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Atrophic Body Gastritis: A Challenge for the Presumptive Endoscopic and Histologic Diagnosis of Autoimmune Gastritis

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1. Introduction

The diagnosis of glandular atrophy of the gastric mucosa (gastric atrophy) remains a challenge in the areas of gastrointestinal endoscopy and pathology. The importance of an endoscopic suspicion of this regressive tissue change is the possibility of alerting the pathologist to its presence. Patients with gastric atrophy, especially in more advanced stages, are more prone to develop intestinal metaplasia, dysplasia and gastric carcinoma, known as the Correa cascade (Correa, 1984, 1992). The most common type of gastric atrophy is that associated with *Helicobacter pylori* (*H. pylori*) infection. In such cases, the glandular atrophy usually occurs in parallel to the course of the inflammatory process that takes place in the gastric antrum (antral gastritis, multifocal gastritis) or in both gastric antrum and body (pangastritis, multifocal gastritis). In contrast, in most cases the chronic atrophic gastritis selective of the gastric body and sparing the antral mucosa is a consequence of an autoimmune inflammatory process. For the purpose of this chapter, the chronic gastritis presenting this histopathological pattern will be called atrophic body gastritis (ABG). Exceptionally, some patients with *H. pylori*-associated gastritis develop gastric lesions with histological pattern very similar to that of ABG which can lead to uncertainty about the differential histologic diagnosis with chronic gastritis of autoimmune origin.

Therefore, the two most important inflammatory diseases of the gastric mucosa, multifocal chronic gastritis and autoimmune gastritis, tend to progress to glandular atrophy of the gastric mucosa. In the first case, gastric atrophy may not occur or it develops more slowly, becoming conspicuous usually in later stages of life. In the second case, which is also more frequent with advancing age, gastric atrophy progresses more rapidly and may induce severe gastric changes also in the younger age groups. This differential course of the development of gastric atrophy in these two inflammatory diseases of the stomach involves different clinical and pathophysiological consequences. Those associated with *H. pylori* infection usually do not involve important pathophysiological changes, while the atrophy resulting from autoimmune disease often leads to well-known pathophysiological changes of the gastric mucosa, often culminating in pernicious anemia. Furthermore, the *H. pylori*-dependent glandular atrophy is considered to be a condition predisposing to gastric adenocarcinoma, which has high rates of morbidity and mortality, while the autoimmune-

dependent glandular atrophy is considered to be a condition predisposing to gastric carcinoids, which, unlike the adenocarcinomas, have low rates of morbidity and mortality. Due to the different regional involvement of the stomach by these two types of chronic gastritis, their histological recognition is relatively easy when tissue samples are properly collected and processed. However, this is not always the case.

Thus, the importance of the differential diagnosis of ABG within this group of chronic gastritis resides in the possibility of its autoimmune origin and in its major clinical consequence, i.e., pernicious anemia. Pernicious anemia and autoimmune gastritis progress in an insidious manner and are usually diagnosed when they have reached florid clinical and morphological characteristics after years of evolution. However, a number of patients with dyspeptic complaints and patients with ABG and pernicious anemia undergo endoscopy in tertiary care settings without a prior knowledge of major illness. (Carmel, R., 1996) Therefore, for some patients seeking a first endoscopy service, the collection of biopsies from the gastric mucosa may be the first opportunity to establish the initial diagnosis of ABG, with or without pernicious anemia. This is important because the evolution of subclinical gastric lesions and systemic symptoms may result in undesirable consequences that become established in a gradual manner. These consequences mainly result from achlorhydria, vitamin B12 deficiency, and the development of hyperplastic polyps and neuroendocrine tumors of the gastric mucosa. For this reason, the diagnosis of ABG acquires importance since it establishes an appropriate clinical and endoscopic follow-up of the patient.

The objective of this article is to present some data on this topic obtained from a tertiary care unit of gastrointestinal endoscopy and to discuss some pitfalls linked to the routine histopathologic diagnosis of atrophic body gastritis.

2. Methods and results

For the purpose of evaluating the outcomes of endoscopic examination of patients with a final histopathological diagnosis of ABG we surveyed all cases of upper gastrointestinal endoscopy with biopsy sampling of gastric antrum and gastric body performed from 2007 to 2009 at a referral center for digestive endoscopy in the city of Belo Horizonte, Brazil.

A total of 6,005 consecutive gastroesophageal endoscopies with gastric biopsies of the antral and body mucosa of the stomach were reviewed. Among these cases 2,564 (42.7%) had the diagnosis of chronic gastritis as the main pathological condition of the gastric mucosa. Of these, 141 (5.5%) had a diagnosis of atrophic body gastritis (type A gastritis) suggestive of an autoimmune nature. However, a conclusive diagnosis could not be made in the remaining 230 patients (9.0%) whose histology report was mainly descriptive, stating the presence of "chronic gastritis of the body with areas of atrophy" or "body-predominant chronic atrophic gastritis ". Therefore, in most cases of chronic gastritis with body mucosa atrophy it seems that the pathologists did not find sufficient morphological evidence for a more conclusive diagnosis.

All histological slides of the 141 cases of atrophic body gastritis as well as those of the 230 patients with inconclusive diagnosis of gastritis were re-examined by an expert gastrointestinal pathologist (AJAB). When necessary the corresponding paraffin blocks were recovered for new histological sections. After reviewing all cases, the previous diagnosis of the 141 patients with atrophic body gastritis was confirmed. Among the 230 patients with an inconclusive diagnosis, 55 (24%) could be confirmed as cases of atrophic body gastritis

(Table 1). The 196 patients with a final histologic diagnosis of ABG ranged in age from 11 to 94 years, with a significant predominance of females (76.0%) over males (24.0%). Fifty patients were in a relatively young age range (31 to 50 years), with an even more significant predominance of females, i. e., 83.3% *vs* 16.7% (Figure 1) .The remaining 175 patients were excluded from the study, either because the biopsy specimens of the antral or oxyntic mucosa were not fairly representative for histology or because they could be considered as cases of multifocal atrophic gastritis, regardless of the presence of *H. pylori* infection. Finally, the histological findings of the 196 patients with a diagnosis of atrophic body gastritis were correlated with the endoscopic reports (Table 2).

