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**Rare gastro-intestinal lymphomas: the endoscopic investigation**

Vetro C *et al*. Endoscopy in rare GI lymphomas

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**Abstract**

Gastrointestinal lymphomas represent up to 10% of gastrointestinal malignancies and about one third of non-Hodgkin lymphomas. Most prominent histologies are Mucosa-associated Lymphoid Tissue Lymphoma and Diffuse Large B-Cell Lymphoma. However, the gastrointestinal tract can be site of more rare lymphoma subtypes as primary or secondary localization. Due to their rarity and the multifaceted histology, an endoscopic classification has not been validated yet. This review aims to analyze the endoscopic presentation of rare gastro-intestinal lymphomas from disease diagnosis to follow-up, according to involved site and lymphoma subtype. Existing, new and emerging endoscopic technologies have been examined. In particular, we investigated the diagnostic and prognostic and follow-up endoscopic features of T-cell and NK lymphomas, Lymphomatous polyposis and Mantle Cell lymphoma, Follicular lymphoma, Plasma-cell related disease, gastrointestinal lymphomas in immunodeficiency and Hodgkin’s lymphoma of the gastrointestinal tract. Contrarily to more frequent gastrointestinal lymphomas, data about rare lymphomas are mostly extracted from case series and case reports. Due to data paucity, a synergism between gastroenterologists and hematologists is required in order to better manage the disease. Indeed, clinical and prognostic features are different from nodal and extranodal or bone marrow (in case of plasma-cell disease) counterpart. Therefore, the approach should be based on the knowledge of the peculiar behavior and natural history of disease.

**Key words**: Endoscopy; Lymphoma; Endosonography; Stomach; Intestine

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**Core tip:** Gastro-intestinal tract can be site of rare lymphomas as primary or secondary localization. Their endoscopic behavior has been scantly evaluated but is emerging as an useful tool with prognostic and therapeutic implications. T-cell lymphomas present mainly with ulcerative lesions, while B-cell lymphomas (follicular or mantle-cell lymphomas) present as duodenal mass or multiple polyposis. Plasmacell-related disorders localize to the gastrointestinal tract, either as neoplastic mass or as amyloid deposition. Immunodeficits (primary or secondary) can lead to gastrointestinal localization of rare, and seldom fatal, high-grade lymphomas. More rarely, Hodgkin’s Lymphoma localizes to the gastro-intestinal tract with uncertain impact on prognosis.

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# Introduction and main of the work

Gastro-intestinal (GI) lymphomas represent 5-10% of primary GI malignanciesand almost two third of extra-nodal non-hodgkin lymphomas (NHL), that in turn account for 24%-49% of all NHL[[1](#_ENREF_1),[2](#_ENREF_2)]. Most common lymphomas are Mucosa-Associated Lymphoid Tissue (MALT) and Diffuse Large B-cell Lymphoma (DLBCL), accounting for 70%-95% of GI lymphomas[[3](#_ENREF_3), [4](#_ENREF_4)]. Apart MALT and DLBCL, GI tract can be site of other lymphomas, either as primary or secondary localization[[5](#_ENREF_5)] and these lymphomas will be the object of this report. The knowledge of their clinical and echo-endoscopic features would help in addressing the clinical question[[3](#_ENREF_3),[6-8](#_ENREF_6)], sparing inappropriate evaluations[[9-13](#_ENREF_9)]. Nonetheless, histology, together with immunohistochemistry and molecular biology, are mandatory for diagnosis[[14](#_ENREF_14)].

While the endoscopic classification for MALT and DLBCL has been already validated[[15](#_ENREF_15),[16](#_ENREF_16)], such an analysis on rare GI lymphomas is still under debate. In 2001 and 2003, the Taiwanese[[17](#_ENREF_17)] and the Korean group[[18](#_ENREF_18)] respectively published a 3/5-item classification of ileo-colonic GI lymphomas. Table 1 shows patterns analyzed in both classifications. Basically, the endoscopic appearance is classified according to the presence and depth of ulcerations and of fungating lesions. To date, these are the only attempts to classify rare GI lymphomas. After that, Kim *et al*[[19](#_ENREF_19)] investigated the endoscopic differences between B- and T- cell lymphomas of the colon, and they observed that B-cell lymphomas occur more often as fungating or ulcero-fungating lesions, while T-cell lymphomas have more frequently an ulcerative or ulcero-infiltrative pattern (Figure 1). Notwithstanding, a clear prognostic implication based on the endoscopic pattern has not been validated yet.

Newer techniques, *i.e.,* capsule endoscopy (CE) and double-balloon enteroscopy (DBE), are emerging as useful tools in detecting small bowel tumors (15% of them represented by lymphomas)[[3](#_ENREF_3),[20-22](#_ENREF_20)]. Surely both techniques can augment the endoscopic diagnostic field (especially for follicular lymphomas[[21](#_ENREF_21)]). Moreover, spiral enteroscopy has been also evaluated as a tool for revealing GI lymphomas of the small intestine. Boudiaf *et al*[[23](#_ENREF_23)], reported that 4 out of 14 patients affected by refractory celiac sprue developed a small bowel mass that was confirmed to be an enteropathy-associated T-cell lymphoma (EATL) by histological evaluation. Even if less spread, single balloon-enteroscopy has been used in the definition of small bowel lesions and recently it has been implemented with the water-exchange method in order to improve the visualization of the lumen to better define and sampling the lesion[[24](#_ENREF_24)]. However, such a deep diagnostic tools have not been validated for routine use in GI lymphoma staging and follow-up since they do not induce a treatment change. Thus, their application in gastric or colonic lymphomas has not fully validated[[25](#_ENREF_25)]. Differently, facing with T-cell lymphomas, with a jejunal tropism, DBE can lead to a definitive diagnosis coupling the endoscopic investigation with the bioptic evaluation[[26](#_ENREF_26),[27](#_ENREF_27)]. However, not many publications related to the usage of these techniques are available to date.

A particular consideration should be given to the role of endoscopic ultrasonography (EUS). Its role has gained more and more importance in MALT lymphomas since the loco-regional staging of the disease has a great impact on the treatment approach[[6](#_ENREF_6)]. Regarding DLBCL, the loco-regional extension has significant prognostic implications, even if its role in treatment definition is still under discussion[[3](#_ENREF_3)]. In contrast, few data are available in rare GI lymphomas. In particular, they are more frequently regarded as general diseases, so that the loco-regional extension is not always evaluated with some reports indicating just the EUS pattern without any clinical implication. Exceptional cases have indicated the role of EUS in defining the limited extension of the disease thus leading to an endoscopic resection of the mass (see the paragraph “Extramedullary Plasmacytoma and Plasma Cell-related Diseases”). That notwithstanding, EUS information are gathered only for describing the behavior of these lesions in most cases without any significant clinical impact.

Definitively, a proper staging for GI lymphomas will include[[28](#_ENREF_28)]: (1) physical examination: evaluation of superficial lymph-nodes and Waldeyer Ring inspection; abdomen palpation in order to detect liver enlargement, splenomegaly and abdominal masses; (2) endoscopic ultrasonography that represents the golden standard in defining the loco-regional GI involvement since it is able to distinguish the involvement of a specific layer and also of regional lymph nodes. However, as stated above, its role is under study and it is not strictly recommended in this setting; (3) computed Tomography of neck, chest and abdomen in order to detect involvement of nodes above and below the diaphragm and also other extra-nodal involvement not pertaining the GI tract. In some cases, Computed Tomography can be of great help in defining the extension of a large bulky mass departing from the GI tract but exteriorizing outside the GI tract (look at the paragraph “Plasma-cell related diseases”; (4) Positron Emission Tomography is not generally indicated as a staging procedure, especially in MALT lymphomas but it retains a role in defining the pre-treatment lymphomatous involvement and response to treatment; and (5) bone marrow biopsy: not withstanding low-grade, indolent, diseases tend to remain localized at the GI tract, bone marrow biopsy should be performed in order to exclude a marrow involvement that could influence treatment and follow-up management. However, the level of evidence on its utility is poor. A recent update of the staging recommendation in nodal lymphomas does not encourage the performance of bone marrow biopsy facing Diffuse-large B cell lymphoma and Hodgkin lymphoma, but this strategy has not been evaluated specifically for GI lymphomas[[29](#_ENREF_29)].

However, these are general guidelines adopted from MALT lymphoma, since in more rare GI lymphomas, these guidelines have not been fully validated.

The aim of the present review is to highlight macroscopic features of rare GI lymphomas using endoscopy and related techniques. In particular we will focus on T-cell lymphomas, Lymphomatous polyposis (LP) and Mantle Cell lymphoma (MCL), Follicular lymphoma (FL), Plasma-cell related diseases, gastrointestinal lymphomas in immunodeficiency and Hodgkin’s Lymphoma (HL). An outline on the endoscopic presentation will be given for diagnostic aspect and follow-up assessment. As a whole, Table 2 summarizes the clinical and molecular characteristics and prognostic features of these lymphomas.

# T-cell and NK lymphomas

GI T-cell lymphomas are rare, representing about 5% of GI lymphomas[[14](#_ENREF_14),[30](#_ENREF_30),[31](#_ENREF_31)]. However, incidence varies according to the geographic zones. European studies reported that 1.3% of primary GI lymphomas is of T-cell origin[[32](#_ENREF_32)], while groups from Eastern Countries reported 7%-15%[[33](#_ENREF_33),[34](#_ENREF_34)] reaching the 41% in other series of intestinal lymphoma[[35](#_ENREF_35)].

Ulcerated lesions are the main endoscopic features[[30](#_ENREF_30),[36-38](#_ENREF_36)]. The first definition of this disease was “ulcerative jejunitis” by Isaacson and Du, given the always present ulcerative pattern[[14](#_ENREF_14)]. Usually, symptoms are related to malabsorption[[14](#_ENREF_14)], even if perforation[[39](#_ENREF_39)] or intestinal bleeding[[40](#_ENREF_40)] can occur. Incidentally, GI perforation or bleeding can occur in cases of nodal T-cell lymphomas independently from GI localization and recognize an infective etiology, reflecting the immune impairment that characterizes these lymphomas[[41](#_ENREF_41),[42](#_ENREF_42)].

Guidelines suggest that diagnostic work-up and follow-up should be done in synergism between hematologists and gastroenterologists in order to better define the staging and the treatment needed and to ensure the best nutritional guidance (evidence level III grade B)[[43](#_ENREF_43)].

In a study from the German group, the most frequent histotype of intestinal lymphoma was represented by T-cell lymphomas[[44](#_ENREF_44)]. The most common involved organs are duodenum and jejunum, followed by ileum and colon. Less frequent is the involvement of the stomach[[45](#_ENREF_45)] also as part of composite lymphoma[[46](#_ENREF_46)], *i.e.,* lymphoma with B- and T-cells origin. In a retrospective study by Matsumoto *et al*, it has been found that this type of lymphomas tends to have a small bowel localization more than other histotype. Regarding gastric involvement, in 30% of cases there is localization in the upper part of the stomach, in 20% the localization is in the middle part and diffuse in 40% of cases[[47](#_ENREF_47)]. Due to the fact that prognosis and treatment strategy depends on the lymphoma histotype, bioptic evaluation is a mandatory step. In addition, each subtype presents peculiar endoscopic behaviors that can drive diagnosis and treatment. GI T-cell lymphomas have typically a mature phenotype, while acute types of T-cell neoplasms do not classically involve GI tract[[48](#_ENREF_48)].

