Regression of intestinal metaplasia to cardiac or fundic mucosa in patients with Barrett's esophagus submitted to vagotomy, partial gastrectomy and duodenal diversion. A prospective study of 78 patients with more than 5 years of follow up

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Background. *Regression of intestinal metaplasia to cardiac mucosa in patients with Barrett's (BE) esophagus could alter the natural history of BE.*
Objective. *To determine the regression of intestinal metaplasia to cardiac mucosa in patients followed more than 5 years after operation, by repeated endoscopy with biopsy.*
Material and Methods. *This prospective study included 78 patients with BE submitted to combined vagotomy, antrectomy (an antireflux procedure), and Roux-en-Y gastrointestinal reconstruction with more than 60 months follow up. Patients were divided in 3 groups: (1) 31 with short-segment BE (≤30 mm length); (2) 42 with long-segment BE (31 to 99 mm length); and (3) 5 with extralong-segment BE (≥100 mm). Each patient had at least three endoscopic procedures with multiple biopsies during a mean follow up of 95 months (range, 60-220 months). Acid and duodenal reflux were also evaluated.*
Results. *Sixty-four percent of patients with short segment BE had regression to cardiac mucosa at a mean of 40 months after operation. Sixty-two percent of patients with long segment BE had regression to cardiac mucosa at a mean of 47 months postoperatively. No regression occurred in the 5 patients with extra-long segment BE. In 20% of patients, regression to fundic mucosa occurred between 78 to 94 months after surgery. One patient progressed to low grade dysplasia, but no patient progressed to high-grade dysplasia or adenocarcinoma. Acid and duodenal reflux studies demonstrated that in asymptomatic patients, reflux was abolished; 90% of the patients had a Visick grade of 1 or 2.*
Conclusions. *Vagotomy and antrectomy combined with duodenal bile diversion abolish acid and duodenal reflux into the distal esophagus in patients with BE, which is accompanied by a regression of BE from intestinal to cardiac or fundic mucosa in about 60% of patients. This regression is time dependent and varies directly with the length of BE. The potential for an antineoplastic effect, especially in young patients with long segment BE, suggests that this operation may become an attractive option as a definitive surgical treatment. Patients with short segment BE submitted to this procedure behave similar to patients submitted to Nissen fundoplication, and therefore in these patients, we do not advocate this complex operation.*

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BARRETT'S ESOPHAGUS(BE) IS AN ACQUIRED condition in which the normal squamous epithelium of the distal esophagus is replaced by an abnormal columnar mucosa containing intestinal metaplasia.[1-4] This complication secondary to chronic and severe reflux of acid and duodenal content into the esophagus[5,6] is associated with a 30- to 125-fold increased risk of development of esophageal

adenocarcinoma.[1-4,7] One of the main purposes of antireflux surgery is to stabilize the intestinal metaplastic mucosa and halt its progression to dysplasia and cancer, as well as to obtain, if possible, regression of intestinal metaplasia to cardiac mucosa, which does not carry a malignant risk.[8,9] We have published our results previously in patients with Barrett's esophagus and low-grade dysplasia, in whom after the operation herein described of acid suppression and duodenal diversion, there was regression to non displastic epithelium in 65% of patients. The current report describes our experience in 78 patients with short segment and long segment BE, but without dysplasia.[10]

The purposes of the present prospective study were: (1) to determine the histologic changes of the columnar lined epithelium by repeated endoscopy with multiple biopsies late after the proposed operation; and (2) to determine any changes in length of the columnar lined mucosa in the distal esophagus late after reoperation.

MATERIAL AND METHODS

Patients studied. The present prospective study started in 1987, for patients with Barrett's esophagus, and included patients operated on until 1997, closing the study on December 2002, in order to have at least 60 months of follow up. The details of this investigation have been published extensively elsewhere.[11,12] From the 166 patients with short- or long-segment Barrett's esophagus with intestinal metaplasia treated by this special surgical procedure, a total of 84 patients were operated on until 1997, and constitute the material of the present study. Six patients (7%) were lost to follow up although they are alive, leaving 78 (93%) patients who fulfilled the criteria for a complete subjective and objective follow up of more than 60 months. The 24 patients with BE containing low-grade dysplasia were excluded, because they were reported elsewhere,[10] as well as patients with intestinal metaplasia of the cardia, high-grade dysplasia, or adenocarcinoma.

