The Detection of Premalignant and Malignant Gastric Lesions by Conventional Endoscopy in a General Population Sample

Boeriu Alina[1], Dobru Daniela[1], Pascarenco Ofelia[2], Mocan Simona[3]

[1] Department of Gastroenterology, University of Medicine and Pharmacy Targu Mures
[2] Department of Gastroenterology, Targu Mures County Hospital
[3] Department of Pathology, Targu Mures County Emergency Hospital

Background and Aims: The identification of patients with premalignant lesions and endoscopic surveillance could improve the early detection of gastric cancer, with better therapy and prognosis. We performed conventional endoscopy with biopsies to identify the incidence rates of premalignant and malignant gastric lesions and the risk of patients for this pathology.
Methods: A total of 1651 patients were investigated with conventional endoscopy. We took biopsies from 1493 patients. Biopsy specimens were analyzed for gastric inflammation, atrophy, intestinal metaplasia, dysplasia and neoplasia.
Results: We demonstrated that major symptoms had a sensitivity of 95.2%, and a specificity of 54.5% for the detection of gastric neoplasia, with a sensitivity of 61.6% and a specificity of 57.2% for the detection of premalignant lesions. We showed the risk of patients over 45 years, with major or minor symptoms, for premalignant and malignant gastric lesions ($p < 0.001$; RR = 3.34; 95%CI: 2.41–4.61). We emphasized the importance of histological evaluation by biopsies of entire gastric mucosa in case of polyps, ulcers, gastric atrophy detection or remnant stomach, for the evaluation of premalignant lesions ($p < 0.,05$). We showed that the prevalence f premalignant lesions increased with age and the presence of Hp infection. We demonstrated the risk of the inflammation in the gastric body for premalignant lesions.
Conclusions: The patient's symptoms were not predictive of endoscopic and histologic findings. Not only symptoms, but also the age, the presence of Helicobacter pylori infection, the histological detection of the extent and location of gastric inflammation and premalignant lesions define the risk for the dyspeptic patients.

Keywords: premalignant lesions, gastric cancer, inflammation, endoscopy, biopsy

Introduction

Gastric cancer remains the second cause of cancer deaths in the world and the improvement in the survival rate depends on the early diagnosis and treatment. Gastric adenocarcinoma is the end stage of a multi-factorial stepwise process. Gastric mucosa undergoes progressive phenotypic changes, from chronic active gastritis to gastric atrophy, intestinal metaplasia and gastric dysplasia, which have been defined as gastric precancerous lesions (1). A precancerous condition is defined as a clinical state associated with a significantly increased risk of cancer. A precancerous lesion represents a morphologically altered tissue in which cancer is more likely to occur than in its apparently normal counterpart (2).

Strategies for prevention gastric neoplasia may differ according to the risk level of population. We studied the incidence rates of premalignant and malignant gastric lesions to identify the risk of different patients according to their age, symptoms, endoscopic and histological findings.

Materials and methods

Patients

One thousand six hundred and fifty-one patients with gastric complaints referred for endoscopic procedure were enrolled. There were 817 men and 834 women. Indications for endoscopic examination included presence of alarm or major symptoms, such as weight loss, vomiting, dysphagia, evidence of gastrointestinal bleeding (hematemesis, melena, anemia) and other gastrointestinal complaints (minor symptoms). Clinical data including demographic characteristics, medical history, medication, indications for endoscopy and findings for all patients undergoing upper endoscopy were prospectively recorded on standard forms and entered into a computer database.