According to 2008 WHO classification of hematological malignancies, most prevalent histotypes are[[48](#_ENREF_48),[49](#_ENREF_49)]: (1) enteropathy-associated T-cell lymphomas (EATL) (distinguished in type I and II); (2) peripheral T-cell lymphomas and Extranodal Natural Killer(NK)/T-Cell lymphoma; and (3) adult T-Cell Leukemia/Lymphoma (ATLL).

In addition, very rare cases have been reported (and represent mostly singular events) of colo-rectal T-cell prolymphocytic leukemia/lymphoma[[50](#_ENREF_50)] or anaplastic T-cell lymphoma (ALCL) ALK+[[51](#_ENREF_51)] or ALK-[[52](#_ENREF_52)]. Distinct entities, not described in the WHO classification are indolent T-cell/NK diseases that will be also taken into account.

Although EUS findings are not usually reported except peculiar cases, submucosal hypoechogeneic lesions destroying the involved layer would represent the main pattern[[53](#_ENREF_53)]. Another proof of the sub-mucosal origin of the tumor is given by narrow band imaging, able to show intact gastric pits elevated from the underlying mass[[51](#_ENREF_51)]. Very rare and unusual is the GI involvement in Sezary syndrome where, despite unremarkable gastric mucosa, EUS can show hyperechogeneic submucosa layer at giant fold level[[54](#_ENREF_54)].

## Enteropathy-associated T-cell lymphoma

EATL can be divided into two forms[[14](#_ENREF_14)]: the first variant is characterized by features of celiac disease with abdominal pain and small intestine obstruction/perforation. Usually there is a large mass with massive necrosis, while the neighboring mucosa shows villous atrophy and crypt hyperplasia as in typical enteropathy. Type II exhibits villous atrophy in the context of tumor mass with normal intestinal mucosa in uninvolved sites. Contrarily to type I EATL, type II EATL does not progress from undiagnosed or refractory celiac disease[[14](#_ENREF_14),[55](#_ENREF_55)]. Prognosis is poor with a median Overall Survival of 7-10 mo[[56](#_ENREF_56)].

The exact incidence and lymphoma risk in celiac patients is still a debated issue[[57](#_ENREF_57)]. Some studies indicates a 200 fold increased risk to develop EATL compared to general population[[58](#_ENREF_58),[59](#_ENREF_59)]. According to other studies, the risk to develop non-Hodgkin lymphomas in Celiac patients appears to be 6-fold higher than in general population and this risk assumes a downward trend over years[[60](#_ENREF_60)]. Nonetheless, it appears clear that the occurrence of complications in celiac patients, even if infrequent, is an event that negatively impact on patient survival[[61](#_ENREF_61)]. In fact, the occurrence of intestinal perforation in a patient affected by celiac disease should lead to a suspicion of lymphoma.

Usually, EATL patients, tend to have a poorer Performance Status than B-Cell Lymphomas (even though tends to be localized), independently from the stage. Fever and diarrhea are the most frequent symptoms[[44](#_ENREF_44)]. Duodenum and jejunum represents the most involved sites with secondary involvement of the gross intestine in 14% of cases[[44](#_ENREF_44)]. The diagnosis of the disease in some cases is difficult since neoplastic lymphocytes can be present in a context of inflammatory background.

Endoscopic features are aspecific, with multiple erosions and ulcers[[31](#_ENREF_31)]. Nodularity and thickened folds can be seen at DBE[[26](#_ENREF_26),[27](#_ENREF_27)]. Strictures and masses are less common[[62](#_ENREF_62)]. In some cases, macroscopic findings together with the occurrence of an intense inflammatory reaction can lead to a mistaken diagnosis of Chron’s disease (CD)[[31](#_ENREF_31),[63](#_ENREF_63)]. However, even if it is not a general rule, CD ulcers are trasversal, while, in presence of T-cell lymphoma, ulcers are longitudinal[[63](#_ENREF_63)].

## Peripheral T-cell lymphomas and Extranodal Natural Killer/ T-Cell lymphoma

Peripheral T-Cell Lymphomas (PTCL) and natural killer (NK) lymphomas are more frequent in South America and Asia. These entities are distinct from other GI T-cell lymphomas by morphological and immunohistochemistry criteria[[62](#_ENREF_62)] and should be diagnosed when other more frequent T-cell lymphomas are excluded[[48](#_ENREF_48)]. Korean and Japanese series indicated that these are the most frequent GI T-cell lymphoma subtype accounting for 40% of primary T-cell GI lymphomas HTLV-1 negative[[64](#_ENREF_64)]. PTCL arises frequently in extranodal sites, especially at the skin. However, the involvement of the gastrointestinal tract represent a severe prognostic factor[[65](#_ENREF_65),[66](#_ENREF_66)]. Stomach an Duodenum accounts for 60% of GI localizations[[52](#_ENREF_52)]. Most frequent findings are ulcerative (46% of cases), infiltrative (9%), ulceroinfiltrative (18%), ulcerofungating (18%), erosive (9%)[[52](#_ENREF_52)]. Multiple polyposis can also be detected[[67](#_ENREF_67)]. In literature there are two reports indicating the involvement by T-cell lymphomas in the ileocolonic anastomosis for previously resected right colon, presenting with polypoid lesions[[68](#_ENREF_68)] or ulcerative lesions[[69](#_ENREF_69)].

Extranodal NK/T-cell lymphoma usually arises in nasal cavities and rarely affect the GI tract. A strict relationship exists between ENKTCL and EBV infection with almost 70% of cases positive for Epstein–Barr virus-encoded small RNAs (EBER) detection[[70](#_ENREF_70)]. Small intestine is the most involved organ, while stomach is rarely involved[[71](#_ENREF_71)]. The endoscopic pattern in majority of cases is given by multifocal ulcers[[72-75](#_ENREF_72)] and infiltrative lesions[[52](#_ENREF_52)]. Sometimes the ulceration leads to intestine perforation and acute peritonitis (60% of the total complications)[[52](#_ENREF_52)]. Additionally, perforation is more frequent in the infiltrative pattern compared to the non-infiltrative. Fungating lesions are not usually reported[[76](#_ENREF_76)]. The most involved organ is small intestine[[77](#_ENREF_77),[78](#_ENREF_78)] and/or colon[[72](#_ENREF_72),[76](#_ENREF_76)] (depending on the case series), followed by small intestine, rectum and stomach[[72](#_ENREF_72)]. However, the location at GI tract does not seem to affect the prognosis[[77](#_ENREF_77)]. Interestingly, since the perforation leads usually to the development of peritonitis, the Lugano staging system has been applied, resulting the advanced stage of the disease a prognostic factor at multivariate analysis[[72](#_ENREF_72)]. Due to the high risk of perforation, many patients undergo surgery as pre-emptive or curative strategy, rarely for diagnosis[[79](#_ENREF_79)]. However, according to Kim *et al*[[77](#_ENREF_77)], patients undergoing surgery followed by chemo/radio-therapy would show a better OS. However, as the authors themselves stated, this benefit would be ascribed to the fact that patients undergoing surgery had a better performance status and more limited disease and this would have affected the outcome. Similarly, as Hong *et al*[[78](#_ENREF_78)] reported, in a multivariate analysis, surgery ensures a better survival compared to chemotherapy. Therefore an appropriate loco-regional staging is also useful to tailor patients treatment.

## Adult T-Cell Leukemia/Lymphoma

As for EATL, ATLL tends to present with ulcers with aggressive behavior. This is a specific variant of peripheral T-cell lymphoma that recognizes HTLV-1 virus as etiological agent[[48](#_ENREF_48)]. This variant is mainly found in endemic areas of Japan[[64](#_ENREF_64)]. In about one third of ATLL cases, GI involvement is secondary to a systemic disease[[49](#_ENREF_49)]. According to first data by Suzumiya, stomach is involved in 40% of cases and small and large intestine in 38% and 34% respectively[[80](#_ENREF_80)]. Even if four types of ATLL have been depicted (*i.e.,* smoldering, chronic, lymphoma, acute) no endoscopic pattern has been related to a peculiar histotype. HTLV-1 infection has no role in determining the macroscopic features[[47](#_ENREF_47)]. Noteworthy, the detection of GI involvement has a prognostic impact[[49](#_ENREF_49)] representing the aggressiveness of the disease[[43](#_ENREF_43)]. In fact, smoldering or chronic ATLL subtypes do not show typically GI involvement[[81](#_ENREF_81)]. However, primary GI smoldering ATLL have been described and show long term disease-free survival after chemotherapy[[82](#_ENREF_82)]. Gastric involvement can be enhanced by *Helicobacter pylori* infection that creates an inflammatory state able to lead lymphocytes (also malignant) to migrate into gastric wall through the expression of specific adhesion molecules[[83](#_ENREF_83)].

Ulcerative pattern is present in more than half cases of gastric involvement[[47](#_ENREF_47)]. Single or multiple yellow-whitish polyposis of the first or second loop are more frequent in the duodenum[[49](#_ENREF_49)] and multiple polyposis is the recurrent lesion in case of colon involvement[[84](#_ENREF_84)]. Even if a single or multiple polyps are the most frequent lesions, flat ulcerations/erosions can also be present[[84](#_ENREF_84)]. Red flat or elevated lesions in the rectum have been also documented[[85](#_ENREF_85),[86](#_ENREF_86)]. Rarely, there is the involvement of the ileum, where polyps are the main features[[87](#_ENREF_87)]. It should be underlined that GI lesions are not always monotone, but can be variegated. For examples, case reports indicate occurrence of protruding masses with normal or eroded mucosa at the stomach and occurrence of flat granular, friable and bled on contact mucosa at the colon[[88](#_ENREF_88)] or reddish irregular flat lesions at the esophagus[[89](#_ENREF_89)].

Narrow-band imaging is able to document irregular microvascular architecture, dilated and destroyed gastric pits and dense aggregations between the pits with variegate irregular nuclei without interglandular infiltration (reflecting the absence of lympho-epithelioid lesions)[[90](#_ENREF_90)].