Clinical questionnaire. A careful clinical assessment was performed in all patients included, asking for the presence of symptoms of gastroesophageal reflux (heartburn, regurgitation, or dysphagia), diarrhea (soft liquid feces) or early dumping syndrome (symptoms of sweating, fatigue and early postprandial diarrhea 10 to 30 minutes after eating). For late clinical evolution, a modified Visick gradation was employed, with the following criteria[13,14]: Visick I: asymptomatic; Visick II: mild or episodic symptoms controlled easily by medical treatment or diet adjustment with no need of permanent medication; endoscopic findings of mild esophagitis without reflux symptoms were included in this category; Visick III: frequent daily symptoms requiring permanent medical treatment; and Visick IV: severe symptoms requiring reoperation or producing a serious metabolic disturbance, such as dumping or incapacitating diarrhea.

Endoscopic examination. All endoscopic procedures were performed by two of the authors (A.C. and I.B.), employing an Olympus GIFXQ-20 endoscope (Tokyo, Japan). After a 12 hour overnight fast with the patient in supine left lateral position, the exact location of the squamous-columnar junction was measured at the beginning and at the end, of the procedure to avoid the "push" and "pull" effect of the endoscope.[14] The length of columnar-lined distal esophagus was measured as the distance between the squamous-columnar junction and the endoscopically located lower esophageal sphincter which is the point where the proximal extent of the gastric surgical folds met with the tubular esophagus.[9,14] The presence of erosions proximal to the squamous-columnar junction was recorded. The endoscopic procedure was performed before operation and at least 3 times post operatively in each patient. Four quadrant biopsies were taken 5 mm distal to the squamous-columnar junction. In-patients with short-segment BE, two more samples were taken 2 cm distal. Among patients with long-segment BE, 2 samples were taken every 2 cm distally, according to the length of the columnar mucosa. Therefore, between 6 to 16 samples were taken in each patient during each endoscopic procedure, with a mean of 8.5 samples per patient. Patients were divided in 3 groups[10]: (1) Short-segment Barrett's esophagus, (length $\leq$30 mm); (2) Long-segment Barrett's esophagus, (length of 31-99 mm); (3) Extralong-segment Barrett's esophagus, (length of $\geq$100 mm).

Definition of regression of BE. (1) Loss of intestinal metaplasia with presence of only cardiac and/or fundic mucosa on two consecutive endoscopic examinations. (2) Decrease in length of columnar lined mucosa of the distal esophagus of at least 3 cm.

Histologic analysis. All samples were submerged immediately in 10% formaline solution, sent for histologic examination, and stained with hematoxylin-eosine and Alcian blue stain at pH 2.5, Fundic mucosa was identified by the presence of parietal and chief cells at the deep glandular layer and cardiac mucosa by the presence of mucous-secreting

Table I. Features of patients with Barrett's esophagus submitted to surgical treatment (N = 78)*

	Short-segment BE (A) n = 31	*Long-segment BE (B) n = 42*	*Extralong-segment BE (C) n = 5*	P *value*
Age (year)	52 (29-73)	50 (16-72)	44 (28-54)	n.s.
Gender				
Men	13 (42%)	23 (55%)	4 (80%)	n.s.
Women	18	9	1	
Follow up (months)	84 (60-204)	101 (60-220)	96 (60-168)	n.s.
Timing for endoscopic procedures after operation (months)				
First endoscopy	28 (31)	27 (42)	24 (5)	n.s.
Second endoscopy	51 (31)	56 (42)	46 (5)	n.s.
Third endoscopy	73 (31)	82 (42)	86 (5)	n.s.
Fourth endoscopy	102 (6)	123 (10)	168 (2)	n.s.

*Parenthesis indicate number of patients.

columnar cells. Intestinal metaplasia was defined by the presence of goblet cells.

Monitoring of esophageal exposure to acid and duodenal juice. All examinations were performed after a 12-hour overnight fast. The details of the 24-hour acid reflux test as well as the 24-hour monitoring of bile reflux into the distal esophagus have been described previously.[10,11,15,16] The results of acid reflux test were expressed as the percentage of time during which the intraesophageal pH was less than 4 (normal values less than 4%); duodenal reflux was expressed as the percentage of time that bilirubin was measured in the esophagus with an absorbance of more than 0.2 (normal values less than 2%).

