, immunochemotherapy remains the standard. Late relapses may be managed with repetition of the initial regimen, whereas early relapses (within 12 months) often necessitate alternative therapies such as R-Bendamustine or R-FCM. Autologous stem cell transplantation (ASCT) may be considered in selected younger patients with chemosensitive disease [45]. In Poland, patients with multiple relapses or resistance to standard therapy may access targeted therapies (e.g., BTK inhibitors, PI3K inhibitors) via clinical trials [45]. These agents represent promising options in heavily pretreated individuals.

Given its indolent course, MALT lymphoma may be monitored conservatively in asymptomatic patients, especially following *H. pylori* eradication. Watchful waiting with periodic endoscopy and biopsy is appropriate, particularly in elderly or comorbid patients [11, 24].

DIFFUSE LARGE B-CELL LYMPHOMA (DLBCL) OF THE STOMACH

Diffuse large B-cell lymphoma (DLBCL) is the most common histologic subtype of non-Hodgkin lymphoma (NHL) and accounts for up to 55% of all primary gastric lymphomas. It may arise de novo or result from transformation of a preexisting MALT lymphoma [1]. Compared to MALT lymphoma, DLBCL is a high-grade, aggressive neoplasm requiring systemic therapy regardless of stage.

DLBCL of the stomach often presents with more acute symptoms than MALT lymphoma. These include epigastric pain, nausea, vomiting, GI bleeding, and weight loss. B-symptoms (fever, night sweats, >10% weight loss) are more frequently observed compared to MALT cases, though still less common than in nodal DLBCL [1, 34, 46, 47]. Endoscopically, lesions may appear ulcerated, infiltrative, or mass-forming, with a high degree of mucosal destruction [1]. Histologically, DLBCL shows diffuse infiltration by large, atypical lymphoid cells with a high proliferation index (Ki-67 often >80%). Immunophenotypically, these lymphomas express CD20, BCL6, MUM1, and variable CD10, allowing subclassification into germinal center (GCB) and non-GCB subtypes [1, 47].

CHEMOTHERAPY

Systemic immunochemotherapy with R-CHOP (rituximab, cyclophosphamide, doxorubicin, vincristine, and

prednisone) is the standard of care for gastric DLBCL, independent of stage [1, 48, 49, 50, 51]. Early studies raised concerns about gastrointestinal complications (e.g., bleeding or perforation) during chemotherapy, but subsequent data confirmed its safety and effectiveness [1, 49]. The addition of rituximab significantly improved survival outcomes in nodal DLBCL and is broadly applied to extranodal forms, including gastric involvement [48]. While some retrospective studies question the survival benefit of rituximab specifically in gastric DLBCL [49, 52, 53], most current guidelines recommend its inclusion. DLBCLs that arise from MALT lymphoma are treated the same way as de novo DLBCLs, particularly if high-grade features dominate the histology [54].

RADIATION THERAPY

The benefit of adding radiation therapy (RT) to R-CHOP remains debated. Retrospective studies have shown lower relapse rates in patients receiving consolidation RT, particularly in limited-stage disease [50, 51]. However, no randomized trials have confirmed a survival advantage. RT (30–36 Gy) may be considered on a case-by-case basis, especially in patients with bulky localized disease or residual PET positivity post-chemotherapy [50, 51, 52].

H.PYLORI AND DLBCL

Eradication of *H. pylori* is not sufficient as monotherapy in pure DLBCL. However, in early-stage cases with histologic or molecular evidence of coexisting MALT components, *H. pylori* eradication may be attempted as an adjunct to systemic therapy [1, 52]. In rare patients with early-stage disease, low tumor burden, and a strong MALT background, isolated eradication has led to remissions — though this approach is not standard and requires careful surveillance [53]. Testing for *H. pylori* remains essential in all patients, both for epidemiologic insight and for guiding initial adjunctive management [1].

RELEVANCE OF MALT COMPONENT IN DLBCL

The presence of a MALT component in gastric DLBCL does not currently change the standard therapeutic approach, which remains full-course systemic immunochemotherapy [1]. However, patients with prominent MALT features and localized disease may benefit from RT or extended observation if systemic therapy is contraindicated. Molecular studies (e.g., FISH for t(11;18)) may clarify the tumor origin, but are not routinely performed once DLBCL is diagnosed [1].

The chromosomal translocation t(11;18)(q21;q21) is a well-characterized genetic aberration in gastric MALT lymphoma. This translocation results in a fusion of the API2 gene on chromosome 11 with the MALT1 gene on chromosome 18, producing a chimeric API2–MALT1 protein that leads to constitutive activation of the NF-κB signaling pathway and promotes B-cell survival [9, 10, 11, 12, 23].