## Indolent lymph-proliferative diseases of the GI tract

A new category of T-cell GI lymph-proliferative disease, namely T-LPD (T-cell lymph-proliferative disease), has been recently introduced[[91](#_ENREF_91)]. The indolent course is the main clinical hallmark while this entity has been previously treated and managed as PTCL. Noteworthy, the etiology of the disease is unknown, even if many patients present a history of inflammatory bowel disease (IBD). Basically, the clinical picture is dyspepsia and mild diarrhea, while endoscopic feature can vary from unremarkable mucosa to erythema. Small intestine and colon are the most frequently involved sites, followed by oral cavity, stomach and esophagus. Usually gastric mucosa is normal despite a disease localization while duodenum can show thickened folds and irregular pattern. In the colon, occurrence of friable mucosa, erythematous mucosa and small polyps can be seen. Ulcerations are not described. At immunohistochemistry, lymphoid cells have a cytotoxic phenotype (CD8+; CD4-; TIA+), clonal T-cell receptor (TCR) gene rearrangements, do not form masses, do not invade the intestinal crypts and do not cover the full thickness of the bowel[[91](#_ENREF_91)]. Additionally, the lymphoid infiltrate is limited to the mucosa and sub-mucosa. The molecular study for TCR can show a monoclonal rearrangement of TCR- chain[[91](#_ENREF_91)]. The recognition of this disease have many therapeutic implications, since aggressive chemotherapy is excessive, being an immunosuppressive treatment virtually sufficient.

Indolent CD4+ T-cell lymphoma have also been described and show a good outcome and survival despite a persistence after immunomodulatory drug-based treatment[[92](#_ENREF_92)]. Rarely, gastric mucosa can show multiple nodularities[[93](#_ENREF_93)]. However, a clinical and endoscopic follow-up of these lesions is always advisable[[93](#_ENREF_93)], also for the risk of progression in a long-run term[[92](#_ENREF_92)].

Similarly to T-LPD, also NK cells can give raise to an indolent form of lymphoid infiltrate in the context of the GI tract, *i.e.,* NK-cell enteropathy[[94](#_ENREF_94)]. Usually the symptoms are vague and the GI lesions can be present at stomach (more frequently), duodenum, small intestine, and colon. At endoscopy, these lesions exhibit superficial ulceration, flat elevations with central depression and are associated with edema and local hemorrhage. Usually these ulcers are 1cm in diameter and the surrounding mucosa is not abnormal. This disease is distinct from ENKTL since gastric involvement in the latter is really infrequent (and if present the localization is not limited to the stomach) and EBER is positive. In addition, in presence of NK-enteropathy, the epithelium can be invaded, showing a lymphoepithelial-like lesion[[95](#_ENREF_95)]. Moreover, contrarily to T-LPD, TCR rearrangement is polyclonal or oligoclonal[[94](#_ENREF_94)].

# Lymphomatous polyposis and Mantle Cell Lymphoma

The pioneer study by Corner *et al*[[96](#_ENREF_96)] in 1961 firstly reported the term “Lymphomatous polyposis”(LP). It is defined as the presence of diffuse proliferation of monotonous small-to-intermediate sized lymphocytes presenting as multiple polypoid tumors from 2 mm to several centimeters in different GI sites. Even if the preferred site is the small intestine[[14](#_ENREF_14)], other sites can be involved alone or at the same time[[97-104](#_ENREF_97)]. Actually, LP is present in 4%-9% of all GI Lymphomas[[14](#_ENREF_14)] resulting more frequent in Western compared to Eastern Countries[[105](#_ENREF_105)]. B-cell lymphomas are more frequent than T-cell lymphomas and this is due to the fact that histologically these polyps originates from the mantle zone of the lymphoid follicle of the Mucosa-associated lymphoid tissue[[106](#_ENREF_106)]. Additionally, this fact justifies the augmented frequency in small intestine (rich in lymphatic tissue) compared to other GI tracts. Additionally, multiple tumors or different kind of lymphomas can be simultaneously present in a context of LP[[107](#_ENREF_107)]. Therefore, the biopsy of more than one polyp and of different types of lesions is always advisable[[108](#_ENREF_108),[109](#_ENREF_109)]. Additionally, it must be underlined that the occurrence of multiple polyposis in a patient with nodal lymphoma is not criterion to absolutely define the involvement of the GI tract, being the histological evaluation always mandatory[[110](#_ENREF_110)].

Typical lymphoma presenting with LP is MCL[[14](#_ENREF_14),[111](#_ENREF_111)], even if other tumors can show this feature[[98-100](#_ENREF_98),[112-114](#_ENREF_112)]. Among 37 case reports of LP since 2000[[67](#_ENREF_67),[84](#_ENREF_84),[98-100](#_ENREF_98),[103](#_ENREF_103),[112-142](#_ENREF_112" \o "Hirata, 2007 #150)], indeed MCL was the most frequent disease (more than 50% of cases) (Figure 2). The most involved site was the colon (Figure 3). In the case series by Saito *et al*., regarding patients affected by MALT lymphomas or MCL at the ileal site, it has been underlined that LP was the most frequent presentation of MCL and the least common lesion in MALT lymphomas (Figure 4)[[143](#_ENREF_143)].

MCL can locate at the GI tract secondarily to the generalized disease[[102](#_ENREF_102)] and, even if only 25% of patients with nodal mantle cell lymphoma suffers GI symptoms, 77%-88% has a localization at the gross intestine and 43%-77% in the upper GI tract also in absence of macroscopic lesions[[14](#_ENREF_14)] (Figure 5). LP is the most frequent endoscopic pattern even if other endoscopic features can be present[[144](#_ENREF_144)], for example granular pattern associated with polyps (Figure 6) or ulcerated polyps[[145](#_ENREF_145)] or masses[[146](#_ENREF_146)]. In addition, the endoscopic pattern varies according to the GI tract involved (Figure 7). EUS has been applied in this setting giving the possibility to identify submucosal lesions[[115](#_ENREF_115)]. MCL appears echopoor, usually departing from the second layer, remaining confined to the GI wall (Figure 8)[[115](#_ENREF_115),[147](#_ENREF_147),[148](#_ENREF_148)]. In some cases the diagnosis of MCL could be incidental during the endoscopic definition of gastric bleeding caused by gastric ulcers[[149](#_ENREF_149)].

Contrarily to GI Follicular Lymphoma (discussed below), the GI tract involvement by MCL assumes a great prognostic implication and is useful to monitor patients after the treatment[[14](#_ENREF_14),[101](#_ENREF_101)]. Indeed, the occurrence of LP designate a median survival of 3-4 years[[14](#_ENREF_14),[101](#_ENREF_101)]. The fact that the small intestine can be also involved by the tumor, the performance of CE or DBE would be advisable in order to correctly stage the patient and assess the follow-up evaluations[[116](#_ENREF_116),[117](#_ENREF_117)].

Even though the disease presentation has been well studied, there are no data about the management of LP during follow-up assessment. Our opinion is that the endoscopic evaluation with mapping biopsies should be performed in these patients since in some cases the presence of aspecific abnormalities during follow-up can lead to the finding of lymphoma reappearance[[146](#_ENREF_146)], sometimes after many years from complete remission[[103](#_ENREF_103),[119](#_ENREF_119)].

# Follicular Lymphoma

GI FL is a rare entity, representing up to 3.6% of all GI NHL[[150](#_ENREF_150),[151](#_ENREF_151)]. Primary GI FL has been recognized as an histological variant of FL in the 2008 WHO classification of haematopoietic tumors[[152](#_ENREF_152)]. Sites most frequently involved are the duodenum (55.6% of cases)[[101](#_ENREF_101)], in particular the second part[[152](#_ENREF_152)], and the terminal ileum (33.3% of cases)[[151](#_ENREF_151),[153](#_ENREF_153)]. Since Positron Emission Tomography and Computed tomography have low sensitivity and specificity[[154](#_ENREF_154)] in catching small intestine involvement, CE and DBE have acquired more and more importance[[155](#_ENREF_155),[156](#_ENREF_156)]. Indeed, these techniques showed that small bowel can be involved in 70%-83% of cases[[157](#_ENREF_157),[158](#_ENREF_158)] even in cases of duodenal lymphoma[[152](#_ENREF_152)].

To date, a clear endoscopic classification of GI-FL could not be done, as for GI MALT lymphomas. However, Yamamoto *et al*[[151](#_ENREF_151)], reviewing 249 GI-FL cases, reported a reliable endoscopic classification of the disease. Whitish polyps usually up to 2 mm[[151](#_ENREF_151),[153](#_ENREF_153)] and/or white granules-nodular aggregates, with or without ulceration of the mucosa layer (Figure 9), represent the typical endoscopic pattern[[150](#_ENREF_150),[159](#_ENREF_159),[160](#_ENREF_160)]. This can be unifocal or multifocal and it is mainly present in intestinal FL. Large mass with or without ulceration is less frequent and in half of cases can be associated with multifocal whitish polyps. The latter is the most frequent endoscopic pattern of primary gastric FL. Multiple lymphomatous polyposis can also be found[[30](#_ENREF_30),[101](#_ENREF_101),[150](#_ENREF_150),[158](#_ENREF_158),[161](#_ENREF_161),[162](#_ENREF_162)]. Interestingly, in the series of 48 patients with GI FL reported by Yanai *et al*[[163](#_ENREF_163)] it has been found that the LP was the most frequent endoscopic feature (more than 50% of cases) followed by polypoid or ulcerative lesions (Figure 10).

Recently, high-definition endoscopy, as well as Magnifying Endoscopy (ME), has been used to describe the surface micro-structures of GI FL such as enlarged whitish villi and tiny whitish depositions and irregular microvascular pattern[[164](#_ENREF_164),[165](#_ENREF_165)]. This fact indicates that the tumor is of not-epithelial origin and usually reflects the formation of lymphoid follicles[[164](#_ENREF_164),[166-169](#_ENREF_166)]. EUS has not widely applied. Few reports have indicated that the echoendoscopic pattern is given by second and third layer thickening, dotted by hypoechogeneic nodules[[170](#_ENREF_170)].

Capsule endoscopy and double-balloon enteroscopy are useful in the definition of small intestine involvement in an non-invasive way. The typical picture is a whitish submucosal elevation presenting as nodules or polyps[[21](#_ENREF_21)], usually multifocal[[171](#_ENREF_171),[172](#_ENREF_172)]. However, the limitation is given by the inability to perform biopsy that is postponed to the execution of enteroscopy and the risk of retention in cases of stenosis (unusual in case of GI-FL).

Nodal spread is rare and GI FL tends to be localized in the gastrointestinal tract (stage IE according to Ann Arbor staging system)[[173](#_ENREF_173)] and to have an indolent course[[152](#_ENREF_152),[174](#_ENREF_174)]. However, transformation to aggressive lymphoma has been documented[[175](#_ENREF_175)]. Differently from other form of lymphomas, the GI involvement is not an adverse prognostic factor[[176](#_ENREF_176)]. Lymphoma grading is low in the majority of cases, while in the nodal counterpart grade I-II FL is documented in 1 case out of 10[[173](#_ENREF_173)]. Furthermore, in contrast to nodal FL, these cells do not acquire additional mutations and this justify the absence of grade 3 GI FL and the very low rate of transformation[[173](#_ENREF_173),[175](#_ENREF_175)].

Treatment strategies are not uniform, even if GI FL are treated more frequently compared to nodal counterpart[[177](#_ENREF_177)]. Different case series have demonstrated that watch and wait approach is as useful as the pharmacological approach, except for relieving clinical symptoms[[163](#_ENREF_163),[178-180](#_ENREF_178)]. However, case series differ greatly in identifying the correct treatment approach to be applied. The surgical resection is not recommended while chemoimmunotherapy should be preferred[[151](#_ENREF_151),[171](#_ENREF_171)]. It must be considered also that the introduction of anti CD20 antibodies has augmented the survival rate and in some series localized/low-grade GI FL have been treated with anti CD20 monoclonal antibody alone, without chemotherapy[[151](#_ENREF_151)].