Surgical procedure. All patients underwent a truncal or selective bilateral vagotomy, closure of the diaphragmatic crura, an antireflux procedure, and antrectomy with a Roux-en-Y gastrojejunal anastomosis (60 cm Roux limb).[10-12] The antireflux procedure consisted of a 360-degree Nissen fundoplication in 35 patients or a posterior gastropexy with calibration of the cardia in 43 patients (Hill procedure).

Statistical analysis. The Chi-square test, the Mann Whitney test, and the variance analysis test were employed, accepting a $P < 0.05$ as significant. The Kaplan Meier test was employed for calculation of the loss of intestinal metaplasia over to time, employing the Mantel-Haenszel test for statistical evaluation. Continuous variables are presented as mean ± SD or SEM.

RESULTS

Demographic features of the 78 patients are shown in Table I. Thirty-one patients (40%) had short-segment BE, 42(54%) had a long-segment BE, and 5(6%) had extra long-segment BE. The mean follow up was similar for the three groups. The timing of the endoscopic examinations performed at least three times in each patient post operatively is outlined in Table I. Table II shows the histologic changes of the columnar mucosa of the distal esophagus after endoscopic surveillance. Patients with short-segment BE had persistence of intestinal metaplasia in only 36% of the patients over the mean follow up of 72 months while patients with long-segment BE demonstrated persistence of intestinal metaplasia in 38% after a follow up of 112 months. In contrast, all patients with extralong-segment BE had persistence of intestinal metaplasia after follow up of 84 months ($P < 0.001$). There was no significant difference in the time of regression of intestinal metaplasia to cardiac mucosa in the two groups (short and long segment BE). Cardiac mucosa persisted in 80 and 81% of the patients at a mean follow up of 78 and 94 months, respectively. However 4 patients (20%) with short-segment BE and 5 patients (19%) with long-segment BE had their cardiac mucosa regress to fundic mucosa 80 and 89 months post operatively. There was no progression to high grade dysplasia or adenocarcinoma in any patient in a total of 608 patients years.

The length of the columnar-lined mucosa did not change late after operation, but in 70% of the patients, there was endoscopic evidence of regenerating small islands of squamous mucosa, usually located near the squamous-columnar junction.

Acid and duodenal reflux into the distal esophagus before and after operation is shown in Table III. Among the patients with Visick grades I-II in all three groups, there was a marked decrease of acid reflux ($P < 0.001$). Among patients with long-segment with Visick grades III-IV, had similar values to their preoperative study. The 24-hour bile

Table II. Histologic changes of the BE in the distal esophagus in patients post operatively at the last endoscopy (N = 78)

Histologic changes	*Short-segment BE (A) n = 31*	*Long-segment BE (B) n = 42*	*Extralong-segment BE (C) n = 5*	P *value*
Persistence of IM	11 (36%)	16 (38%)	5 (100%)	AvC $P < 0.01$ BvC $P < 0.001$ AvB $P > 0.5$
Regression to cardiac mucosa	20 (64%)	26 (62%)	0	n.s.
time of occurrence (months)	40	44	—	
Regression to fundic mucosa	4 (20%)	5 (19%)	0	n.s.
time of occurrence (months)	75	89		
Progression to low-grade dysplasia	0	1 (2%)	0	n.s.
		108 months after surgery		
Progression to high-grade dysplasia or Adenocarcinoma	0	0	0	
Length of the columnar-lined mucosa (mm)				
Preop (mean ± SD)	27 ± 3.6	55.6 ± 10.3	112 ± 12	n.s.
Post	25 ± 4.1	53.5 ± 9.8	110 ± 11	

n.s., Not significative; *IM*, inestinal metaplasia; *BE*, Barrett's esophagus.