	Total of cases	%
All cases of chronic gastritis	2.564	100
Atrophic body gastritis	141	5,5
Unspecified atrophic gastritis	230	9,0
ABG after review of unspecified atrophic gastritis	55	2,2
Total of ABG diagnosed	196	7,6

Table 1. Main histological diagnosis of 6,005 gastroesophageal endoscopies carried out from 2007-2009 in a tertiary care unit of gastrointestinal endoscopy, Belo Horizonte, Brazil

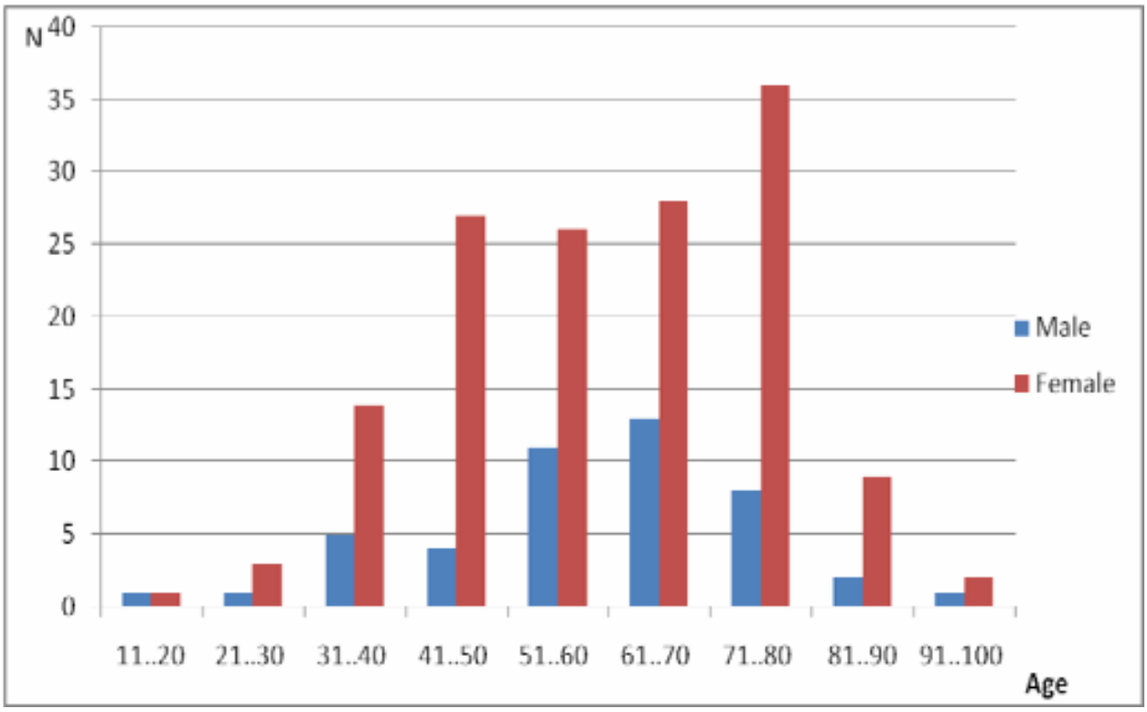

Fig. 1. Distribution of the 196 patients with the final diagnosis of ABG according to the age and gender

Among the 2,564 patients with chronic gastritis, 196 (7.6%) had a conclusive histologic diagnosis of atrophic body gastritis (type A chronic gastritis). Gastric mucosa samples of almost all of these patients showed well preserved antral mucosa, and severe glandular

atrophy of the body mucosa (Figure 2 A, C, D). This atrophy was mainly represented by partial or complete replacement of oxyntopeptic glands with glands with intestinal metaplasia and antral-type mucous glands known as pseudoantral or pseudopyloric metaplasia (Figs. 2 D and 3). In several small fragments with poor representation of gastric mucosa, the pseudoantral metaplasia seemed to be a factor responsible for the failure to distinguish between antral and body atrophic mucosa. Small biopsy fragments of atrophic