Endoscopic and histological evaluation

All endoscopies included in the analysis were performed by experienced gastroenterologists, and an informed consent for endoscopy was obtained before the procedure. A videoendoscope Olympus Exera was used. Four biopsy specimens were taken, two from the antrum and two from the body of the stomach (the greater and lesser curvature). For the purpose of detection of early gastric cancer and premalignant lesions, we took multiple biopsies from any lesions found at endoscopy. They were fixed in formalin, stained with hematoxilin and eosin, and assessed for Helicobacter pylori, the degree of neutrophil infiltration and intestinal metaplasia, atrophy, dysplasia. The degree of neutrophil infiltration was classified according to four grades (0 no inflammation, 1 mild, 2 moderate, 3 marked) according to the updated Sydney system. Active gastritis was classi-

Table I. Endoscopic findings

Pathology	Gastric polyps	Gastric ulcer	Remnant stomach	Atrophic gastritis	Gastric cancer
Patients N (%)	90 (5.5%)	55 (3.3%)	101 (6.1%)	294 (17.8%)	79 (4.8%)

Table II. Histological diagnosis

Pathology	Intestinal metaplasia	Gastric atrophy	Dysplasia	Gastric adenocarcinoma	Gastric lymphoma	High grade intraepithelial neoplasia
Patients N (%)	655 (43.9%)	305 (20.4%)	55 (3.7%)	76 (5.1%)	3(0.2%)	4 (0.26%)

fied into four categories (no gastritis, antrum-predominant gastritis, corpus-predominant gastritis, pangastritis). Intestinal metaplasia was classified in two grades (absent or present).

Statistical methods

The primary aims of the study were to determine the frequency of premalignant and malignant gastric lesions and to identify predictors of such lesions. All statistical analyses were performed with statistical software EpiInfo. Data were compared by using the chi-square test. The significance of factors associated with gastric lesions was evaluated using 2 × 2 contingency tables and the Fisher exact tests. All p values were two-tailed, with the level of statistical significance specified as 0.05.

Results

A total of 1651 endoscopies were performed between January 2007–December 2008. The indications for endoscopy were major symptoms: hematemesis and/ or melena in 80 patients (4.8%), anemia in 259 (15.7%), dysphagia in 59 (3.6%) vomiting in 370 (22.4%), weight loss in 385 (23.3%) and minor symptoms: abdominal pain in 808 patients (48.9%), discomfort in 500 (30.3%), heartburn and regurgitation in 269 (16.3%), abdominal bloating in 246 (14.9%), loss of appetite in 350 patients (21.2). Patients were grouped according to the age: less than 25 years of age=24 patients (1.5%), between 25 and 35 years = 87 patients (5.3%), 35–45 years = 202 (12.2%), 45–55 years = 320 patients (19.4%), 55–65 years = 428 (25.9%), 65–75 years = 398 (24.1%), over 75 years of age = 192 patients (11.6%).

Endoscopic and histological findings

Endoscopic findings are summarized in Table I. We identified 90 patients (5.5%) with gastric polyps, 294 (17.8%) with atrophic gastritis, 55 (3.3%) with gastric ulcer, 79 (4.8%) with gastric cancer, 101 patients (6.1%) with remnant stomach after gastrectomy. Biopsy specimens were obtained from 1493 patients. We identified gastric inflammation, premalignant and malignant lesions by histological examination (Table II). Intestinal metaplasia was detected in 655 patients (43.9%), gastric atrophy in 305 (20.4%), dysplasia in 55 (3.7%), gastric adenocarcinoma in 76 (5.1%), gastric lymphoma in 3 (0.2%), high grade intraepithelial neoplasia in 4 patients (0.26%).

Association between symptoms and endoscopic/ histological findings

The correlation between symptoms and endoscopic/ histological findings are presented in Table III and IV. Gastric malignancy was associated with major symptoms: weight loss, vomiting, dysphagia, anemia and with abdominal pain and the loss of appetite ($p < 0.001$). We found that major symptoms are strong correlated with the detection of premalignant lesions by biopsies: atrophic gastritis, intestinal metaplasia and dysplasia (Table IV). The loss of appetite was associated with the detection of premalignant and malignant lesions ($p < 0.001$).