Table III. Gastroesophageal reflux into the before and after surgical treatment in patients with Barrett's esophagus (BE)

	Short-segment BE (A)	*Long-segment BE (B)*	*Extralong-segment BE (C)*
Acid reflux % of time with pH < 4 in 24 hrs			
Before operation	16 ± 12 (27)	21 ± 16 (26)	46 ± 28 (4)
After operation			
Visick I-II	1 ± 1 (17)*	3 ± 3 (18)*	2 ± 1 (4)*
Visick III-IV	– (a)	21 ± 24 (3)	– (b)
Duodenal reflux % of time with bilirubin absorbance >0.2 in 24 hrs			
Before operation	21 ± 18 (17)	33 ± 28 (21)	46 ± 13 (3)
After operation	1 ± 1 (12)*	1 ± 2 (14)*	2 ± 1 (3)*
	$P < 0.0001$	$P < 0.0001$	$P < 0.0001$

Values are x ± SD; Parentheses indicate number of patients evaluated.
(a) The 2 patients with Visick III and IV had no reflux studies after surgery.
(b) There is no Visick III or IV patient.
*Different from before operation, $P < 0.001$.

monitoring showed a significant decrease in duodenal bile reflux post operatively in all groups, independent of Visick grade and length of the columnar mucosa.

Table IV shows the late clinical results in all patients, according to the length of the columnar mucosa. The reason for classifying patients as Visick grades II and III in the patients with short-segment BE was the presence of persistent diarrhea. The one patient with Visick grade IV had severe dumping syndrome. None of the patients in this group had any reflux symptoms. All 5 patients with extralong-segment BE, were Visick grade I and none had reflux symptoms. In contrast, only 28 of the 42 patients (67%) with long-segment BE were Visick grade I. The causes for Visick grade II were endoscopic esophagitis with erosions in 5 patients, mild strictures in 2 patients who need periodic dilatation and intermittent diarrhea in one patient. None of these 8 patients had symptoms of reflux. Four patients were graded as Visick III gradation because of persistant daily heartburn

Table IV. Late clinical results in 78 patients with Barrett's esophagus submitted to surgical treatment

	Short-segment BE n = 31	*Long-segment BE n = 42*	*Extralong-segment BE n = 5*	*Total*
Visick I	28 (90%)	28 (67%)	5 (100%)	61 (78%)
Visick II	1 (3%)	8 (19%)	—	9 (11%)
Visick III	1 (3%)	5 (12%)	—	6 (8%)
Visick IV	1 (3%)	1 (2%)	—	2 (3%)
Recurrence of reflux	0	9	0	9 (12%)
Metabolic disturbances	3	2	0	5 (6%)

Table V. Regression of the length of the columnar lined epithelium of the distal esophagus in patients with Barrett's esophagus submitted to some form of antireflux surgery

First author (Reference)	*Year*	*No. of cases*	*Follow up (months)*	*Length BE*	*Regression of CLM*
Radigan (17)	1977	13	¿?	> 3 cm	0
Skinner (18)	1983	12	42	> 3 cm	0
Starnes (19)	1984	8	26	> 3 cm	0
Ranson (26)	1983	6	68	> 3 cm	4 (67%) Partial regression
Williamson (27)	1990	37	50	> 3 cm	4 (11%) Partial regression
DeMeester (20)	1990	35	36	> 3 cm	0
Attwood (28)	1992	19	36	> 3 cm	2 (10.5%)
Sagar (29)	1995	56	66	> 3 cm	5 (8.9%)
Csendes (21)	1997	65	28	> 3 cm	0
Csendes (15)	1998	151	100	> 3 cm	0
Stein (22)	1998	16	12	> 3 cm	0
Patti (23)	1999	21	23	> 3 cm	0
Chen (30)	1999	45	36	> 3 cm	Partial regression 2 cm
Csendes (24)	2000	39	120	> 3 cm	0
Csendes (11)	2002	215	58	> 3 cm	0
Bamehriz (31)	2002	21	39	> 3 cm	8 (38%)
Parrilla (25)	2003	52	84	> 3 cm	0

BE, Barrett's esophagus; *CLM*, columnar lined mucosa.

and endoscopic esophagitis; all needed permanent proton pump inhibitors. One patient also had dumping and diarrhea. Only 1 patient was Visick grade IV because of severe dumping syndrome. In summary, 9 patients (12%) had recurrence of reflux esophagitis, while 5 patients (6%) had some metabolic disturbances.

DISCUSSION

The present study suggests that regression of intestinal metaplasia to cardiac or fundic mucosa is a frequent finding after eliminating acid and bile reflux by vagotomy and antrectomy, fundoplication and Roux-en-Y reconstruction. This regression of intestinal metaplasia, the important pre malignant mucosa, is time-dependent and inversely related to the length of the columnar mucosa lining the distal esophagus (length-dependent), at that time of surgical treatment. This study has some special features: it has the longest follow up (95 months), every patient has been studied at least 3 times by endoscopy and biopsy post operatively and the results are closely correlated with the length of BE at the time of surgical treatment.