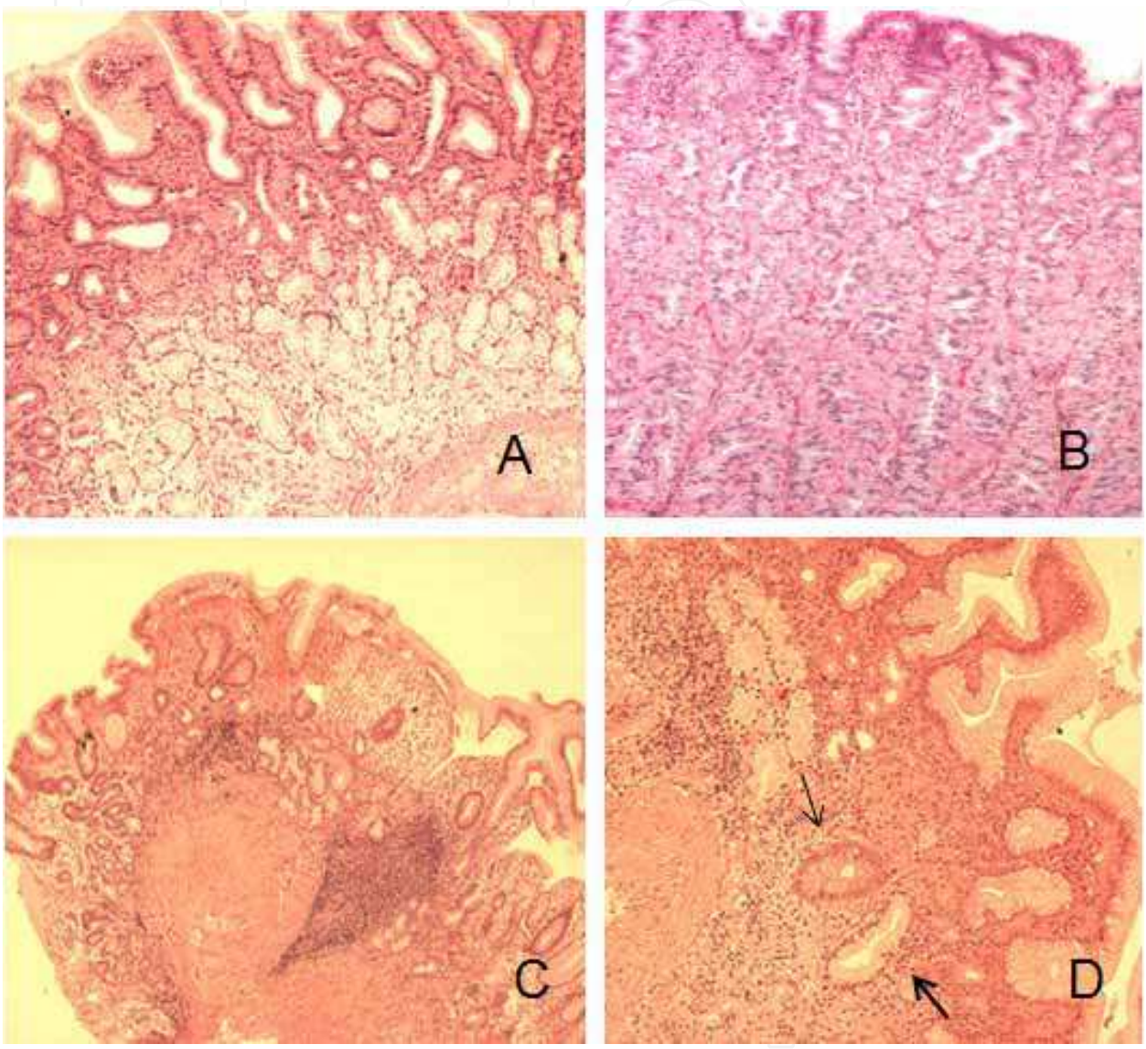

Fig. 2. Normal gastric mucosa of the antrum (A) and body (B) regions. Note that the foveolar region of the gastric antrum occupies approximately half the mucosa thickness while the mucus-secreting glands are distributed in the basal half of the mucosa. The connective tissue of the lamina propria is visible between the glands. Differently, the body mucosa (B) have short foveolas, and the oxintopeptic glands are numerous and justaposed occupy almost the entire space of conjunctive masking the lamina propria. C and D: body mucosa with severe glandular atrophy from a patients with ABG. Presence of glands with intestinal metaplasia (thin arrow) and pseudoantral metaplasia (thick arrow). The antral mucosa seen in A is from the same patient with ABG seen in C and D

body mucosa, or fragments that were cut tangentially, could present a morphological pattern similar to that of pyloric mucosa with chronic inflammation, thus leading to a diagnosis of multifocal gastritis instead of ABG (Figure 3). The boundaries between these two histological types of chronic gastritis become even more critical when *H. pylori* is present, which is an unusual occurrence. In the present series of ABG, *H. pylori* was found in only 9 (4.6%) cases.

The main endoscopic diagnosis of the 196 cases of atrophic body gastritis were as follows: (a) 76 (38.8%) cases of atrophic pangastritis, (b) 63 (32.1%) cases of pangastritis with apparent atrophy of the gastric body mucosa, (c) 42 (21.4%) cases of light enanthematous pangastritis, (d) 10 (5.1%) cases of pangastritis with gastric polyps, (e) 2 (1.0%) cases with endoscopically normal gastric mucosa, and 2 (1.0%) endoscopically unspecified cases (Table 2).

Main endoscopic reports	N	%
Atrophic pangastritis	76	38,8
Enantematous pangastritis/ likely body mucosa atrophy	63	32,1
Light enantematous pangastritis	21	10,7
Antropyloric-predominant enantematous pangastritis	21	10,7
Body-predominant enentematous pangastritis	01	0,51
Gastric polyps	10	5,10
Normal gastric mucosa	02	1,02
Unspecified	02	1,02

Table 2. Main endoscopic report conclusions of the 196 patients with histologic ABG

3. Discussion

For many dyspeptic patients the diagnosis of ABG is the first step indicating the presence of autoimmune gastritis, associated or not with pernicious anemia. Since some of these patients should be monitored periodically from a clinical point of view and regarding the changes in their gastric mucosa, care should be taken not to overlook cases of gastritis with predominant or selective atrophy of the gastric fundus and body on the occasion of the first medical procedures they undergo. Knowing that the final diagnosis of this pathological entity usually depends on a close interaction between endoscopist and pathologist, an indication of the presence of these endoscopic changes to the pathologist directs more attention to the correct diagnosis. This correlation becomes even more necessary if we consider that the histologic processing artifacts, the absence of oxintopeptic glands in the histological sections plus the presence of extensive areas of intestinal and pseudoantral metaplasia, may complicate at first sight the differential diagnosis between ABG and atrophic multifocal gastritis.

Table 2 shows that standard endoscopic examination detected signs of gastric mucosa atrophy in 139 (70.9%) patients with ABG, although the affected gastric region was not specified. In the other 57 (29.1%) patients this change was not found although severe glandular atrophy of the body was found at histology. A more precise endoscopic report could direct the pathologist to a more conclusive histologic diagnosis. Most of the 55 patients confirmed as ABG cases among the 230 patients with varying degrees of mucosal atrophy at histology had an inaccurate endoscopy report.