It is debatable whether CE and/or DBE are truly useful. Indeed, no studies have demonstrated that the detection of small bowel involvement (especially if duodenal lymphoma is present) would have changed the treatment needed. Surely, these procedures would change the treatment strategy in case of radiation or surgical treatment and are needed in cases of obscure gastrointestinal bleeding[[172](#_ENREF_172),[181](#_ENREF_181)]. Apart these occurrences, the effectiveness of chemoimmunotherapy or immunotherapy alone would render these procedures not so practical in patient management. However, since no clear data exists regarding survival and quality of life in dependence of small bowel involvement, clinician choice is the only way to proceed.

That notwithstanding, the diagnostic suspect based on the endoscopic feature, together with the patient history, is fundamental in addressing the pathological diagnosis. Indeed, in almost 20% of cases, FL can be misdiagnosed by endoscopic biopsy evaluation[[182](#_ENREF_182)]. Therefore, multiple biopsies would be necessary. In particular, biopsies of the peripheral mucosa would be more informative than biopsies from the erosion/ulceration, since the probability to catch necrotic tissue decreases significantly.

# Extramedullary Plasmacytoma and Plasma Cell-related Diseases

Extramedullary Plasmacytoma (EMP) belongs to a precise type of lymphoid malignancies, *i.e.,* Plasma Cell Neoplasms, among whose represent 3%-4% of cases[[183](#_ENREF_183)]. It is important to distinguish this subtype from lymphomas with plasmacytic differentiation, particularly MALT lymphomas[[48](#_ENREF_48)]. The upper respiratory tract is the most involved organ (almost 80% of cases), while GI localization is rare[[48](#_ENREF_48)]. Among these cases, the stomach is the most involved site, followed by the liver, colon and the small intestine (duodenum, jejunum and ileum)[[184](#_ENREF_184)].

Usually, gastric localization is secondary to a plasma cell myeloma (PCM) and often emerges through a clone selection process. Indeed, Multiple Myeloma treatment itself can select a particular chemo-resistant PC clone able to migrate at extra-nodal organs. In these cases, an accurate endoscopic investigation is critical for the diagnostic assessment and disease monitoring[[185](#_ENREF_185)]. Due to the strict relationship with plasma cell myeloma, the clinical course is poor. Most frequent endoscopic finding consists of an infiltrating mass or masses in the stomach and/or the duodenum[[186](#_ENREF_186),[187](#_ENREF_187)] or well-demarcated, flat, yellow-whitish mucosal changes[[188](#_ENREF_188)] or nodular lesion with central umbelication[[189](#_ENREF_189)]. Endoscopic appearance as diffusely thickened mucosal folds simulating linitis plastica is rare[[190](#_ENREF_190)]. Sometimes, large ulcerations can be seen[[191](#_ENREF_191),[192](#_ENREF_192)]. However, gastric mucosa can appear normal, while the extramural growth is incredibly vast (Figure 11). EUS could be of great help in defining the disease extension that appears as a large echo-poor mass infiltrating surrounding organs[[186](#_ENREF_186)]. However, sometimes, EUS can be useful to detect limited gastric wall involvement and in these cases an endoscopic resection of the mass can be performed resulting safe for the patient and effective for the treatment of the disease[[188](#_ENREF_188),[193](#_ENREF_193),[194](#_ENREF_194)]. Alternatively, patients with localized disease can be treated with radiation treatment[[190](#_ENREF_190),[195](#_ENREF_195)].

Small intestine involvement is generally primary and have a benign course. These lesions can be explored by Enteroscopy and/or Capsule Endoscopy[[196](#_ENREF_196)] paying attentions to the cases in which obstructions or retention are expected. Differential diagnosis is with other cases of sub-mucosal masses in the small intestine, as reported by Lopes da Silva[[196](#_ENREF_196)]. Colon involvement appears more frequently as stricture[[197](#_ENREF_197),[198](#_ENREF_198)] in some cases difficult to differentiate from colon adenocarcinoma[[199](#_ENREF_199)], returning in the differential diagnosis of sub-mucosal tumors[[193](#_ENREF_193)]. Rarely it can determine rectal bleeding[[200](#_ENREF_200)]. The localization at rectum appears as a mild granularity as well as a reddish, protruded lesion[[201](#_ENREF_201)]. Usually these lesions disappear after treatment and this is a confirmation of treatment efficacy[[186](#_ENREF_186)], even if mucosal atrophy and non-specific inflammation can resituate[[195](#_ENREF_195)].

Apart EMP, other Plasma Cell-related disorders can involve the GI tract. This is due to the production of amyloid protein in AL Amyloidosis (Light chain Amyloidosis)[[187](#_ENREF_187)]. The most involved organ is the small intestine. In some cases the amyloid deposition is synchronous with EMP[[187](#_ENREF_187),[193](#_ENREF_193),[195](#_ENREF_195)] or other GI lymphomas[[202](#_ENREF_202)]. Usually, the amyloid protein in AL amyloidosis involves the submucosa and the muscolaris mucosae, resulting in thickened folds and valvulae conniventes and polypoid lesions in GI tract. The typical deposition of AL amyloid protein justify pseudo-obstruction, constipation and mechanical obstruction as main symptoms[[203](#_ENREF_203)]. Intestinal bleeding can also occur[[204](#_ENREF_204)] and, if this event occurs in a patient with Multiple Myeloma, the occurrence of aspecific elevated lesions at the endoscopic evaluations should lead to the suspect of systemic Amyloidosis. More rarely, submucosal hematoma, ulcers and hemorrhagic bullous colitis can be seen[[205](#_ENREF_205)]. On the other hand, nodularity, fine granular appearance and mucosal friability are more frequent in another type of Amyloidosis, *i.e.,* AA Amyloidosis (Amyloidosis secondary to systemic disorders). This is due to the deposit of amyloid proteins into the lamina propria with impaired absorption and subsequent diarrhea[[203](#_ENREF_203)].

# Immunodeficiency and GI lymphomas

Immunodeficiency is defined as a state of impaired function of the immune system that can be congenital, acquired or iatrogenic. The reduced immune-surveillance can determine an augmented rate of lymphomas. Two conditions mainly determine the arising of lymphomas: Human immunodeficiency virus (HIV) infection with the correlated Acquired Immuno Deficiency Syndrome (AIDS) and post-transplant immune-suppression. In both conditions, GI tract is the most involved site[[206](#_ENREF_206)]. Apart HIV and PTLD, Common variable immunodeficiency (CVID) has been related with the development of gastrointestinal NHL, even if this is a very rare finding[[207](#_ENREF_207),[208](#_ENREF_208)].

In HIV patients, the rate of GI lymphomas was higher in pre-HAART era, before 1996[[209](#_ENREF_209)] and the risk of gastric NHL was 353 fold compared to normal subject, being aggressive lymphomas the most prevalent features[[59](#_ENREF_59)]. In cases of AIDS-related lymphoma, GI tract is involved in 20% up to 50% of cases[[206](#_ENREF_206),[210](#_ENREF_210)]. However, the decrease of GI lymphoma incidence has not been so high compared to Central Nervous System Lymphomas[[209](#_ENREF_209)]. A recent analysis on 243 HIV patients performed at University of Sao Paolo revealed an incidence of Gastric NHL of 2.5%[[211](#_ENREF_211)]. Co-infection with EBV and/or CMV would complicate the prognosis[[212](#_ENREF_212)], even if the occurrence of viral infection is less pathogenetically important compared to PTLD[[206](#_ENREF_206)]. The main histologies are B-cell lymphomas (67%) (DLBCL, Burkitt lymphoma, MALT lymphoma)[[213](#_ENREF_213)] while T-cell lymphomas are less frequent (33%)[[209](#_ENREF_209)] and anecdotal are other types of hematologic malignancies[[214](#_ENREF_214),[215](#_ENREF_215)]. In 5%-10% of cases cMyc rearrangement is present and confers a poor prognosis[[212](#_ENREF_212)]. Additionally, the prompt recognition of this lymphoma subtype has a great impact in patient management since presenting symptoms are usually alarm symptom in about half of patients. However, in majority of patients, the lymphoma is diagnosed at stage III-IV AnnArbor[[206](#_ENREF_206)]. Most frequent endoscopic features are multifocal ulcerations, followed by polypoid or bulky mass together with bloody spots[[206](#_ENREF_206),[212](#_ENREF_212)]. The most involved sites are the stomach and the duodenum[[216](#_ENREF_216)] followed by small bowel and colon-rectum (Figure 12)[[211](#_ENREF_211)]. However, unusual presentations can be seen more commonly than in immunocompetent patients[[206](#_ENREF_206)]. At narrow-band, honeycomb-like pattern is present without irregularity in microvasculature[[212](#_ENREF_212)]. The localization can also be perirectal and in these cases, EUS-guided fine needle biopsy would be a valid tool to pone diagnosis given the high grade nature of this kind of lymphomas[[217](#_ENREF_217)]. Noteworthy, EUS appearance is of hypoechoic poorly defined mass[[217](#_ENREF_217)] and is important for the loco-regional staging[[206](#_ENREF_206)]. Prognosis is poor with a median survival of 6 months with a rate of complete remission less than 40%[[211](#_ENREF_211)]. Prognosis is also impaired by the occurrence of opportunistic infections[[210](#_ENREF_210)]. Extremely suggestive, is the development of EBV-related DLBCL in patients suffering other types of lymphomas that induce a state of immunosuppression, such as AITL[[218](#_ENREF_218)]. In these peculiar cases, the outcome is really poor and alarm symptoms and perforation can occur with fatal implications[[218](#_ENREF_218)].

GI lymphomas are also more frequent in solid organ transplant recipients, particularly after renal and heart and small bowel transplantation, encompassing the spectrum of PTLD (Table 3). The pathogenetic events seem to be different compared to HIV-related lymphomas since in this kind of lymphomas Epstein-Barr Virus re-activation due to immunosuppressants plays a pivotal role[[219](#_ENREF_219)]. Apart negative EBV serology prior to transplantation, length of immunosuppression is an overt risk factor[[220](#_ENREF_220),[221](#_ENREF_221)]. EBV-positive lymphomas arise earlier than EBV-negative lymphomas[[221](#_ENREF_221)]. In adults, majority of cases arises beyond 12 mo from transplantation[[222](#_ENREF_222)], at a median of 36 mo[[223](#_ENREF_223)]. A second peak is after 5-10 years[[206](#_ENREF_206)]. Median overall survival is 8 years and the principal histotype is B-cell lymphoma, even if lymphomas of T-cell origin can also be present. Noteworthy, the GI tract is involved in one third of cases. Endoscopy is of great help in establishing the diagnosis. Specially in small bowel transplantation, endoscopic follow-up has gained a pivotal role in defining the transplant-related complications, including the onset of PTLD[[224](#_ENREF_224)]. Typically, lesions are raised rubbery erythematous or ulcerated[[222](#_ENREF_222),[225](#_ENREF_225),[226](#_ENREF_226)]. Most involved organ is the colon, followed by small intestine and stomach[[223](#_ENREF_223)]. However, the recognition of symptoms together with the patient history is of great help in driving the diagnosis. Additionally, endoscopic procedures are essential in order to follow the course of disease[[225](#_ENREF_225)], being valid also in long-run term[[226](#_ENREF_226)]. Interestingly, early stage PTLD can be safely removed endoscopically and this would be a valid approach in the treatment of localized PTLD[[224](#_ENREF_224)].