Concerning the "regression" of BE after antireflux surgery, this topic has been a matter of discussion and confusion for many years. In the early studies, the investigators were mainly concerned with whether there was a regression or decrease in the length of the columnar-lined mucosa, which seemed to be synonymous with success and "regression" of Barrett's esophagus. Many earlier studies defined Barrett's esophagus as the presence of 3 or more cm of the distal esophagus lined by columnar epithelium with no clear mention of the presence of intestinal metaplasia, and therefore, regression was confused with the eventual loss of intestinal metaplasia. Table V shows all studies concerning the regression or decrease in length of the columnar lined mucosa of the distal esophagus. There are 17 publications, eleven of which (references 11,15,17-25) clearly

stated that there was no decrease in the length of the columnar mucosa. Only 6 reports(26-31) mention some element of partial regression.

The presence of islands or patches of squamous re-epithelization is a frequent endoscopic finding late after our form of antireflux surgery. This endoscopic findings was seen in 70% of our patients despite the lack of a significant change in the total length of columnar mucosa. It is important to notice that after operation, the esophagus can "descend" 2 cm due to the antireflux component of the operation, which means that the squamous-columnar junction will also descend 2 cm, but with no change in the total length, of the BE.[10,11,24] In contrast, beneath the island of squamous epithelium, mixed components of squamous and columnar epithelium can be seen, as first described many years ago by Skinner et al.[18] and later by others.[26,28,32,33] It is important to "underline" the report of Skinner et al[18] in 1983 which recognized that some patients can have an overlayer of squamous epithelium and an underlayer of glands with incomplete intestinal metaplasia, and therefore this re-growth of squamous epithelium over the Barrett's epithelium could "mask" the true presence of underlying intestinal metaplasia.

The most important observation concerning "regression" of BE, is related to the disappearance of intestinal metaplasia from the Barrett's mucosa after surgical treatment. Regression of low-grade dysplasia to nondysplastic mucosa, has been published previously.[10] Other groups have studied the loss of intestinal metaplasia in patients with short segment BE after antireflux surgery. DeMeester's group published 2 articles concerning this issue, reporting 15 patients in 1998 and 22 patients in 2002.[9,34] The loss of IM among these patients was 59%. Bowers et al[35] and Oelschlager et al[36] reported regression in 33% and 55% of patients, respectfully. Our values were similar, although follow up was much longer.

Table VI shows the loss of intestinal metaplasia in patients with long segment BE after antireflux surgery. From a total of 33 articles concerning surgical treatment of BE over a period of 24 years (1980-2004), there are 11 articles which mention the effect of some form of antireflux surgery on intestinal metaplasia[9,15,21,25,26,30,32,34,36,37,38]; 9 were published from 1998 to 2004, and are included in this table. Six articles report no regression of IM, while two reported a 2% to 30% of loss of IM.[33,35] In the same table we show our results concerning 42 patients with long segment BE in whom 62% had loss of intestinal metaplasia. The loss of intestinal metaplasia that we observed is greater than in classic antireflux surgery and this regression appears to be time dependent and length dependent. We believe that our more impressive results are related to the surgical procedure we used to treat these patients with BE. In addition to a classic antireflux procedure (fundoplication), we also performed an acid suppression procedure (vagotomy-gastrectomy) and a complete and permanent diversion of duodenal content (Roux-en-Y-reconstruction), This operative procedure was very effective in controlling both acid and duodenal reflux as shown by our objective measurements. We and others (references 39-42) believe that bile reflux, especially among patients with long-segment BE, has an important and crucial role in the pathogenesis of intestinal metaplasia —> dysplasia———> carcinoma sequence. We have demonstrated previously that our operative approach of acid suppression diversion of duodenal content and fundoplication, produces regression of low-grade dysplasia to non-displastic mucosa in 65% of the patients.[10] In addition there was no progression to high-grade dysplasia or adenocarcinoma. In the current study we have tried to minimize the potential for error in biopsy sampling, by taking several multiple biopsy samples in each patient several times after operation. Besides, intestinal metaplasia is usually located in the area distal or beneath the squamous-columnar junction and therefore that is why 4 quadrant biopsy samples are taken at that area, as we and others have shown before.[6,43-45] We acknowledge that this technique may include some sample