The t(11;18) translocation is present in approximately 25–35% of gastric MALT lymphomas, particularly in cases that are negative for *H. pylori* or resistant to *H. pylori* eradication therapy [9, 10, 11, 12]. It is rarely found in other extranodal sites. The translocation is more common in Western populations than in East Asian cohorts [9]. Detection is performed using fluorescence in situ hybridization (FISH) or reverse transcription polymerase chain reaction (RT-PCR) on formalin-fixed biopsy material [1]. Though not universally available in routine practice in Poland, testing may be performed in reference laboratories as part of extended immunopathological work-up in selected cases [43].

t(11;18) positivity is considered a negative predictive factor for response to *H. pylori* eradication. Numerous studies have demonstrated that patients with this translocation rarely achieve remission following antibiotic therapy alone [9, 10, 12, 11, 23]. In such cases, treatment should proceed directly to radiotherapy or systemic therapy, even in stage I disease [1]. According to Polish recommendations, the presence of t(11;18) is an indication for second-line treatment and precludes prolonged observation after failed eradication . Importantly, patients with t(11;18)-positive tumors typically have an indolent but disseminated disease course, often involving extranodal sites such as bone marrow and lungs. However, they have a low risk of transformation to DLBCL compared to t(11;18)-negative MALT lymphomas [1].

Although t(11;18) correlates with treatment resistance to antibiotics, it does not predict a worse overall survival, especially in patients receiving appropriate second-line therapy. This reinforces the necessity of early molecular characterization to guide individualized treatment [23]. The presence of t(11;18) also appears to limit the efficacy of alkylating agents, including chlorambucil and cyclophosphamide, though rituximab and purine analogs like cladribine remain effective, even in translocation-positive patients [44, 43, 54].

THE ROLE OF SURGERY IN MANAGEMENT OF GASTRIC LYMPHOMAS

Historically, gastrectomy was the primary treatment for gastric lymphomas, both for diagnosis and curative intent. However, the introduction of accurate endoscopic biopsy techniques, advanced imaging, and highly effective nonsurgical therapies (including eradication therapy, radiotherapy, and immunochemotherapy) has almost completely eliminated the role of surgery as first-line therapy in gastric lymphomas [1].

Modern international and national guidelines, including Polish recommendations, emphasize that surgical resection should not be used as standard treatment for either MALT lymphoma or DLBCL of the stomach [1, 40].

Today, surgery is reserved for managing complications such as:

- Perforation
- Intractable hemorrhage unresponsive to endoscopic or pharmacologic therapy
- Gastric outlet obstruction
- Fistula formation (e.g., gastro-colic or gastro-bronchial fistulas)
- Diagnostic uncertainty, when repeated biopsies fail to establish a diagnosis

In Poland, similar indications are followed. Surgery is considered only in emergency situations or when complications from conservative treatment occur. The standard approach is organ preservation, using radiotherapy or systemic therapies to control the disease [40].

Surgical interventions, especially partial or total gastrectomy, are associated with significant morbidity and long-term complications including dumping syndrome, malabsorption, and nutritional deficiencies. Several retrospective series comparing surgery vs. conservative treatment (e.g., R-CHOP or RT) have demonstrated no survival advantage for patients undergoing resection. Moreover, with current treatments achieving high response rates (>90%) in both MALT lymphoma and DLBCL, the rationale for surgery is virtually absent unless emergent indications exist.

In rare cases where endoscopic biopsy is inconclusive and lymphoma is still suspected (e.g., persistent mass, negative histology, and high FDG uptake), diagnostic laparoscopy or surgical biopsy may be considered [1]. However, this is seldom necessary with modern EUS-guided biopsies and adequate immunophenotyping.

PROGNOSTIC FACTORS IN GASTRIC LYMPHOMAS

The prognosis of gastric lymphomas depends on a range of variables, including histologic subtype, clinical stage, molecular characteristics, treatment response, and clinical condition. Stratifying risk based on these factors allows for tailored treatment and better-informed surveillance strategies.

The most important prognostic factor is the histology. MALT lymphoma is a low-grade, indolent subtype with an excellent long-term prognosis; studies show that the 5-year overall survival (OS) in early-stage MALT lymphoma exceeds 90% [23]. In contrast, DLBCL is an aggressive malignancy; with systemic R-CHOP immunochemotherapy, the 5-year OS for localized gastric DLBCL ranges from 70–80%, provided there is a good treatment response [1, 4].