We found that individual major symptoms have different sensitivities and specificities for the detection of gastric neoplasia (Table V). The sensitivity and specificity of associated major symptoms for the detection of premalignant and malignant lesions are illustrated in Table VI. Major

Table III. Correlations between symptoms and endoscopic findings (positive correlations are marked with bold)

Symptoms	Gastric ulcer	Polyps	Gastric remnant	Atrophic gastritis	Gastric cancer
Bleeding	**p < 0.0001**	p = 0.10	p = 0.35	p = 0.16	p = 0.25
Vomiting	**p < 0.01**	p = 0.20	p = 0.09	**p = 0.02**	**p < 0.0001**
Weight loss	p = 0.23	p = 0.49	**p = 0.01**	**p < 0.0001**	**p < 0.0001**
Dysphagia		p = 0.15	**p = 0.04**	p = 0.19	**p < 0.0001**
Anemia	**p = 0.05**	**p = 0.01**	p = 0.26	**p < 0.0001**	**p < 0.001**
Pain	**p < 0.0001**	p = 0.09	p = 0.09	**p = 0.037**	**p < 0.0001**
Discomfort	**p < 0.0001**	p = 0.33	p = 0.36	p = 0.13	**p < 0.0001**
Bloating	**p = 0.01**	p = 0.23	**p = 0.01**	p = 0.37	**p < 0.0001**
Loss of appetite	p = 0.21	p = 0.30	p = 0.13	**p < 0.0001**	**p < 0.0001**
Reflux symptoms	p = 0.13	p = 0.22	**p = 0.01**	**p < 0.0001**	**p < 0.01**

Table IV. Correlations between symptoms and histological findings (positive correlations are marked with bold)

Symptoms	Atrophic gastritis	Intestinal metaplasia	Dysplasia	Gastric adenocarcinoma
Bleeding	p = 0.24	p = 0.15	p = 0.11	p = 0.15
Vomiting	p = 0.24	p = 0.20	**p = 0.02**	**p < 0.0001**
Weight loss	**p = 0.001**	**p < 0.001**	**p < 0.01**	**p < 0.0001**
Disphagia	**p < 0.01**	p = 0.24	**p = 0.05**	**p < 0.0001**
Anemia	**p < 0.0001**	**p < 0.01**	**p = 0.02**	**p < 0.001**
Pain	p = 0.13	**p = 0.01**	p = 0.20	**p < 0.001**
Discomfort	**p = 0.04**	**p < 0.001**	p = 0.22	0
Bloating	p = 0.28	p = 0.20	p = 0.01	**p < 0.001**
Loss of appetite	**p < 0.0001**	**p < 0.0001**	**p < 0.001**	**p < 0.0001**
Reflux symptoms	**p < 0.0001**	**p < 0.0001**	p = 0.25	**p = 0.01**

Table V. Sensitivities and specificities of individual major symptoms for the detection of cancer

Major symptoms	Sensitivity % (95% CI)	Specificity % (95% CI)
Bleeding	8.4% (3.5%–16.6%)	95.3% (94.2%–96.3%)
Anemia	31.3% (21.6%–42.4%)	85.1% (83.3%–86.8%)
Vomiting	48.2% (37.1%–59.4%)	79% (76.8%–80.9%)
Weight loss	77.1% (66.6%–85.6%)	79.5% (77.4%–81.5%)
Disphagia	16.9% (9.5%–26.7%)	97.1% (96.1%–97.9%)

symptoms have a sensitivity of 95.2% for the detection of gastric neoplasia, with a specificity of 54.5%, and a sensitivity of 61.6% with a specificity of 57.2% for the detection of pre-neoplasic lesions.

We correlated the age and the symptoms to identify the risk of patients for premalignant and malignant pathology. We found that the risk of malignancy and premalignant lesions increased with age. Patients less than 45 years of age had a low risk for premalignant and malignant lesions, in the presence ($p = 0.27$; RR = 0.81; 95%CI: 0.42–1.54) or absence of major symptoms ($p < 0.001$; RR = 0.36; 95%CI: 0.20–0.65). Patients over 45 years of age had an increased risk of malignancy and premalignant lesions, in case of minor symptoms ($p = 0.01$; RR = 1.53; 95%CI: 1.06–2.21) and especially in case of major symptoms ($p < 0.001$; RR = 3.34; 95%CI: 2.41–4.61). Malignancy was strongly associated with male sex (RR = 2.45; 95%CI: 1.51–3.99).