3.1 Gastric mucosal atrophy: A problematic area of histopathology

The histologic diagnosis of gastric mucosa atrophy remains a difficult area to handle. In addition to the subjectivity of the histologic diagnosis, other factors contribute to blurring the histological interpretation, such as: (*a*) representativeness of the limited endoscopic biopsy. This bias occurs more often in cases in which a small number of biopsies are obtained for this purpose. It should be remembered here that both major types of chronic gastritis and glandular atrophy of the gastric mucosa occur with multifocal distribution. Therefore, the number of gastric biopsies, while important, need not exceed 2 to 4 fragments per region of the stomach, except for special conditions. Consensus among pathologists and endoscopists advocates, in routine cases, two biopsies from the antral region, two from the body and one from the *incisura angularis* (angular notch) (Dixon et al, 1999); (*b*) important for a more accurate diagnosis of glandular atrophy of the gastric mucosa is the good representativeness of the tissue sections. Slices containing the entire thickness of the mucosa, from the more superficial epithelial lining to the *muscularis mucosa*, favor a more reliable histopathological analysis for the interpretation of the presence of glandular atrophy (Fig. 2 A, C, D); (*c*) endoscopic biopsies showing histologic processing distortions arising from inappropriate tangential sections of the mucosa limit the observation of tissue components; (*d*) prominent inflammatory changes and exuberant lymphoid follicles can cause distortion or separation of glands, giving false impression of glandular rarefaction; (*e*) endoscopic biopsies from only one region of the stomach, for example, only from the antral region, leave the mucosa of the body without proper histological analysis. In these cases, selective glandular atrophy of the mucosa of the body may be missed in cases where the body mucosa presents a false endoscopic appearance of normality.

Although highly subjective and dependent on many factors inherent in sampling, the diagnosis and grading of glandular atrophy of the gastric mucosa rests on the final decision of the pathologist. Moreover, in cases of severe atrophy histopathological diagnosis consistently achieves high sensitivity and reproducibility when the endoscopic and histotechnical procedures prior to histological analysis run with relative normality.

It should be remembered that the oxyntic mucosa usually presents a much higher glandular density than that of the antral mucosa. At first, connective tissue and small vessels of the *lamina propria* are not so evident because they are masked by the large number of juxtaposed oxintopeptic glands (Fig. 2 B). This fact becomes more relevant because of tissue shrinkage caused by fixatives commonly used, such as formaldehyde. Tissue fixatives retract the mucosal structures, especially the loose connective tissue of the *lamina propria*. Thus, when examining a histological section of normal oxyntic mucosa the first impression one gets is that it appears to be constituted by almost only glandular epithelial tissue.

Therefore, the loss of oxyntic glands and their replacement with connective tissue tends to be clearly identified by histology. In these cases the degree of subjectivity for the diagnosis of atrophy can be considered small. In contrast, the antral mucosa usually presents less dense glandular tissue and the *lamina propria* is more apparent with a consequent more problematic histologic interpretation of moderate degrees of atrophy in this region (Fig. 2 A).

The presence of areas of intestinal metaplasia that occur with relative frequency in the antral and body mucosa in *H. pylori* chronic gastritis is an objective histological sign indicating the replacement of specialized glands of the gastric mucosa with glands of the intestinal type. This fact strengthens the diagnosis of evolving or established glandular atrophy. What the presence of intestinal metaplasia may represent regarding the existence and grading of gastric atrophy is still a debatable issue (Genta, 1996, 1997). Whether in some cases of

chronic gastritis the presence of few foci of intestinal metaplasia may correspond to the existence of established glandular atrophy is still an unresolved question. However, the presence of these foci in the gastric mucosa should be considered at least a signal of ongoing glandular loss, since in most cases intestinal metaplasia is associated with patent gastric atrophy. Some authors recommend that the histological diagnosis of moderate or severe atrophy of the gastric mucosa should be limited to cases of a glandular loss of at least 50%, replaced or not with metaplastic glands (Dixon et al, 1996).