Table VI. Loss of intestinal metaplasia in-patients with long segment Barrett's esophagus submitted to antireflux surgery

First Author (Ref.) Year	*No. of patients*	*Follow up (months)*	*Loss of IM*
Csendes (15) (1998)	152	108	0
Chen (30) (1999)	45	48	1 (2%)
Patti (23) (1999)	21	23	0
Low (32) (1999)	14	25	2 (14%)
Bowers (35) (2002)	33	54	10 (30%)
Parrilla (25) (2003)	52	84	0
Gurski (9) (2003)	44	60	0
Oelschlager (36) (2003)	36	40	0
Abbas (38) (2004)	26	29	?
Csendes (current study) (2005)	42 (31-99 mm)	101	26 (62%)
	5 (≥100 mm)	96	0

Parenthesis indicate length of Barrett's esophagus.

error, however, our technique should have minimized this possibility.

In the present study, we have tried to address some of the difficulties proposed by Shaheen and Bozurski[46] who called the attention to the following facts concerning the question whether antireflux surgery can alter the natural history of Barrett's esophagus: (1) the number of patients included in each report is insufficient to draw any firm conclusion; (2) the changes in histology may be secondary to sampling error; (3) attempts to measure the length of the columnar mucosa are often confounded by several factors; (4) there is often no control group to compare surgically altered patients; and (5) no clear data on the durability of any histologic change has been firmly provided.

Our study addresses the majority of their comments because: (1) we included a large number of patients (n = 78); (2) we took multiple biopsy samples during each endoscopic procedure and each patient underwent three or four surveillance endoscopies; (3) we measured carefully the length of the columnar mucosa; and (4) although we have no control group; (5) we have objective data for a long period of follow up to demonstrate the durability of the histologic changes. Classic antireflux surgery is able to obtain regression of intestinal metaplasia into cardiac mucosa in patients with short segment BE, but no regression is seen in patients with long-segment BE. We maintain that our surgical treatment differs form these reports for several reasons. First in a long-term follow up (more than 7 or 8 years) after classic antireflux procedures, recurrence of reflux increases progressively and therefore is time-dependant. As we have shown in previous publications the majority of publications concerning antireflux surgery in Barrett's esophagus have less than 60 months of follow up.[10-12,47] Second in patients with Barrett's esophagus, duodenal reflux has an important role in the pathogenesis of intestinal metaplasia and dysplasia. Three studies have evaluated the presence of duodenal reflux after antireflux surgery. Stein et al[22] studied 16 patients one year after fundoplication and showed no duodenal reflux in any patient. Parrilla et al[25] studied 12 patients before and 1 year after operation, finding 8.3% of positive test (1 patient) early after operation. We evaluated 24 patients 8 to 10 years after only fundoplication, finding a positive reflux test in 95% of patients.[15] Therefore, it seems clear that there is need for long term follow up and not only an evaluation 1 year postoperatively. If this duodenal reflux together to acid reflux is completely controlled for a very long period after operation, as seen in the present study, the stimulus for the presence and persistence of intestinal metaplasia is absent. As this type of mucosa seems to be dynamic and reversible, there is a progressive regression to cardiac mucosa, which is clearly dependent on the length of Barrett's mucosa and the duration of follow up.[9,35] We agree with the hypothesis of DeMeester et al[9] who postulate that IM is a cell response to luminal stimuli (duodenal reflux), which is reversible.

In summary, our study shows that combining acid suppression, duodenal diversion and fundoplication effectively abolishes permanently acid and duodenal reflux into the distal esophagus and produces marked histologic regression of intestinal metaplasia to cardiac mucosa, which is dependent on the length of columnar mucosa (length dependent) and a long term follow up (time dependent). Among patients with short segment BE the regression of IM is similar to the results of fundoplication alone, and therefore we believe that laparoscopic fundoplication is an adequate operation in these patients. In contrast, in patients with long segment BE, we have shown that regression of intestinal metaplasia occurs after our operative approach. This regression of intestinal metaplasia should decrease the risk of future cancer and thereby can change the natural history of this disease. We maintain that our operative approach in young patients with long segment BE, may be a better treatment approach than classic antireflux surgery.

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