Correlation between endoscopic and histological findings

We studied the association between endoscopic findings: gastric polyps, gastric and duodenal ulcers, gastric cancers, remnant stomach, atrophic gastritis and the detection of premalignant and malignant lesions by biopsies. We found an association between the detection of gastric polyps or gastric ulcers and the presence of gastric atrophy ($p = 0.04$ in case of polyps; $p = 0.03$ in case of ulcer) and intestinal metaplasia ($p = 0.01$ for polyps and $p < 0.001$ for gastric ulcer). We found a positive correlation between remnant stomach, atrophic gastritis ($p = 0.04$) and intestinal metaplasia ($p = 0.01$) and also between gastric atrophy at endoscopic examination and the histological evidence of intestinal metaplasia ($p < 0.001$) and dysplasia ($p = 0.02$). The detection of a gastric ulcer with irregular margins, irregular base, nodular surrounding mucosa (malignant-appearing) was associated with the detection of atrophic gastritis ($p = 0.05$), intestinal metaplasia ($p = 0.006$), dysplasia ($p = 0.01$) and adenocarcinoma ($p < 0.05$).

Table VI. Sensitivity and specificity of associated major symptoms

Lesions	Major symptoms	
	Sensitivity % (95% CI)	Specificity % (95% CI)
Gastric cancer	95.2% (88.1%–98.7%)	54.5% (52%–57.0%)
Premalignant lesions	61.6% (57% – 66.1%)	57.2% (54.4%–60.1%)

We demonstrated a strong correlation between the detection of gastric ulcer by endoscopy and the diagnosis of corpus-predominant gastric inflammation by biopsies ($p = 0.04$; RR = 2.70; 95%CI: 1.02–7.15) and pangastritis ($p < 0.001$; RR = 9.54; 95%CI: 5.34–17.05); the detection of duodenal ulcer was associated with histological evidence of antrum-predominant gastritis ($p < 0.001$; RR = 15.59; 95%CI: 8.99–27.02). We found a positive association between gastric ulcer and atrophic gastritis ($p = 0.03$; RR = 1.74; 95%CI: 1.22–3.04), with a negative association between duodenal ulcer and gastric atrophy ($p < 0.01$; RR = 0.30; 95%CI: 0.15–0.62).

The identification of corpus-predominant gastritis was strong correlated with the risk of intestinal metaplasia (RR = 1.71; 95%CI: 1.41–2.07) atrophic gastritis (RR = 2.03; 95%CI: 1.39–2.98) and dysplasia (RR = 3.45; 95%CI: 1.45–8.21); pangastritis was also associated with the risk of premalignant lesions: intestinal metaplasia (RR = 2.28; 95%CI: 2.06–2.51), atrophic gastritis (RR = 2.35; 95%CI: 1.93–2.85) and dysplasia (RR = 2.04; 95%CI:1.19–3.49).

Prevalence of premalignant lesions

The frequencies of premalignant lesions were stratified by age and the presence or the absence of H pylori infection. We showed that the prevalence of atrophic gastritis and intestinal metaplasia was higher in older and infected patients than in non-infected and younger patients (Table VII and VIII). In patients over 75 years, the extent of atrophic gastritis and intestinal metaplasia was followed by the loss of H pylori infection.

We studied the presence of H pylori infection in patients of different ages (Fig. 1), and also the diagnosis of gastric cancer (Fig. 2) and premalignant lesions (Fig. 3). We found the presence of H pylori infection in all groups of patients. The diagnosis of gastric cancer started before the age of 35 years.

Table VII. Prevalence of atrophic gastritis

Age	Positive Hp infection N; (%) (95%CI)	Negative Hp infection N (%) (95%CI)
< 25 years	0; 0% (0–20.6%)	0; 0% (0–45.9%)
25–35 years	1; 2.1% (0.1–11.3%)	0; 0% (0–13.7%)
35–45 years	9; 6.9% (3.2 – 12.7%)	2; 4.3% (0.5–14.5%)
45–55 years	18; 9.1% (5.5–14%)	2; 3.5% (0.7–9.9%)
55–65 years	54; 20% (15.4–25.3%)	5; 4.5% (1.5–10.1%)
65–75 years	98; 38.1% (32.2 – 44.4%)	21; 18.4% (11.8–26.8%)
> 75 years	54; 54.5% (44.2– 64.6%)	30; 45.5% (33.1– 58.2%)