Plasmablastic lymphoma (PBL) is a rare and aggressive type of lymphoma characterized, at histological valuation, by the presence of large immunoblasts with plasmacytic differentiation with an high replication index[[227](#_ENREF_227)]. Usually, this lymphoma arises in the oral cavity in HIV-infected patients and in literature there are few cases of GI localization (Table 4)[[228-237](#_ENREF_228)]. Stomach is the most involved site (about 50% of cases), followed by small intestine, anal region, cecum, colon and esophagus[[237](#_ENREF_237)]. Large masses and exophitic processes are the main endoscopic appearance in stomach and anal region. Intestinal localization is extremely rare and when present the endoscopic appearance is of multiple nodularity[[227](#_ENREF_227)]. Moreover, PBL can arises also in immunocompetent patients with ulcerated lesions at the stomach[[236](#_ENREF_236)]. These patients are normally older than HIV+ patients, tend to present GI localizations more that HIV+ patients and show a worse overall survival[[236](#_ENREF_236),[237](#_ENREF_237)].

Additionally, CD has been also linked to the development of lymphomas of the gastrointestinal tract. Most of them are of B origin, comprising DLBCL and HL, even if also T-cell lymphomas can arise. In the recent report by Kappelman *et al*[[238](#_ENREF_238)] patients with CD showed a greater risk to develop haematological malignancies compared to general population. This study confirmed the previous report by Askling *et al*[[239](#_ENREF_239)], showing also an augmented rate of haematological malignancies compared to general population and 10% of developed lymphomas where of T-type. Probably it would be related to the state of immunosuppression leading to infection of lymphotropic and oncogenic viruses, but the specific mechanism remains to be clarified. This predisposition seems to be unrelated to the immune-suppressive treatment. In this setting, anti-TNF treatment have been related to development of hepatosplenic T-cell lymphoma[[240](#_ENREF_240)]. However, two years later, a metanalysis by Siegel *et al*[[241](#_ENREF_241)] indicated that the immunosuppressive treatment is not a risk factor for the development of NHL in CD patients. However, it is still a matter of discussion since the augmented incidence of GI lymphomas in these patients would be related to the more intensive examinations. Moreover, the histological evaluation is a crucial point since the inflammatory background can lead to a false positive result. That notwithstanding, anti-TNF treatment seems to be safe regarding the incidence of NHL and should not be regarded as a risk factor. Therefore, more epidemiological studies will be needed in order to better define the link between CD and GI lymphomas.

## Hodgkin’s Lymphoma

Lymphomatous GI involvement in HL appears as a stricture (Figure 13) or ulceration[[242-245](#_ENREF_242)].The abundant lymphoid tissue, present at this site, renders it one of the most involved region[[246](#_ENREF_246)]. HL rarely present a colonic localization (almost 1%-3% of extra-nodal HL cases[[247](#_ENREF_247)] and less than 5% of gastrointestinal lymphomas[[243](#_ENREF_243)]) and the prognostic impact is still obscure. Mixed-cellularity subtype represents the most common feature[[248](#_ENREF_248)]. As for nodal counterpart, the inflammatory background represents a key feature of HL[[249](#_ENREF_249)]. In some cases, the endoscopic and histological presentation can resemble inflammatory bowel disease (IBD), that, in turn, can seldom be associated with colonic HL[[244](#_ENREF_244),[250](#_ENREF_250)]. Additionally, immunodeficiency represents a risk factor[[251](#_ENREF_251)], even if this type of lymphoma can also arise in immune-competent patients[[247](#_ENREF_247)].

Recently, a new entity has been proposed, *i.e.,* ‘‘EBV Associated Mucocutaneous Ulcer” (EBVMCU)[[252](#_ENREF_252)]. This disease subtype would resemble HL, but there are peculiar clinical and histological differences from HL. Indeed, the presence of ‘‘plasmacytoid’’ apoptotic cells and the confinement to mucosa and sub-mucosal layers are the histological hallmark that can lead to a differential diagnosis from cHL. However, EBV infection is always present as in GI-HL.

# CONCLUSION

Endoscopic features of GI lymphomas are variegated encompassing ulcers, erosions, polyps and so on. It represents a fascinating matter of study for both hematologists and gastroenterologists. As stated in guidelines, a synergism between these two figures is fundamental. This is due to the lack of data and the fact that information regarding rare GI lymphomas are extrapolated from case series or case reports. Actually, the scientific community is gaining more and more knowledges on the recognition and management of these lymphomas with the creation of proper guidelines for specific lymphoma subtypes. In this setting, the collection of different case series and their analysis will assume a pivotal role in drawing general guidance on disease characterization. Certainly, as emerged into the manuscript, the management of these lymphomas is different compared to nodal or medullary counterpart and a proper understanding of the endoscopic features together with clinical and histological characteristic is crucial in a better management of patients with the ultimate goal of improving clinical outcome and quality of life of patients.

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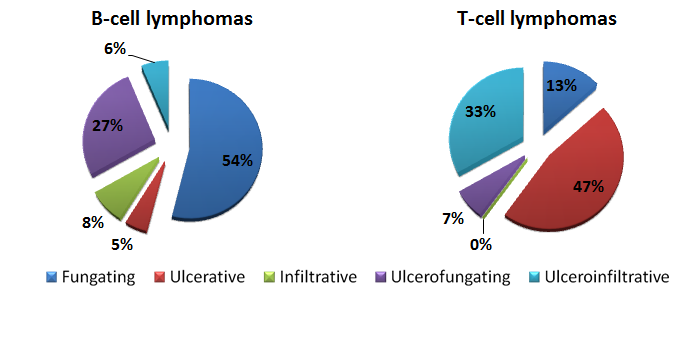
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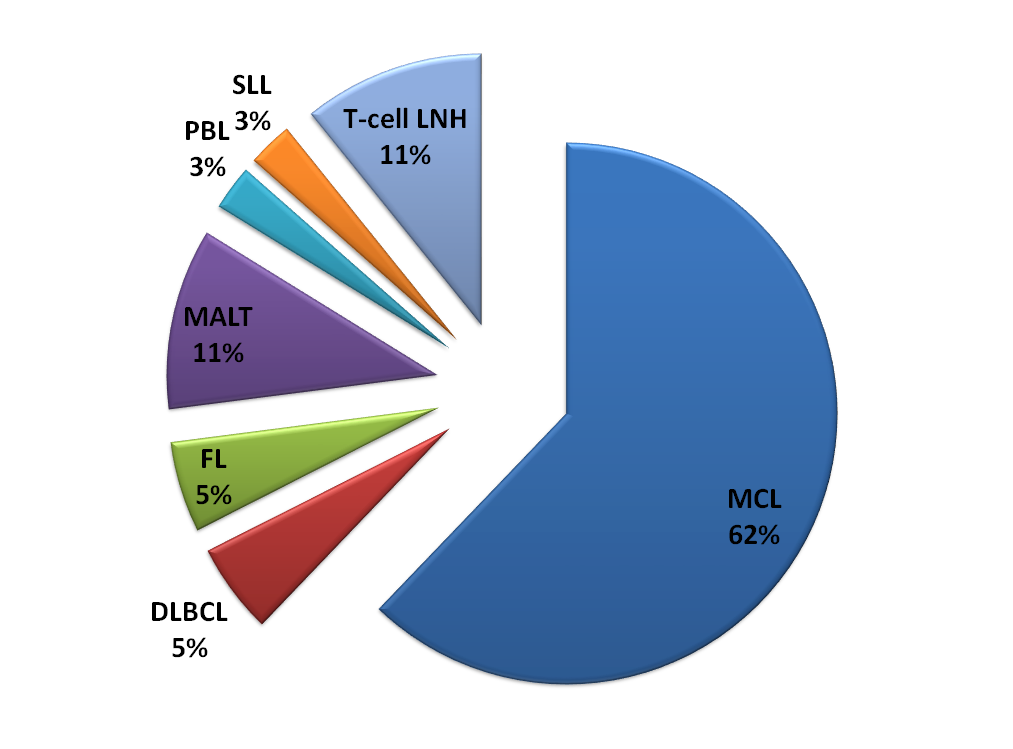
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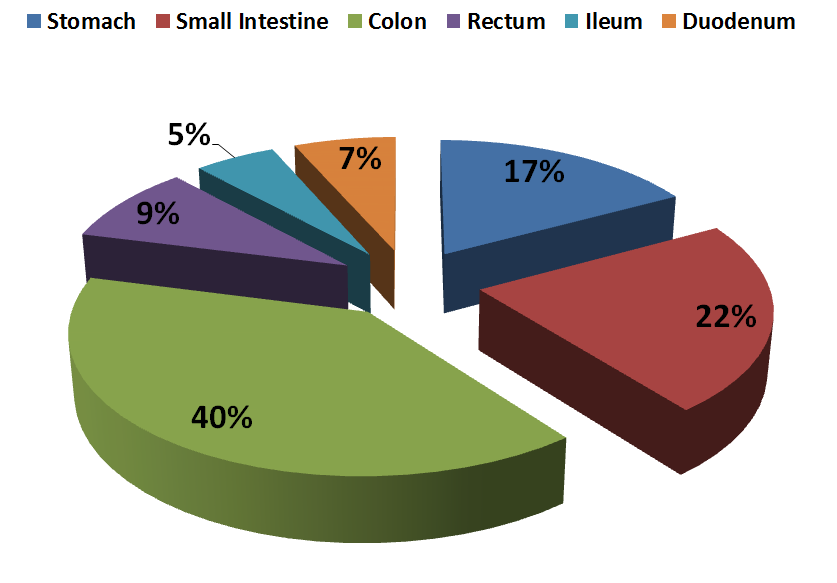
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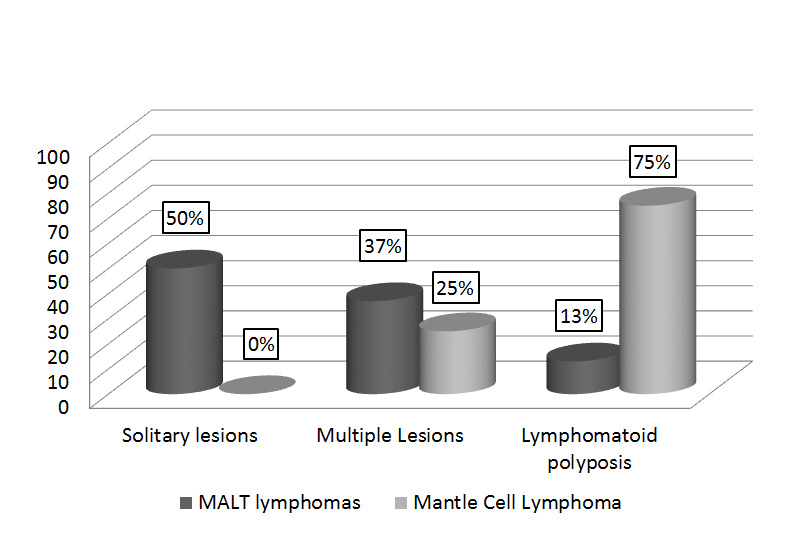
**Figure 1 Differences in endoscopic pattern between B-cell and T-cell lymphomas of the gastro-intestinal tract.** Data extrapolated from Kim *et al*[[19](#_ENREF_19)] 2005.