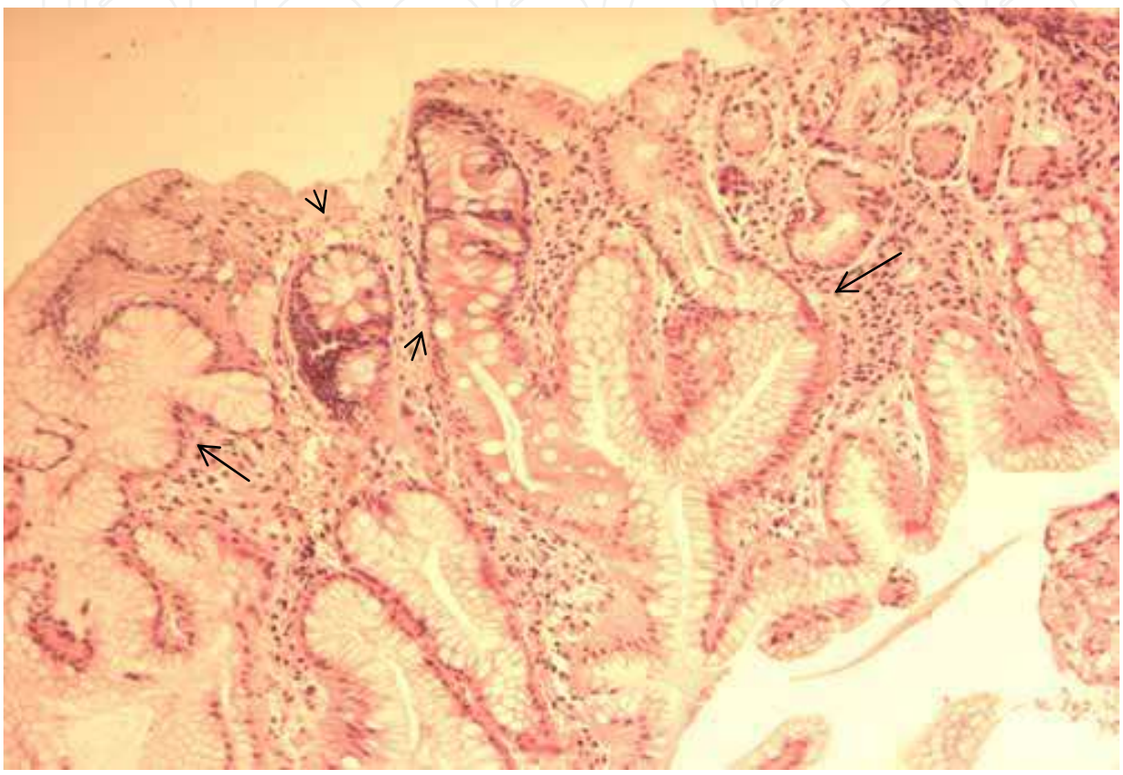

Fig. 3. Histological aspect of the body mucosa of a patient with ABG. Extensive areas of pseudoantral metaplasia (long arrows) and glands exhibiting intestinal metaplasia (short arrows). Oxyntopeptic glands are not observed. The picture may simulate atrophic antral mucosa and the diagnosis of multifocal gastritis, especially under conditions of limited interaction between endoscopists and pathologists and of inappropriate tissue processing

3.2 ABG: Waiting for a pathological definition

The designation of ABG has been extensively used without a specific definition of its limits. It is possible that the roots of this problem reside in the conceptual vagueness of what is called "atrophic gastritis" or even "gastric atrophy" (Genta, 1997). The criterion of ABG adopted for the present study involves only advanced cases of oxintopeptic mucosal atrophy with the antral mucosa showing no relevant histological changes. From a histological point of view, these patterns overlap those described for gastritis of autoimmune etiology. This definition of ABG is close to the one designated as "metaplastic autoimmune atrophic gastritis" (Park et al., 2010), and far from those defined by other authors (Vannella et al. 2011).

Therefore, when the atrophy of the gastric mucosa is clearly developed, with severe atrophy of the gastric body and fundus, the histological diagnosis of ABG can be relatively easy. Oxintopeptic glands are replaced entirely, or almost entirely, with intestinal glands (intestinal metaplasia), mucous glands (pseudoantral metaplasia) and other poorly differentiated glandular structures (Figs. 2 C, D, and 3). The endocrine cells of the oxyntic mucosa are spared from the process of atrophy, become hyperplastic what is commonly found in the ABG of autoimmune origin. Although these hyperplastic cells are considered to be enterochromaffin-like cells which are strongly reactive to the neuroendocrine marker chromogranin they can also express immunoreactivity to some peptide hormones such as ghrelin (Moreira et al. 2010).

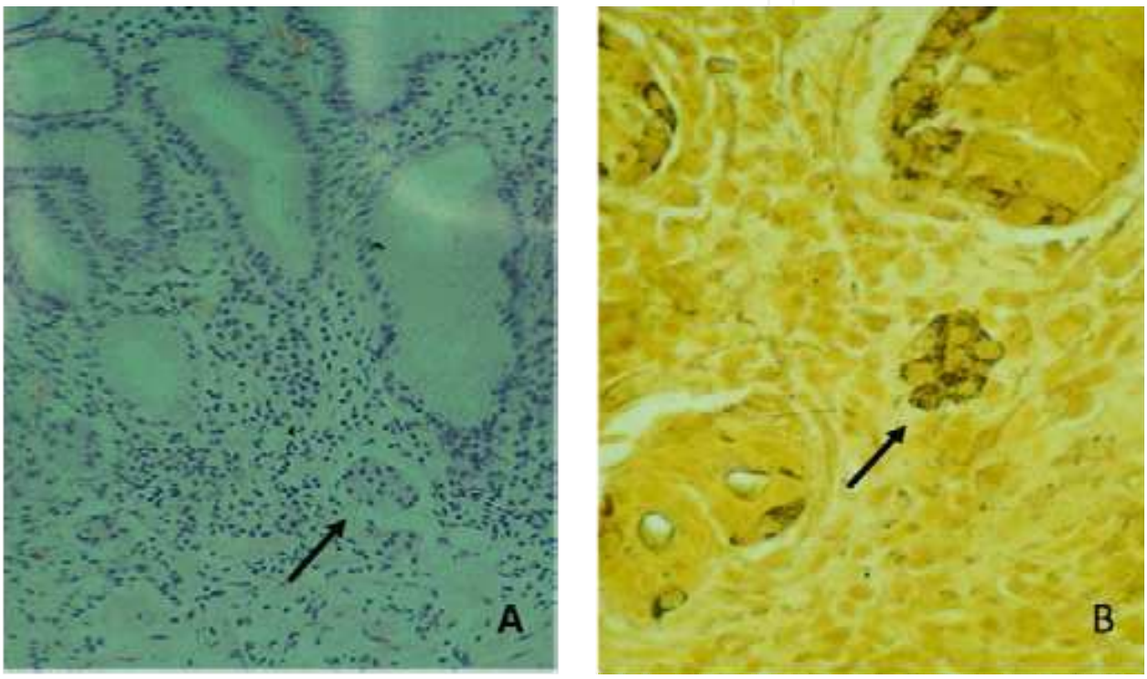

Fig. 4. Body gastric mucosa from a patient with autoimune ABG. A - Immersed in the *lamina propria* there is a nodule (arrow) composed of small cells, overlapping and even suggesting that it is related to endocrine cells (HE staining). B – Histological section of the same paraffin block stained by Grimelius technique showing hyperplasia of endocrine cells (argyrophilic cells) in the wall of the glands and hyperplastic nodules immersed in the *lamina propria* (arrow). These small nodules can be visualized in routine histological preparations, stained by HE and should raise the suspicion for the diagnosis of ABG. The presence of more intense inflammatory infiltrate in the *lamina propria* may mask the visualization of these endocrine nodules. For demonstrating the endocrine nature of these nodules, it is necessary special stainings as Grimelius technique for argyrophilic cells or immunohistochemistry using neuroendocrine markers, eg., chromogranin and neuron specific enolase