Table VIII. Prevalence of intestinal metaplasia

Age	Positive HP infection N; (%) (95%CI)	Negative HP infection N (%) (95%CI)
< 25 years	0; 0%(0–20.6%)	0; 0% (0–45.9%)
25–35 years	3; 6.4% (1.3–17.5%)	1; 4% (0.1–20.4%)
35–45 years	39; 30% (22.3–38.7%)	6; 12.8% (4.8–25.7%)
45–55 years	85; 42.9% (35.9–50.1%)	17; 19.8% (12–29.8%)
55–65 years	146; 54.1% (47.9–60.1%)	30; 26.8 % (18.9–36%)
65–75 years	165; 64.2% (58–70.1%)	44; 38.6% (29.6–48.2%)
> 75 years	68; 68.7% (58.6–77.6%)	36; 54.5% (41.8–66.9%)

Table IX. The detection of active pangastritis at different ages

Ages	Frequency	Percent	Cum Percent
25–35	4	1.2%	1.2%
35–45	43	12.6%	13.8%
45–55	55	16.2%	30.0%
55–65	85	25.0%	55.0%
65–75	107	31.5%	86.5%
>75	46	13.5%	100.0%
Total	340	100.0%	100.0%

Table X. The detection of corpus-predominant gastritis at different ages

Ages	Frequency	Percent	Cum Percent
25–35	0	0.0%	0.0%
35–45	3	7.1%	7.1%
45–55	9	21.4%	28.6%
55–65	13	31.0%	59.5%
65–75	15	35.7%	95.2%
> 75	2	4.8%	100.0%
Total	42	100.0%	100.0%

Topographical patterns of premalignant lesions

We studied the extent of gastric inflammation, gastric atrophy and intestinal metaplasia from antrum to the body of the stomach, to identify the risk of the patients for gastric neoplasia. We identified by biopsies the extent and location of premalignant lesions, in patients with different ages. We showed an extent of gastric inflammation, atrophy and intestinal metaplasia in the body at the stomach at older ages. Patients over 65 years presented corpus-predominant gastritis and pangastritis (Tables IX and X). Patients over 75 years had predominant atrophic pangastritis (Table XI). The prevalence of atrophic gastritis and intestinal metaplasia in antrum and corpus biopsies increased with age (Tables XI and XII).

Discussion

The primary aims of this analysis were to determine the prevalence of premalignant and malignant lesions and the characteristics of patients who have this pathology, to determine which patients referred for endoscopy are at risk.

Patients with alarm or major symptoms were much more likely to have an important endoscopic finding. We showed that alarm symptoms were associated with the detection of malignancy and premalignant lesions. In patients over 45 years of age, with complicated (major symptoms) or uncomplicated dyspepsia, we must to perform endoscopic and histological evaluation, to identify premalignant and malignant gastric lesions and the risk of the patients. The results of this analysis support European recommendations. The Maastricht Consensus guidelines recommended that patients over the age of 45 who have severe dyspeptic symptoms or those with alarm symptoms should be referred for endoscopy (3). In United States, this age threshold was 55 years (4).

The value of alarm symptoms in younger patients is controversial. Vakil N et al. showed that alarm symptoms have limited predictive value for an underlying malignancy (5). The sensitivity of alarm symptoms varied from 0% to 83% and the specificity also varied significantly from 40% to 98% between studies. We found a sensitivity of individual major symptoms for the prediction of malignancy between 8.4% and 77.1%, with a specificity varying from 79% to 97.1%.The combination between alarm symptoms had a sensitivity of 61.6% for the detection of premalignant lesions and a specificity of 57.2%. Alarm symptoms do not perform well as diagnostic indicators of an underlying malignancy or premalignant lesion in patients with dyspepsia, as it has been showed in our study and in previous studies

Substantial information on the prevalence and the patterns of premalignant gastric lesions can be gathered by histological evaluation of an appropriately representative range of biopsy samples taken from the visible mucosal changes and from the unaffected gastric mucosa. The value of examining the mucosa that surrounds a gastric polyp, ulcer or lesion consists in the detection of some microscopic changes. We demonstrated strong correlation between these endoscopic findings and the histological evidence of intestinal metaplasia, atrophic gastritis and dysplasia ($p < 0.05$).