Clinical staging remains one of the most significant independent predictors of survival. The following staging systems are used:

- Lugano staging system – the most widely accepted for GI lymphomas, with simplified groupings (I–II for localized, IV for disseminated).
- Ann Arbor (Musshoff modification) – still used in some centers but less specific for GI involvement.
- Paris TNMB classification – the most detailed system, incorporating tumor depth (T), nodal involvement (N), metastasis (M), and systemic symptoms (B) [1].

Patients with early-stage (I–II1E) disease have significantly better outcomes than those with disseminated disease (II2E–IV).

Table 1 summarizes the most commonly used histopathological classifications of gastric lymphomas. Among these, the Lugano classification plays a central role in staging and guiding therapeutic decisions, particularly in gastrointestinal lymphomas.

Table 1. Lugano Classification for Hodgkin and Non-Hodgkin Lymphoma

Stage	Stage Description
Limited stage	
I	Lymphoma confined to the gastrointestinal tract
II	Lymphoma extending into the abdomen
II1	Local nodal involvement
II2	Distant nodal involvement

IIE	Penetration of serosa to involve adjacent organs or tissues
Advanced stage	
III	Not applicable in Lugano Classification
IV	Disseminated extranodal involvement or a gastrointestinal tract lesion with supradiaphragmatic nodal involvement

Source: [47].

The translocation t(11;18)(q21;q21) is a well-known genetic aberration found in approximately 30% of MALT lymphomas. It is strongly associated with resistance to *Helicobacter pylori* eradication therapy, but does not predict decreased overall survival if appropriate second-line treatment is implemented [9, 10, 12, 11, 23, 40]. On the other hand, *H. pylori* positivity is linked to a favorable prognosis in MALT lymphoma and correlates with an excellent response to antibiotic therapy [19, 32]. The Ki-67 proliferation index is also an important prognostic marker, especially in diffuse large B-cell lymphoma (DLBCL); a high index (>80%) indicates a more aggressive disease course and an increased risk of progression [1]. In Polish clinical practice, both *H. pylori* testing and detection of the t(11;18) translocation are routinely performed in major oncology centers, and the Ki-67 index is assessed in all gastric lymphoma specimens [40, 43].

Response to first-line therapy is a key determinant of long-term outcome. In MALT lymphoma, achieving complete remission following *Helicobacter pylori* eradication is associated with prolonged disease-free survival and excellent long-term prognosis [23, 26, 27, 28, 29, 32]. In the case of DLBCL, a complete metabolic response (CMR) observed on PET-CT after six cycles of R-CHOP immunochemotherapy predicts both prolonged overall survival (OS) and progression-free survival (PFS) [1]. Conversely, failure of first-line treatment—whether antibiotic therapy in MALT lymphoma or systemic immunochemotherapy in DLBCL—is associated with a poorer PFS, although effective second-line therapeutic options remain available.

Other established clinical prognostic variables include age over 60 years, elevated lactate dehydrogenase (LDH) levels, poor performance status defined as ECOG ≥ 2, the presence of B-symptoms, and bulky disease characterized by a mass larger than 10 cm. These factors are incorporated into the International Prognostic Index (IPI), which remains applicable to both nodal and extranodal forms of diffuse large B-cell lymphoma (DLBCL), including gastric presentations [1].

In Poland, prognostic stratification is conducted in line with international standards. The Lugano system is most commonly used for staging, while IPI is applied to all aggressive lymphomas [40, 45, 46]. *H. pylori* status is routinely evaluated, and molecular diagnostics (including FISH for t(11;18)) are increasingly accessible through central pathology laboratories [40, 46]. Data from the Polish National Cancer Registry confirm that the prognosis for early-stage MALT lymphoma is excellent, with 5-year OS often exceeding 90%, while outcomes in DLBCL are heavily influenced by stage, age, and response to R-CHOP [4, 32, 40].

CLINICAL MANAGEMENT ALGORITHM

Optimal management of gastric lymphoma is based on histological subtype, clinical stage, molecular characteristics (e.g., t(11;18)), and *H. pylori* status. The approach differs substantially between MALT lymphoma and DLBCL, requiring a tailored algorithm to guide therapy.

Upon suspicion of gastric lymphoma, based on endoscopic findings or clinical symptoms, several diagnostic steps are mandatory. These include obtaining multiple deep endoscopic biopsies to allow for histopathological evaluation and immunophenotyping [1], testing for *Helicobacter pylori* using methods such as the urea breath test, rapid urease test, or histological assessment [1, 32], and performing comprehensive staging with contrast-enhanced CT and/or PET-CT, endoscopic ultrasound (EUS), and bone marrow biopsy. In cases of suspected MALT lymphoma, molecular testing for the t(11;18) translocation using fluorescence in situ hybridization (FISH) or polymerase chain reaction (PCR) should also be conducted [1].