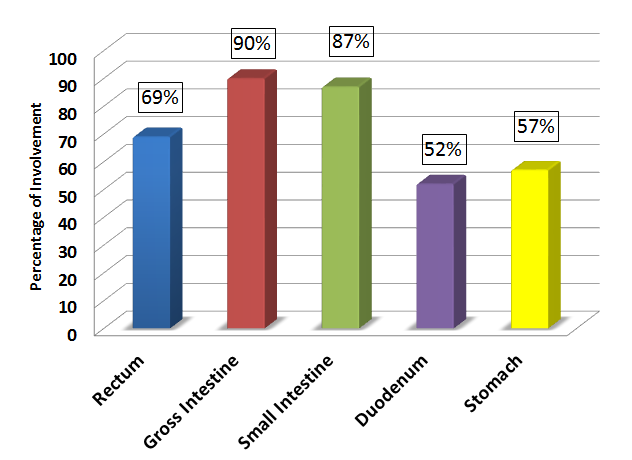
**Figure 2 Pie chart describing the distribution of lymphomatous polyposis as a presenting gastro-intestinal feature in gastro-intestinal non-hodgkin lymphomas according to histotype.**



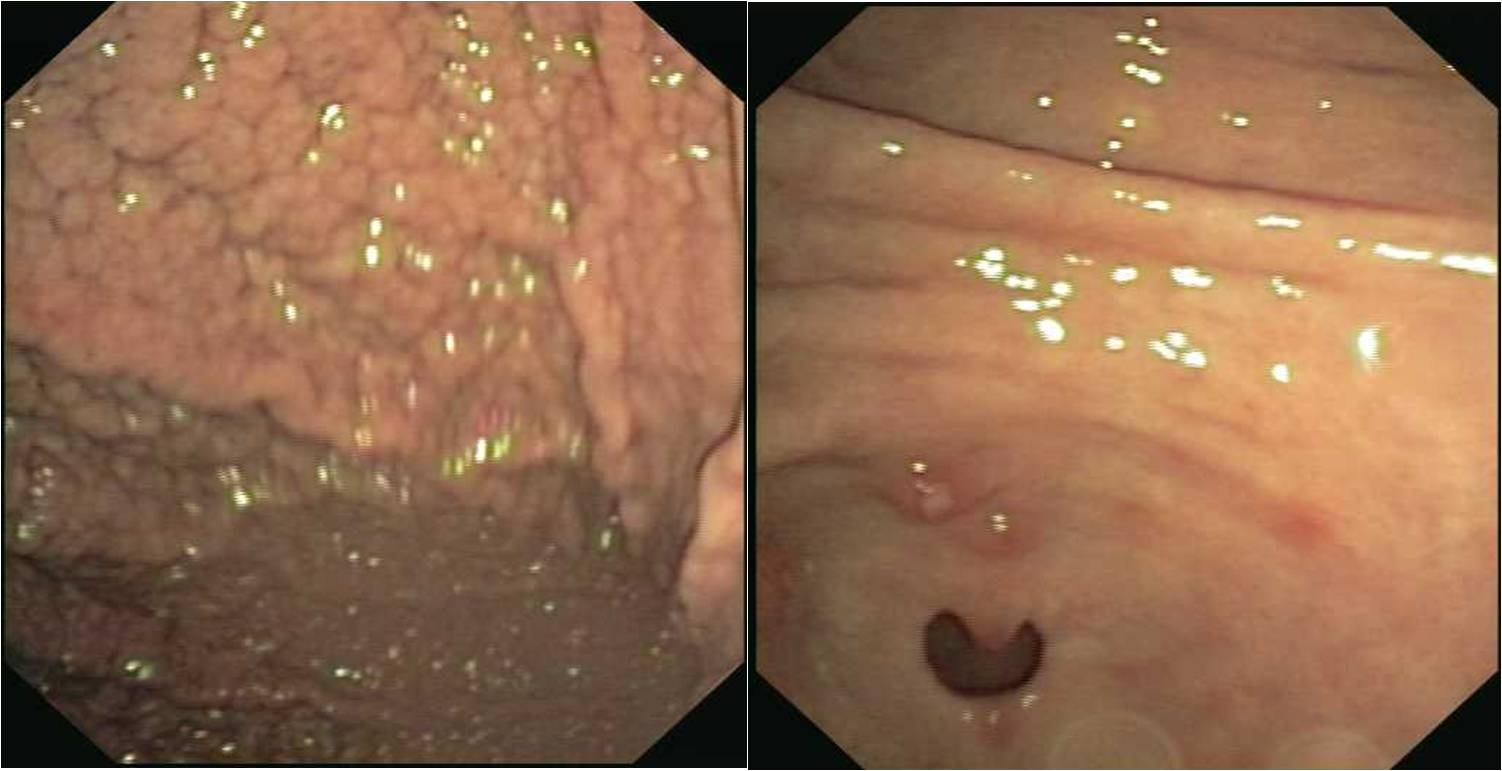
**Figure 3 Pie chart describing the most involved gastro-intestinal site in lymphomatous polyposis.**



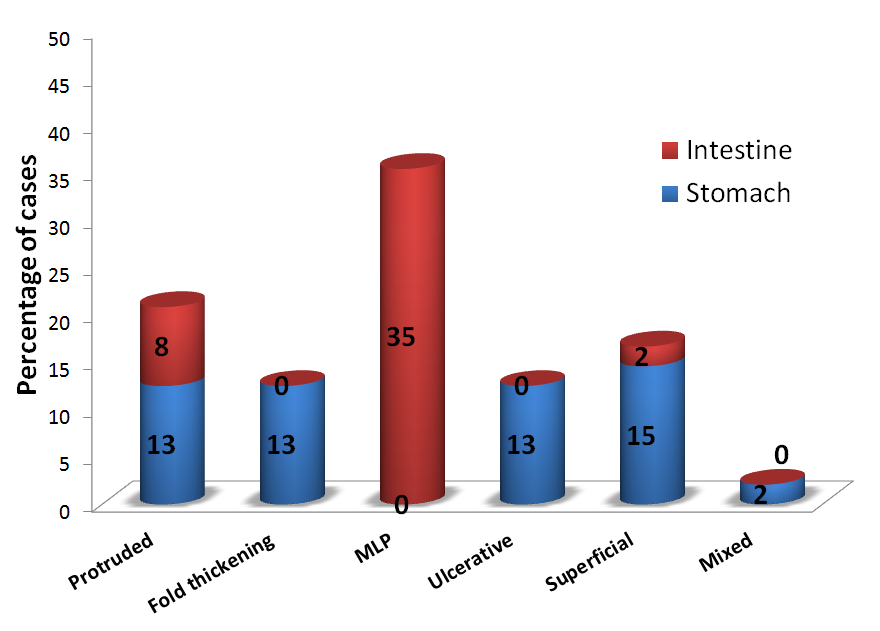
**Figure 4 Frequency of lymphomatous polyposis at ileum in mucosa-associated lymphoid tissue lymphoma and mantle cell lymphoma.** Adapted from Saito *et al*[[143](#_ENREF_143)], 2005.



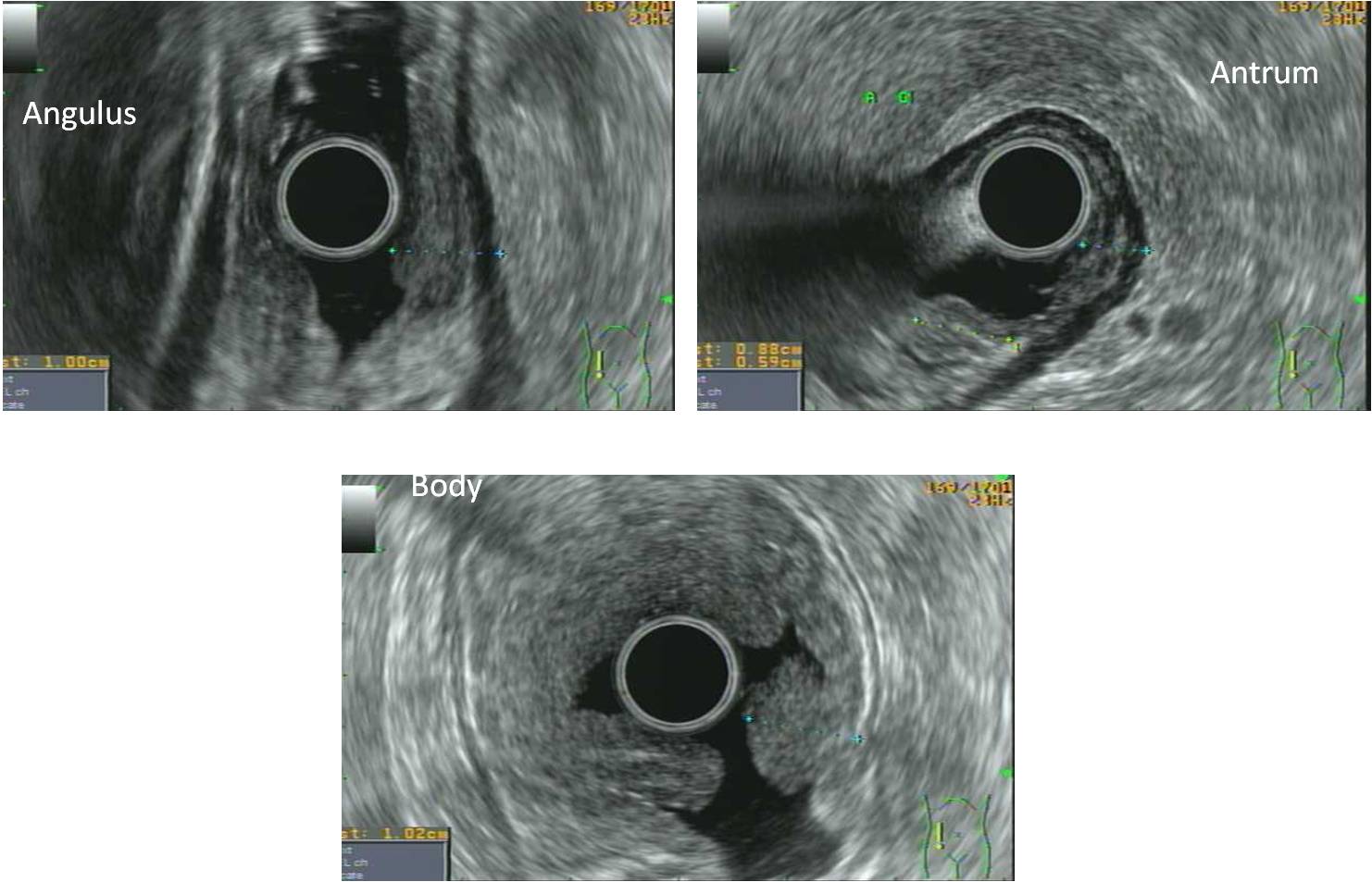
**Figure 5 Frequency of the sites involved in mantle cell lymphoma.** Adapted from Ruskone-Fourmestraux *et al*[[101](#_ENREF_101)], 2010.