Carmack SW et al. showed that adenomatous polyps larger than 2 cm contain foci of adenocarcinoma. These polyps arise most often in patients with chronic, atrophic, metaplasic gastritis and they share a common epidemiological pattern (6). We found a positive relation between the presence of gastric polyps and the detection of premalignant lesions: intestinal metaplasia ($p = 0.01$), atrophic gastritis ($p = 0.04$) and dysplasia ($p < 0.05$). We demonstrated the risk of larger polyps (bigger than 10 mm) for dysplasia: RR = 5.06; 95%CI: 3.07–8.31 and adenocarcinoma: RR = 11.00; 95%CI: 2.04–59.28.

Table XI. The detection of atrophic pangastritis at different ages

Ages	Frequency	Percent	Cum Percent
25–35	0	0.0%	0.0%
35–45	3	3.4%	3.4%
45–55	4	4.6%	8.0%
55–65	13	14.9%	23.0%
65–75	31	35.6%	58.6%
> 75	36	41.4%	100.0%
Total	87	100.0%	100.0%

Table XII. The detection of extensive intestinal metaplasia (antrum and corpus) at different ages

Ages	Frequency	Percent	Cum Percent
25–35	0	0.0%	0.0%
35–45	9	4.7%	4.7%
45–55	28	14.5%	19.2%
55–65	41	21.2%	40.4%
65–75	71	36.8%	77.2%
> 75	44	22.8%	100.0%
Total	193	100.0%	100.0%

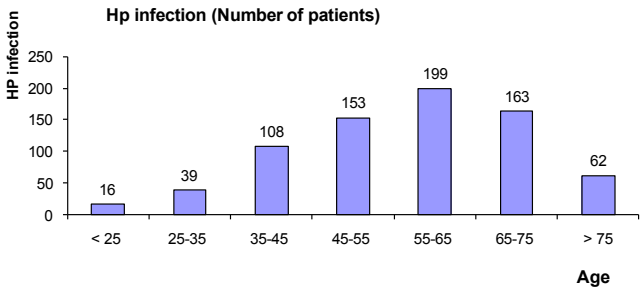

Fig. 1. The number of patients with H pylori infection at different ages

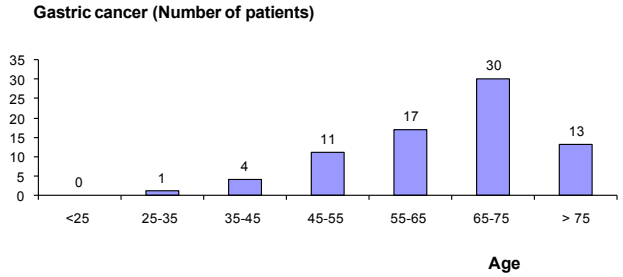

Fig. 2. The detection of gastric cancer at different ages

We showed that the prevalence of premalignant lesions increased with age and the presence of H pylori infection. With the increasing of the age of the patient, gastric atrophy and intestinal metaplasia extended from antrum to the body of the stomach. We detected corpus-predominant gastritis and pangastritis over the age of 65, and also atrophic pangastritis and extensive intestinal metaplasia at the older ages. With advanced age (>75 years), the rate of H pylori infection decreased, and premalignant lesions advanced (atrophic pangastritis), indicating that these patients had atrophy severe enough to lead to the natural disappearance of H pylori (modified mucosa was not a favorable niche for H pylori colonization).

The persistent H pylori infection on the different ages shows the necessity to test and treat the bacteria, before the extension of premalignant changes in gastric mucosa. Previous authors showed that the risk of gastric cancer correlates with gastric atrophy (7). H pylori eradication therapy should therefore be administered early and targered to prevent the progression to atrophy (8).