According to recent results from our laboratory regarding a large series of patients with ABG, we observed that most of the subjects presented an expressive number of ghrelin-immunoreactive cells in the different types of endocrine hyperplasia that occur in the atrophic body mucosa of these patients. Thus, ghrelin- expressing endocrine cells can be

easily detected in both the hyperplastic endocrine nodules present in the *lamina propria* and diffusely in the walls of metaplastic glands (Fig. 5). Both intestinal metaplasia and pseudoantral metaplasia were frequent findings in these patients with ABG. Among 60 patients with a diagnosis of ABG, 51 presented areas of pseudoantral metaplasia in the body atrophic mucosa and most of them, i.e., 37 (72.5%) presented ghrelin-immunoreactive endocrine cells in the wall of pseudoantral metaplastic glands (Moreira & Barbosa, 2011). This fact may be of help in the differentiation between antral and body mucosa in cases of ABG. One of the methods currently used in this differentiation is the demonstration the absence of gastrin-producing cells (G cells) in the metaplastic glands of the body (pseudoantral metaplasia) since these cells are only present in the glands of the antral mucosa. In doubtful cases, this differential characteristic between the antral and pseudoantral mucosa has been used to characterize the mucosa of the body with pseudoantral metaplasia, which does not contain G cells, and to differentiate it from the true antral mucosa, which contains G cells (Park et al. 2010).Since this type of characterization of pseudoantral metaplasia is based on a negative fact (absence of G cells), the frequent presence of ghrelin-immunoreactive cells in pseudoantral metaplasia (a positive fact) could be a more reliable marker for this purpose, since these cells are rare in the glands of the normal antropyloric mucosa.

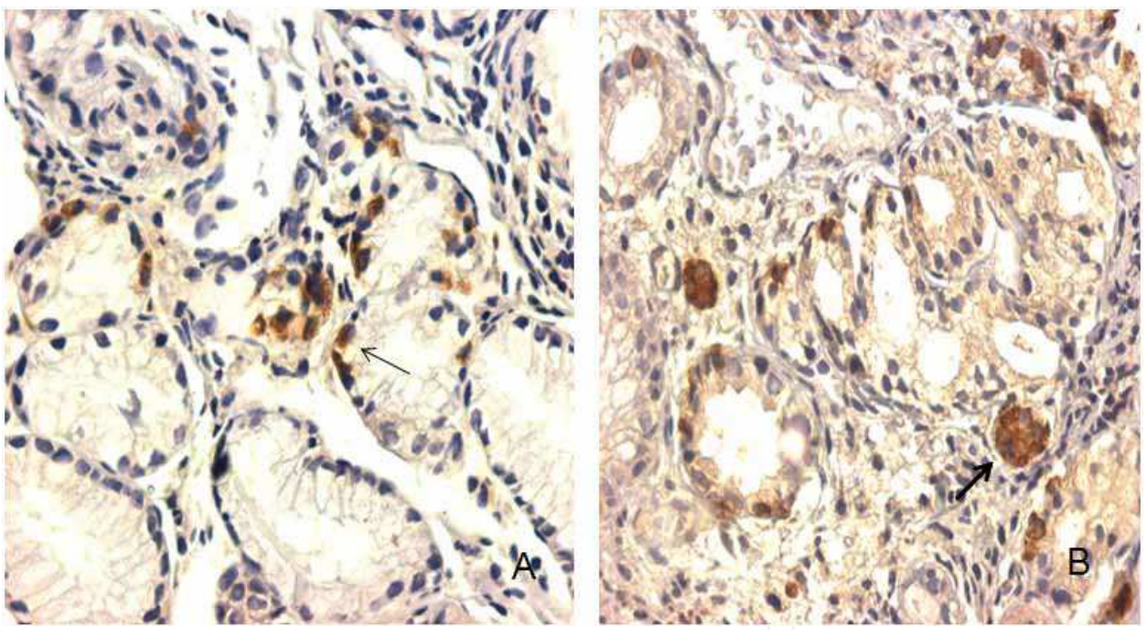

Fig. 5. A and B - Atrophic body mucosa of the stomach of a patient presenting ABG. Numerous glands exhibit pseudoantral metaplasia and most of them have endocrine cells immunoreactive to the peptide ghrelin (arrows). As ghrelin-immunoreactive cells are usually not present or are rare in antral mucosa this could be used as an reliable indication of pseudoantral metaplasia

This endocrine cell hyperplasia does not seem to occur in multifocal atrophic gastritis. Therefore, the presence of endocrine cell hyperplasia in the body mucosa of patients with ABG is a strong signal indicating the autoimmune etiology of the ABG. These florid changes of the body mucosa contrast with the discrete histological findings of the antral mucosa.

H. pylori is usually negative in these patients and, regardless of its presence, the atrophic gastritis with the morphological characteristics described above should preferably be regarded as autoimmune (Capella et al, 1999; Park et al, 2010). If necessary, serum anti-parietal cell and anti-intrinsic factor antibodies can be investigated to confirm this possibility. It should be noted that in a small percentage of patients with the pattern of morphological changes of the gastric mucosa typical of ABG, and clinically supposed to have autoimmune gastritis with pernicious anemia, the search for anti-parietal cells and/ or anti-intrinsic factor antibodies may give negative results.