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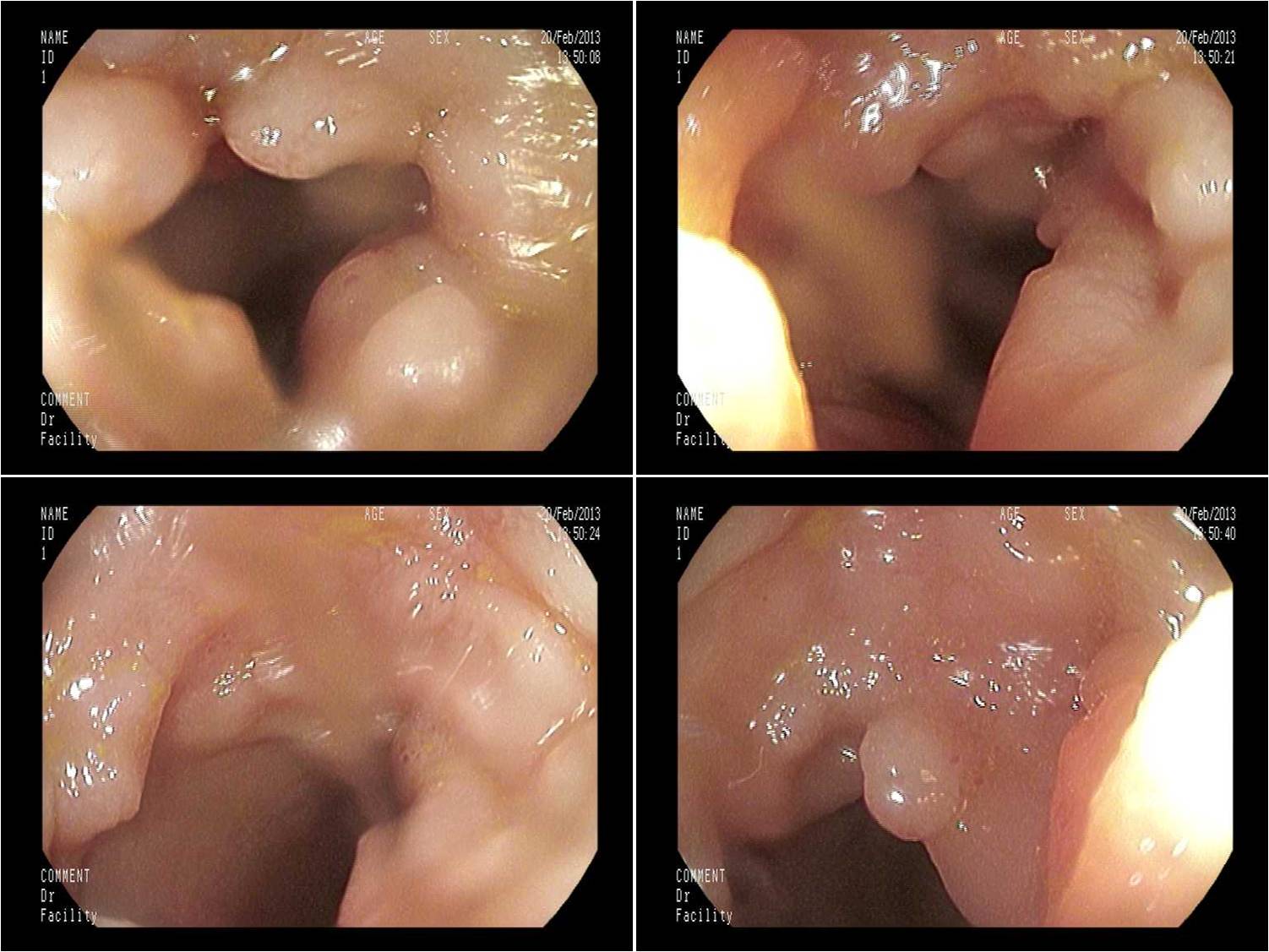
**Figure 6 Endoscopy Stomach: granular pattern of the fundus and body of the stomach (left side of the figure) and polyps in the Antrum (right side of the figure).**



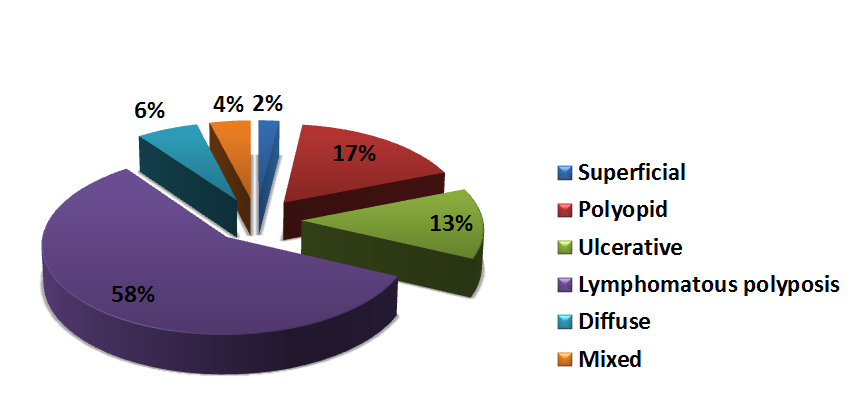
**Figure 7 Endoscopic lesions in mantle cell lymphomas according to the gastric and intestinal localization.** Adapted from Iwamuro *et al*[[144](#_ENREF_144)], 2010.

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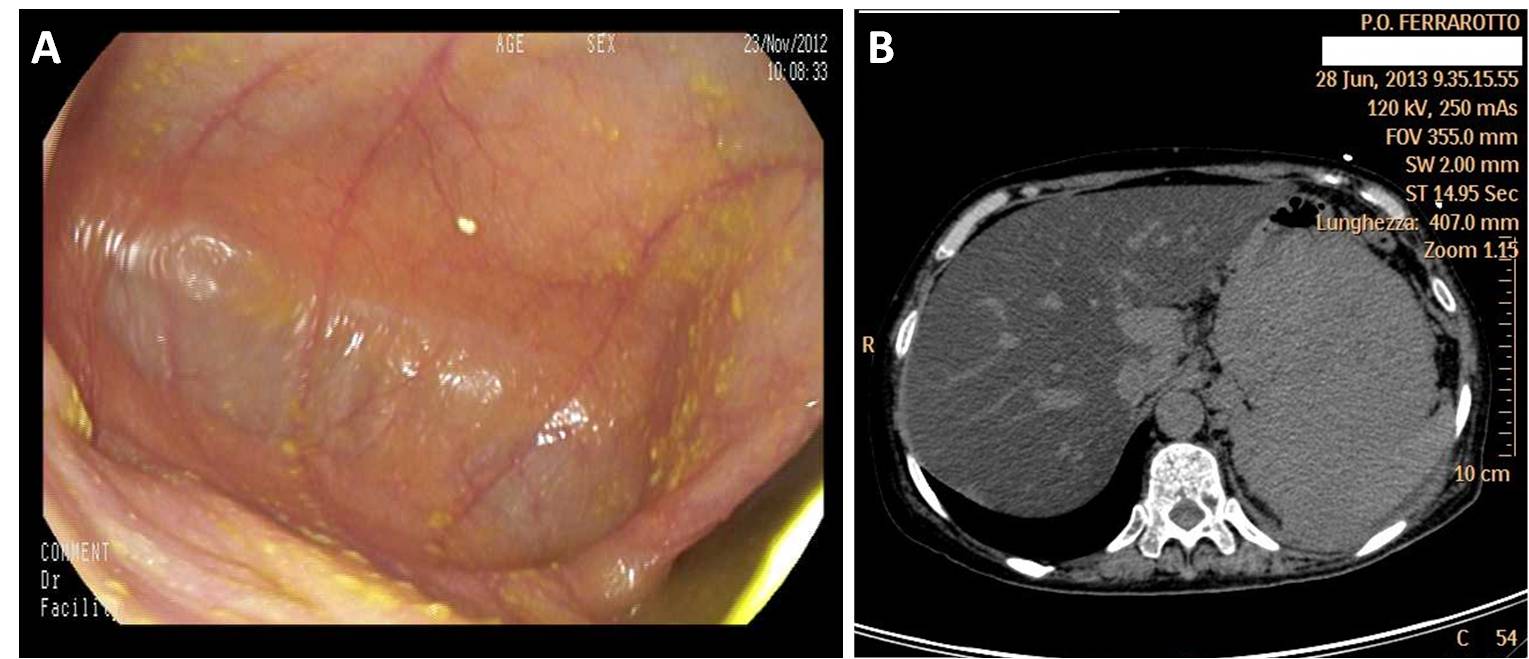
**Figure 8 endoscopic ultrasonography (Radial Scanning): marked thickening of the muscolaris propria and increased wall thickness (12 mm) in the Angulus (up-left); Antrum (up-right); Body (bottom).**

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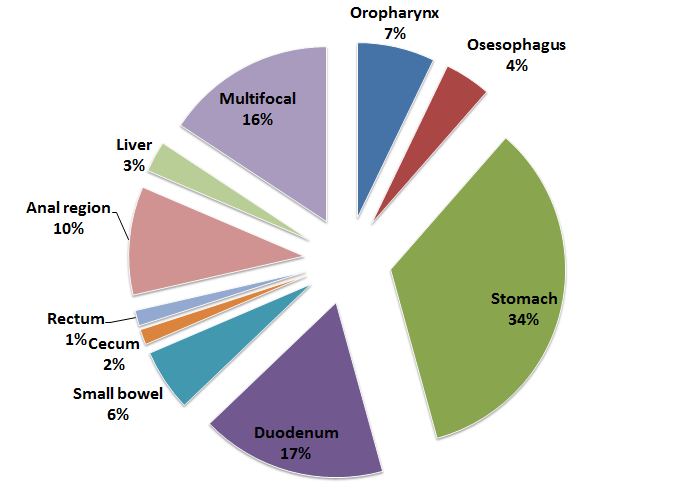
**Figure 9 Ileoscopy revealing the presence of hyperemic mucosa with whitish polypoid nodularity.** The subsequent diagnosis was a grade 2A Follicular Lymphoma.