Table 2.Algorithm for MALT Lymphoma

Patient Factor	Recommended Action
MALT + *H. pylori* (+), t(11;18)(−), Lugano Stage I–II1	*H. pylori* eradication alone

No response to eradication after 12 months OR t(11;18)(+)	*Involved-field radiotherapy (24–30 Gy)*
Advanced disease (Stage II2 or IV) or systemic spread	Systemic therapy (e.g., rituximab ± chlorambucil or cladribine)
Relapse or refractory	Radiotherapy or immunochemotherapy

Source: .[1, 32, 40, 43, 45]

Table 3. Algorithm for DLBCL of the Stomach

Patient Factor	Recommended Action
Localized or disseminated DLBCL (any *H. pylori* status)	Systemic immunochemotherapy (R-CHOP × 6)
Bulky disease or PET-positive residual lesion	Consider consolidation RT (30–36 Gy)
MALT component present	*H. pylori* eradication may be adjunctive, but not standalone
Refractory/relapse after R-CHOP	Salvage chemotherapy ± ASCT

Source:[1, 45, 48, 49, 50].

Surgery is not a part of routine management and is reserved only for life-threatening bleeding, perforation, or obstruction and cases with diagnostic uncertainty unresolvable with endoscopy [1, 40].

FOLLOW-UP STRATEGY

For MALT lymphoma after eradication: endoscopy with biopsies every 3–6 months for the first 2 years, then annually [1, 32]. And for DLBCL: imaging (PET/CT) 6–8 weeks after chemotherapy, then every 6–12 months for 2–5 years [1].

PRACTICAL RECOMMENDATIONS FOR CLINICAL PRACTICE

- In patients with early-stage MALT lymphoma, routine testing and eradication of Helicobacter pylori should be the first-line approach.
- Molecular testing for t(11;18)(q21;q21) should be performed in MALT cases, as it predicts resistance to antibiotic therapy.
- DLBCL of the stomach requires immediate systemic R-CHOP treatment; H. pylori status does not alter the primary approach.
- Radiotherapy may be considered in residual or bulky disease, but is not routinely required.
- Surgery should be reserved for acute complications (e.g., perforation, hemorrhage).
- Prognostic stratification should include Lugano staging and biomarkers such as Ki-67.
- Clinical practice in Poland supports evidence-based, organ-preserving strategies and avoids unnecessary surgical interventions.
- Increased access to molecular diagnostics can significantly enhance individualized care and treatment outcomes.

CONCLUSION

1. DISEASE HETEROGENITY AND NEED FOR TAILORED TREATMENT

Gastric lymphomas are a heterogeneous group of malignancies that require treatment strategies tailored to histologic subtype, clinical stage, molecular profile, and individual patient factors.

2. MANAGEMENT OF MALT LYMPHOMA

- MALT lymphoma is the most common indolent subtype.

- It is closely associated with Helicobacter pylori infection.
- In early-stage disease, eradication therapy is often effective.
- However, t(11;18)(q21;q21) translocation, H. pylori negativity, or treatment failure indicate the need for radiotherapy or systemic immunochemotherapy.

3. MANAGEMENT OF GASTRIC DLBCL

- Diffuse large B-cell lymphoma (DLBCL) is an aggressive entity.
- It requires systemic R-CHOP therapy, irrespective of H. pylori status.
- Adjunctive radiotherapy is considered in selected cases (e.g., bulky or residual disease).
- Surgery is reserved for managing complications such as bleeding, perforation, or obstruction.

4. PROGNOSTIC TOOLS AND BIOMARKERS

- Prognostic evaluation should include staging systems (e.g., Lugano, IPI).
- Molecular markers (e.g., t(11;18), Ki-67) further aid in risk stratification and treatment planning.

5. POLISH CLINICAL PRACTICE AND STANDARDIZATION

- Polish practice aligns with international standards.
- Emphasis is placed on bismuth-based eradication regimens, immunochemotherapy, and radiotherapy, with minimal use of surgery.
- Molecular diagnostics are becoming more widely implemented, enhancing consistency and treatment quality.

6. FUTURE PERSPECTIVE

Ongoing research and integration of molecular tools are essential for refining personalized care in the management of gastric lymphomas.

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CONFLICT OF INTEREST

The authors declare no conflict of interest.

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