Some patients with pernicious anemia have been reported to have severe histological changes of the antral mucosa indistinguishable from those seen in the gastric body (Lewin et al, 1976). Recently, severe atrophic gastritis of the antrum and body mucosa has been reported in association with systemic autoimmune diseases. This pathological condition was not associated with *H. pylori* or endocrine cell hyperplasia and may affect a especific group of patients with a particular type of autoimmune gastritis. Hypothetically, these patients would develop the production of antibodies directed against multiple cell lines of the gastric mucosa (Jevremovic, 2006).

3.3 Gastric polyps and atrophic body gastritis

As seen in this series, the main endoscopic diagnosis of "gastric polyps" was relatively common among patients undergoing endoscopy who had a final diagnosis of ABG as the most important disease. We do not know how many of the 196 cases of ABG studied also had gastric polypoid lesions that were not described in the findings of endoscopic diagnostic by being considered irrelevant or by having been omitted. Likewise we are not aware of the frequency of cases of gastric polyps which, considered being the major and only injury, were removed by polypectomy without adequate sampling for histology of the mucosa of the gastric body and antrum. We believe that many pathologists face this reality in the laboratory routine, which may result in the omission of the diagnosis of the most important underlying gastric disease often responsible for the presence of polyps, such as ABG (Jain & R Chetty, R, 2009; Haruma et al, 1993).

Endoscopically, the term "gastric polyp" is applied to any bulge or swelling of the gastric mucosa, whether of epithelial origin or from underlying tissues. From a structural viewpoint, however, the term is being used by pathologists to designate lesions mainly consisting of epithelial proliferation that project into the lumen of the organ. However, the nomenclature of these lesions has not been fully defined. Moreover, the terminology used for polyps of the stomach and the description of their morphological structure are very similar to those used to describe polypoid lesions of the colon, although the biological behavior of gastric and colonic polyps does not always show the same evolutionary pattern. Gastric polyps can be divided into several types, many of which are still poorly understood in terms of their etiopathogenesis. The types for whose definition there is a more general consensus are: hyperplastic polyps, adenomatous polyps (adenomas), mixed polyps, fundic gland polyps, hamartomatous polyps, and retention polyps (juvenile). These different types of polyps may be associated with clinical syndromes (syndromic polyps) including the Peutz-Jeghers, Gardner, Cronkhite-Canada and Crowden syndromes. Among these various types of gastric polyps, the most common are hyperplastic non-syndromic polyps, which are also those most often associated with ABG. It should be added that the histological pattern of gastric hyperplastic polyps may be indistinguishable from that of polyps of syndromic origin (Lam-Himlinet al, 2010).

The gastric hyperplastic polyps are the result of reactive hyperplasia of the foveolar epithelium in response to injury or to primary disease of the gastric mucosa, including chronic gastritis with glandular atrophy, as previously mentioned (Abraham et al, 2001). Apparently, these types of polyps almost never occur in normal gastric mucosa (Fig. 6 A).

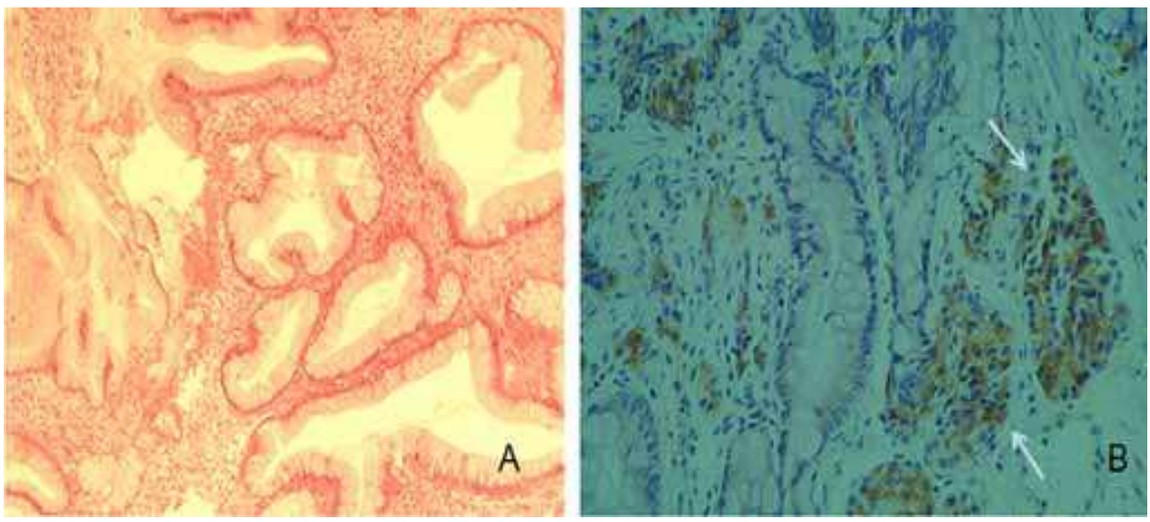

Fig. 6. Body gastric mucosa of a patient with the diagnosis of atrophic body gastritis. Intense hyperplasia of the foveolar epithelium (A), and of endocrine cells in the *lamina propria* (B) which could origin small gastric polyps. A – H.E. staining; B- Immunoperoxidase staining for chromogranin

Other endoscopic abnormalities often associated with ABG are the "gastric polyps" that occur in the body and fundus of the stomach and that derive from nodular proliferation of endocrine cells in the connective tissue of the *lamina propria (*Figure 6 B). They are usually multiple lesions located only in the body and fundus of the stomach and their presence should raise the suspicion of endoscopic ABG. Fusion of the endocrine hyperplastic nodules frequently occurs and the differential diagnosis of a neuroendocrine tumor should be considered. Since these nodules are usually multiple and not uniform, sampling of some of them does not always provide sufficient information for a final conclusion about the presence of endocrine neoplasia. In conclusion, there is a clinical and pathological significance of gastric polyps in relation to ABG because of its frequent association with both hyperplastic polyps and the polyps derived from endocrine cells proliferation (endocrine hyperplastic nodules or carcinoid tumors). However, the removal of these polyps must be accompanied by biopsies from the gastric antrum and body in order to characterize the primary process responsible for the appearance of polypoid lesions. The presence of these polyps may serve as a signal to the endoscopist of the presence of underlying disease or injury of the gastric mucosa, which is not always perceived by standard endoscopy.