The Operative Link on Gastritis Assessment (OLGA) group proposed a standardized staging system to estimate the risk of gastric malignancy, based on the topography and severity of gastritis (9). We identified the patients with extensive premalignant lesions; in this group, surveillance endoscopy is potentially useful for early detection of cancer.

We demonstrated the correlation between corpus-predominant gastritis, pangastritis and premalignant lesions. Previous studies have shown that individuals with chronic gastritis in the corpus, including pangastritis, have a higher risk for gastric cancer than those with antrum-predominant gastritis and those with intestinal metaplasia have an even higher risk (10). These results confirm the hypothesis of Correa that severe atrophic gastritis accompanying intestinal metaplasia caused by persistent H pylori infection is closely related to the development of intestinal-type gastric cancer (11).

A comparative topographic analysis of a grade and activity of Hp gastritis in the antrum and corpus with a search for corpus-dominant gastritis is probably a better risk marker for carcinogenesis than the search for intestinal metaplasia, witch is subject to sampling error (12). These data showed the importance of evaluation of gastric inflammation and its control, from the perspective of cancer prevention or screening (13).

Duodenal ulcer and gastric cancer are considered to be two ends of the clinical spectrum of Hp infection. While duodenal ulcer is characterized by antral predominant gastritis and acid hypersecretion, gastric cancer is characterized by corpus predominant or pangastritis with acid hyposecretion (14). Uemura et al. found that the risk of gastric cancer development was lower in patients with duodenal ulcers, but higher in patients with gastric ulcers and non-ulcer dyspepsia (15). The different rates of progression of gastritis suggest that genetic and environmental factors are involved in this divergent clinical outcome of H pylori infection. We found the association between duodenal ulcers and antral gastritis, with non-atrophic pattern and the positive correlation between gastric ulcers, corpus inflammation and the atrophic pattern. These last patients may warrant endoscopic surveillance to detect early premalignant and malignant changes. The same relationship between gastric cancer, gastric ulcer and the atrophic pattern was emphasized by other authors (16).

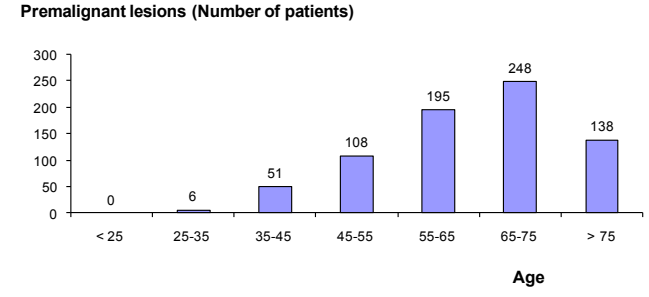

Fig. 3. The detection of premalignant lesions at different ages

Conclusions

Carcinomas of the stomach are thought to arise from a series of changes within the gastric mucosa: premalignant lesions. For the assessment of the extent of atrophic gastritis and intestinal metaplasia and for the diagnosis of gastric inflammation and H pylori infection, endoscopic observation and biopsy from localized lesions such as cancer, ulcer, polyp, and from surrounding mucosa, are essential. The patient's symptoms were not predictive of endoscopic and histologic detection of neoplasia and premalignant changes in gastric mucosa. Not only symptoms, but also the age, the presence of H pylori infection, the histological detection of the extent and location of premalignant gastric changes define the risk for the dyspeptic patients. The eval-

uation of the extent of gastric inflammation, metaplastic and atrophic gastritis is necessary in older patients, as we demonstrated the tendence of progression of these lesions with age. Eradication of H pylori should be considered in all age groups to prevent the progression of premalignant lesions.

The method of conventional endoscopy with biopsies for the detection of premalignant gastric lesions is laborious. Many biopsy specimens are required from detected lesions and from mapping the distribution and extent of intestinal metaplasia and gastric atrophy. For the evaluation of the extent of premalignant lesions, several alternative and supplementary strategies have been developed: serological biomarkers (like serum pepsinogen level), new endoscopic techniques (magnifying endoscopy, narrow band imaging).

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