

THE ENDOSCOPIC DIAGNOSIS OF PREMALIGNANT GASTRIC LESIONS

abstract

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Introduction

Gastric cancer represents one of the most serious causes of mortality in the world. The occurrence of gastric cancer is marked, especially in the developed countries, by an important decline in the second half of the twentieth century. In our country, the rate of gastric cancer is of 2,9 in 100000 inhabitants, with relatively high variations at national level, according to a study elaborated in 2003 which included 11 university centers.

At present, more attention is paid to the secondary prevention of gastric cancer at european level, under the form of the Guide To Managing Premalignant Gastric Lesions, issued in Porto, Portugal, in 2011, under the care of The European Society of Digestive Endoscopy, of The European Study Group of Helicobacter Pylori and of the Portuguese Society of Digestive Endoscopy.

At national level, any approach should take into consideration results of studies directed on subjects such as: the presence of Helicobacter Pylori infection, the existence of premalignant gastric lesions grouped on age, sex, environmental factors, the detection of these lesions by digestive endoscopy (conventional, magnification chromoendoscopy, narrow band imaging), the presence of aggravating factors, the existence of groups of populations more likely to develop such a condition, the best observation methodology.

Objectives, study material and method

The main objectives of this study are: the evaluation of a group of patients who display one or more premalignant lesions (gastric atrophy, intestinal metaplasia, gastric dysplasia), investigated by upper digestive endoscopy , and the description of this group from a demographic point of view, their endoscopic information, symptomatology, the presence or absence of risk factors, etc. The second study is centered on the description of a group of patients who were investigated by means of conventional endoscopy, as well as by magnification chromoendoscopy, and it observes the contribution of this type of investigation in the diagnose process of premalignant lesions. The third study follows another group of patients investigated by means of conventional endoscopy and narrow band imaging, also observing its contribution to the diagnose of gastric atrophy, intestinal metaplasia and gastric dysplasia.

Results

The preneoplastic malignant lesions occur more frequently with men (53,16%) and with patients of urban environment (70%). The patients with hereditary gastric cancer records show a higher risk of developing

preneoplastic lesions: intestinal metaplasia ($p<0,0001$, OR: 16,30, RR: 1,42, IC: 1,29-1,57), atrophy with metaplasia ($p=0,0035$, OR: 10,37, RR: 1,26, IC: 1,15-1,37), premalignant lesions ($p=0,035$, OR: 9,740, RR: 1,243, IC: 1,14-1,35).

The presence of intestinal metaplasia at corpus level per primam or by extension from the antral level increases the risk of progress towards dysplasia, as opposed to the antral area, by an amplification rate of 2,93 ($p=0,0003$, RR: 2,93, IC: 2,5-3,41).

Gastric cancer stands in an inducing, direct relation to dysplasia ($p<0,0001$, RR: 17,7, IC: 6,15-51,14), to incomplete intestinal metaplasia ($p=0,01$, OR: 9,30, RR: 1,59, IC: 1,33-1,89), but not to complete intestinal metaplasia ($p=0,90$, OR: 2,05, RR: 1,04, IC: 0,5-2,15).

Smoking is a risk factor in the development of preneoplastic lesions: intestinal metaplasia ($p=0,0035$, OR=3,26, RR=1,20, IC: 1,80-1,33) with preneoplastic lesions ($p=0,0017$, OR: 3,69, RR: 1,2, IC: 1,09-1,32).

Helicobacter Pylori is a risk factor for the development of preneoplastic lesions: intestinal metaplasia ($p=0,0123$, OR=3,8, RR=1,2, IC: 1,23-1,70), incomplete intestinal metaplasia ($p=0,0032$, OR: 2,05, RR: 1,31, IC: 1,10-1,56), gastric atrophy ($p=0,0068$, OR=1,1, RR=1,06).

Enteral reflux is a risk factor for the development of preneoplastic lesions ($p=0,0017$, OR: 3,69, RR: 1,20, IC: 1,09-1,32), intestinal metaplasia ($p=0,0293$, OR=4,43, RR=1,20, IC: 1,08-1,34).

For the conventional endoscopy associated with sampling of random biopsies we have obtained a sensitivity in the detection of gastric atrophy at antrum and corpus level of 74,19% and 66,66%; in the detection of intestinal metaplasia at antrum level and corpus level of 72%, respectively 60%, and in the detection of gastric dysplasia 58,82%.

For gastric atrophy at antrum and corpus levels, we have obtained by means of magnification chromoendoscopy a sensitivity in reading of 83,87%, respectively of 77%, and a specificity of 78,94%, respectively of 79%; for intestinal metaplasia at antrum and corpus level a sensitivity of 88%, respectively 80%, and a specificity of 76%, respectively 78%; for dysplasia a sensitivity of 88% and a specificity of 77%.

The sensitivity and specificity of narrow band endoscopy are: for gastric atrophy of 90%, respectively of 88%, and for intestinal metaplasia of 89%, respectively of 93%.

Conclusions

The patients who should be included in an observation program are those with hereditary gastric neoplasm records, and those who display extension to the corpus level of the preneoplastic lesions.

Patients who display only complete intestinal metaplasia do not need observation.

Smoking, bile acids, Helicobacter Pylori represent risk factors in the development of preneoplastic gastric lesions.

Conventional endoscopy cannot detect accurately enough gastric atrophy or intestinal metaplasia, neither by direct observation, nor after the random biopsy sampling. Magnification chromoendoscopy and narrow band imaging supply superior sensitivity and specificity than the conventional endoscopy.