3.4 Diagnosis of gastric mucosa atrophy: Endoscopic perspectives

The disagreement between endoscopic and histologic diagnosis for the presence of glandular atrophy of the gastric mucosa is not new and is relatively frequent (Torbenso et al, 2002). Standard endoscopy, although it can employ technical methods to study the thickness of the gastric mucosa such as stretching of the stomach wall by inflating air into the gastric

cavity, continues to have a low accuracy index. The degree of distension of the stomach wall and visualization of the submucosal vascular network depends primarily on the professional skill and experience of the examiner, as well as on the availability of good quality equipment. Even at centers specialized in gastrointestinal endoscopy, the endoscopic-histologic correlation is weak, with sensitivity and specificity of about 40 to 60% (Eshmuratov, et al. 2010).

Therefore, since the histological method is also not reliable, at least for cases of mild or moderate atrophy, its use in combination with endoscopy continues to rest on quicksand. Opening good perspectives for the near future, the endoscopic method has progressed with the description of new visualization techniques to amplify the resolution and definition of gastrointestinal mucosa details. Thus, endoscopic techniques involving magnification with high resolution have been reported to be considerably more reliable than standard endoscopy to identify normal gastric mucosa, chronic gastritis and gastric atrophy (Anagnostopoulos et al. 2007). The progress of endoscopic techniques and the availability of high-resolution confocal laser endomicroscopy are now starting to gain firmer ground in the detection of minute lesions of the gastrointestinal mucosa, among them the different degrees of gastric mucosa atrophy (Li, CQ and Li, YQ, 2010; Goetz, M. and Kiesslich, R. 2010; Canto, 2010).

4. Conclusions

The term ABG was used in the present article to designate cases of chronic gastritis with selective glandular atrophy of the mucosa of the stomach body while the antral mucosa continued to show a normal aspect or only minimal inflammatory changes. Extensive areas of intestinal metaplasia, pseudoantral metaplasia and endocrine cell hyperplasia often occur in the atrophic mucosa of the body. Taken together, these changes of the gastric mucosa strongly suggest the presence of an inflammatory process of autoimmune etiology accompanied or not by pernicious anemia of subclinical or even clinical evolution. Thus, the major importance of the histologic diagnosis of ABG resides in the fact that, for many dyspeptic patients submitted to upper digestive endoscopy this may be the first opportunity they have to receive a correct diagnosis of their main disease.

The histologic diagnosis of glandular atrophy of the stomach of mild or moderate grade is highly subjective and depends on many factors linked to tissue collection and processing. From an endoscopic viewpoint, the diagnostic imprecision is even greater. However, the more extreme grades of atrophy of the gastric mucosa are easy to interpret histologically when tissue samples are collected, processed and interpreted in an appropriate manner. When this is not the case, the histologic diagnosis of ABG is frequently inconclusive or equivocal. In the present study, out of 230 cases with an inconclusive histological diagnosis reviewed by an expert pathologist in the gastrointestinal area, 55 (24%) were confirmed as ABG, many of them after new histologic sections were obtained from paraffin blocks. Although in the cases studied the degree of gastric atrophy was intense and circumscribed to the body, the standard endoscopic exam showed a very low correlation with the histological findings. This fact associated with problems regarding tissue collection and processing impairs a conclusive diagnosis of ABG in many cases, with a consequent delay in the diagnosis of the principal disease of a patient who seeks a service of digestive endoscopy due to nonspecific dyspeptic complaints. However, new endoscopic techniques show a clear progress that promises to reverse the current secondary role of endoscopy in combination

with histology for the evaluation of patients with different degrees of gastric mucosa atrophy and consequently with chronic gastritis of autoimmune etiology.

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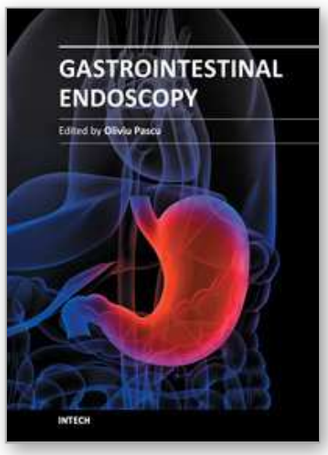

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Endoscopy has had a major impact in the development of modern gastroenterology. By using different data it provided a better understanding of pathogenic mechanisms, described new entities and changed diagnostic and therapeutic strategies. Meanwhile, taking advantage of many technical advances, endoscopy has had a developed spectacularly. Video-endoscopes, magnification, confocal and narrow-band imaging endoscopes, endoscopic ultrasounds and enteroscopes emerged. Moreover, endoscopy has surpassed its function as an examination tool and it became a rapid and efficient therapeutic tool of low invasiveness. InTech Open Access Publisher selected several known names from all continents and countries with different levels of development. Multiple specific points of view, with respect to different origins of the authors were presented together with various topics regarding diagnostic or therapeutic endoscopy. This book represents a valuable tool for formation and continuous medical education in endoscopy considering the performances or technical possibilities in different parts of the world.

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