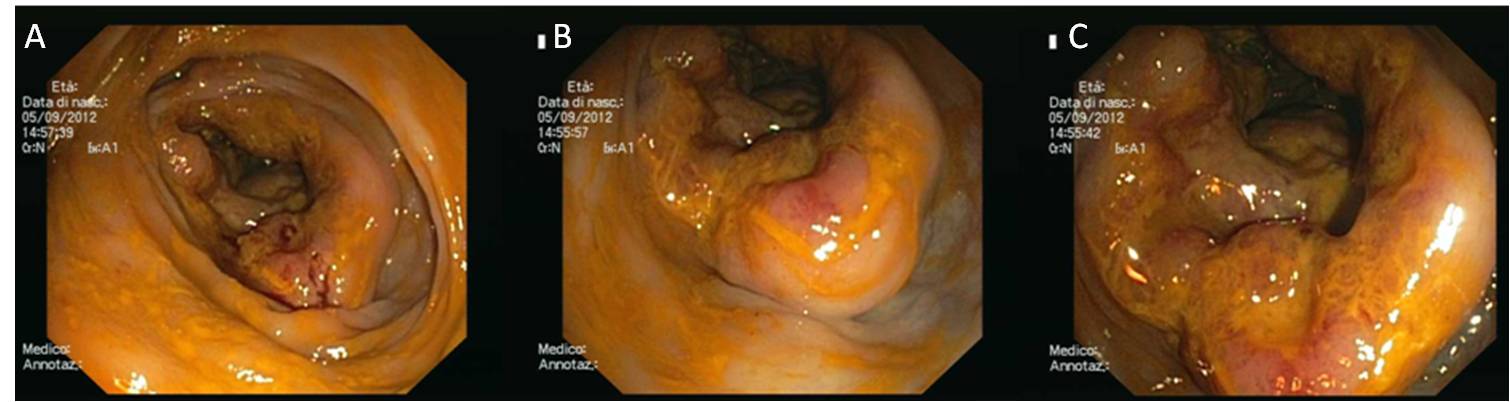
**Figure 10 Endoscopic features of follicular gastrointestinal lymphomas.** Adapted from Yanai *et al*[[163](#_ENREF_163)], 2011.



**Figure 11 Extra-medullary Plasmacytoma with gastric localization arising after treatment for multiple myeloma.** A: Gastroscopy resulted negative for tumor detection; B: CT scan analysis of the upper abdomen showing a bulky mass departing from the stomach.



**Figure 12 Frequency of the involved gastro-intestinal tract in human immunodeficiency virus-related gastro-intestinal lymphomas.** Adapted from Heise[[206](#_ENREF_206)], 2010.



**Figure 13 Exophytic erythematous cyrcumpherantial non-ulcerated mass determining a stenosis of the ileo-caecal region.** The mass arises from the deep layer and the mucosa presents reddish areas suggestive for lymphomatous infiltration of the caecum.

**Table 1 Endoscopic features of rare gastro-intestinal lymphomas according to two classification systems**

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
| **Author** | **Wang *et al*[18]** | | **Myung *et al*[17]** | |
| Number of patients | 13 | | 32 | |
| Endoscopic pattern | Mucosal – ulcerative | 30.7% | Fungating | 39% |
| Mucosal – erosive | 15.3% | Ulcerative | 6% |
| Polypoid | 23% | Infiltrative | 14% |
| Massive | 31% | Ulcerofungating | 31% |
|  |  | Ulceroinfiltrative | 11% |

**Table 2 Listing gastro-intestinal lymphoma with main gastro-intestinal organ involvement, typical presenting characteristics, typical immunophenotype and genotype and prognosis**

|  |  |  |  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- | --- | --- | --- |
| **Lymphoma histotype** | | | **Presenting characteristics** | **Main GI involvement** | **Main endoscopic pattern** | **Typical immunophenotype** | | **Typical genotype** | **prognosis** |
| T and NK lymphomas | EATL | | Celiac patients with abdominal pain and small intestine obstruction/perforation | Duodenum and jejunum | multiple erosions and ulcers | CD3+, CD4-, CD8+/-, CD7+, CD5-, CD2+, TIA+, GrBPer+, CD30-/+, CD25-/+, CD56-/+, CD16-, CD57-, BCL6-, CD10-, EBV-, EMA-/+ | | TRB and TRG clonally rearranged  +9q31.3  -16q12.1  +1q32.2-q41  +5q34-q35.2  +8q24(MYC) | Poor |
| PTCL | | Poor performance status | Stomach and Duodenum | Ulcerative | CD3+, CD4+, CD8-, CD7+, CD5+, CD2+, TIA-, GrBPer-, CD30-/+, CD25-, CD56-, CD16-, CD57-, BCL6-, CD10-, EBV-, EMA- | | 1TCR clonally rearranged  +7q/+8q/+17q/+22q/-4q  -5q/-6q/-9p/-10q/-12q/-13q | 54% survival at five year  Poor in case of high IPI score and stage III-IV disease |
| Extranodal NK/T-cell lymphoma | | Gastrointestinal bleeding and B symptoms | Small Intestine | multiple erosions and ulcers | cyCD3+, CD4-, CD8-/+, CD7-, CD5-, CD2+, TIA+, GrBPer+, CD30-, CD25-, CD56+, CD16-, CD57-, BCL6-, CD10-, EBV+, EMA- | | TCR in germinal configuration  No specific cytogenetic studies on this specific subtype | Poor especially if perforation occurs |
| Adult T-Cell Leukemia/Lymphoma | | abdominal pain, diarrhea, general  fatigue, weight loss | No site preferences | Ulcers | CD3+, CD4+, CD8-, CD7-, CD5+, CD2+, TIA-, GrBPer-, CD30-/+, CD25++, CD56-, CD16-, CD57-, BCL6-, CD10-, EBV-, EMA- | | TCR clonally rearranged  Monoclonal integration of HTLV-1 | Poor2  Good3 |
| Indolent lympho-prolipherative diseases of GI tract | T-LPD | dyspepsia and mild diarrhea | Small intestine and colon | Unremarkable/friable or erythematous mucosa | CD3+, CD4-, CD8+, CD7+/-, CD5+/-, CD2+, TIA+/-, GrBPer-/+, CD30-, CD56-, EBV- | TCR- monoclonal | | Indolent course |
| NK-cell enteropathy | Vague symptoms (dyspepsia) | Stomach and small intestine | lesions exhibit superficial ulceration, flat elevations with central depression and are associated with edema and local hemorrhage | cCD3+, CD4-, CD8-, CD7+, CD5-, TIA+, GrBPer+, CD56+, EBV- | TRC polyclonal or oligoclonal | | Indolent course |
| Mantle Cell lymphoma | | | Vague symptoms (dyspepsia) | Colon | Multiple polyposis, seldom with ulcerations | CD19+, CD20+, CD5+, CD10-, CD43+, sIg+, BCL6-, IRF4/MUM1-, Cyclin D1+ | | BCR rearranged  t(11;14)(q13;q32) | Negative impact on prognosis |
| Follicular lymphoma | | | Vague symptoms (dyspepsia) | Second part of duodenum | Whitish polyps | CD19+, CD20+, CD5-, CD10+, CD43-, sIg+, BCL6+, IRF4/MUM1-/+, Cyclin D1-, 47+ | | BCR rearranged  t( 14:18)(q32 :q2 1) | Good |
| Extramedullary Plasmacytoma | | | Alarm symptoms and obstruction | Stomach | Infiltrating mass | Plasmacells expressing CD79a+, CD38+, CD19-, CD138+, CD56+, usually CD20- | | BCR rearranged  t(11;14)(q32;q13) | Poor |
| PTLD | | | Alarm symptoms | Colon | rubbery erythematous or ulcerated | Similar to DLBCL  and Burkitt’s lymphoma  CD19+, CD20+, CD5-/+, CD10-/+, CD43-/+, sIg+/-, BCL6-/+, IRF4/MUM1-/+, Cyclin D1- | | Monoclonal BCR | Poor median survival 6 mo |
| Plasmablastic lymphoma | | | Alarm symptoms | Stomach | Large masses and exophitic processes | CD79a+, CD138+, CD38+, IRF4/MUM1, CD45-, CD20, PAX5-, CD56- | | Clonal IgH chain gene rearranqement | Poor |
| Hodgkin’s Lymphoma | | | obstruction | Colon | Protruding mass | CD30+, CD15+, CD45-, CD20-, CD79a-, PAX5+, Ig-, OCT2-, BOB1-, CD3-, CD2-, CD5-, ALK- | | clonal immunoglobulin  (IG) gene rearrangeme nts | Prognostic impact not known |

1estrapolated from nodal counterpart but not explored in Primary GI lymphoma; 2ATLL acute and lymphoma types; 3ATLL chronic and smoldering types. TCR: T-cell receptor; BCR: B-cell receptor; EATL: Entheropathy–associated T-cell lymphoma; PTCL: Perypheral T-cell lymphoma; T-LPD: T-cell lymph-prolipferative disease; NK: Natural killer; DLBCL: Diffuse large B-cell lymphoma; PTLD: Post-transplantation lymph-proliferative disease; GI: gastro-intestinal.

**Table 3 Prevalence of gastro-intestinal lymphomas among transplant receipts according to transplanted organ**

|  |  |
| --- | --- |
| Transplant | Prevalence |
| Bone Marrow | 0.5% |
| Liver | 1%-2% |
| Kidney | 0.7-4% |
| Heart | 2%-10% |
| Small bowel | up to 30% |

The data extracted from Heise[206], 2010.

**Table 4 Reports of gastro-intestinal plasmablastic lymphoma from 1998**

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
| Manuscript | Year | localization | Endoscopic appearance | HIV |
| Pruneri *et al*[[230](#_ENREF_230)] | 1998 | Stomach | Large polypoid mass | - |
| Colomo *et al*[[231](#_ENREF_231)] | 2004 | Anal region | Mass | + |
| Dong *et al*[[232](#_ENREF_232)] | 2005 | GI tract | Not reported | + |
| Small Intestine | Not reported | + |
| Tavora *et al*[[228](#_ENREF_228)] | 2006 | Anal region | Not reported | + |
| Anal region | Exophytic mass | + |
| Taddesse-Heath *et al*[[233](#_ENREF_233)] | 2010 | Small Intestine/Colon (2 cases) | Not reported | + |
| Brahmania *et al*[[234](#_ENREF_234)] | 2011 | ano-rectal junction | Hypervascular cauliflower-like mass | - |
| Mihaljevic *et al*[[235](#_ENREF_235)] | 2012 | Stomach | Not reported | - |
| Hashimoto *et al*[[236](#_ENREF_236)] | 2012 | Stomach | Not reported | - |
| Chapman-Fredricks *et al*[[229](#_ENREF_229)] | 2012 | Stomach | Not reported | + |
| Luria *et al*[[237](#_ENREF_237)] | 2014 | Anal region | Mass | + |
| Sigma | Mass | - |
| Small bowel | Not reported | - |
| Ileum | Not reported | - |

HIV: Human immunodeficiency virus; GI: gastro-intestinal.