IS GASTROESOPHAGEAL REFLUX DISEASE INFLUENCED BY DUODENOGASTRIC REFLUX ?

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IS GASTROESOPHAGEAL REFLUX DISEASE INFLUENCED BY DUODENOGASTRIC REFLUX ? (Abstract): BACKGROUND: It is well known that the gastroesophageal reflux is a frequent disease in medical practice. A lot of factors are involved in its pathogenesis. The aim of our study was to find out if the duodenogastric reflux is one of these factors. We also looked for the pathological conditions associated with duodenogastric reflux. MATERIAL AND METHODS: We studied a lot of 59 cases (30 females and 29 males) with ages between 39 and 54 years old suffering from gastroesophageal reflux disease (GERD). GERD, duodenogastric reflux were confirmed by upper gastrointestinal endoscopy. The abdominal ultrasound exam were used to evaluate the duodenal and gallbladder motility (ejection fraction, EF). The severity of GERD was appreciated using GERD Q-score. RESULTS: The women to men ratio was 30 to 29 and the mean age was 42 ±2 years. Cholesterolosis was found in 45.7% (n=27) from the patients. From these patients, 92.6% (n=25) had a ejection fraction (EF) above normal (hyperkinesia) and only two patients had normal gallbladder motility. From the patients with cholesterolosis, 20 (74%) had duodenogastric reflux. The other 32 patients without cholesterolosis had no duodenogastric reflux neither. We noted a strong statistical correlation between the cholesterolosis and duodenogastric reflux (P=1,115x10^{-8}). The duodenogastric reflux was found in 33.89% (n=20) from the patients and half of them (n=10) had associated esophagitis; we found a strong statistical correlation between duodenogastric reflux and esophagitis: P=1,543x10^{-13}. All the patients had GERD Q-scores higher than 7 points; the mean score was 11.25±4.04 points (range 8-18). The mean Q-score value for the cases with duodenogastric reflux was 16.2±3.37; the cases without duodenogastric reflux (n=39) had a mean Q-score of 9.2±1.23 (P=3,138x10^{-9}). CONCLUSION: These results suggest that biliary disease can contribute to the duodenogastric reflux and the duodenogastric reflux aggravates gastroesophageal reflux.

KEY WORDS: GASTROESOPHAGEAL REFLUX; DUODENOGASTRIC REFLUX; DUODENAL MOTILITY; GALLBLADDER MOTILITY; GALLBLADDER EJECTION FRACTION; GERD Q SCORE

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INTRODUCTION

Our interest for gastroesophageal reflux is due to the high frequency of this disease. The well-known causes of gastroesophageal reflux disease (GERD) are: impaired esophageal clearance, the altered antireflux mechanism and the diminished gastric motility. The antireflux mechanism include: the pressure of low esophageal sphincter, Hiss's angle and the abdominal part of esophagus. There are some date which describe also a duodenogastric reflux associated with gastric ulcer, esophagitis and gallbladder lithiasis [1,2].

We wanted to know how frequent the duodenogastric reflux in patients with GERD is. We also tried to find which are associated disease with the duodenogastric

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reflux and if the duodenogastric reflux can aggravate GERD.

MATERIAL AND METHODS

We retrospectively reviewed 59 patients with GERD. Different data (demographic, GERD severity, duodeno-gastric reflux, gallbladder ejection fraction, duodenal motility) were analysed.

The diagnosis of GERD was done using clinical data and upper endoscopy examination. The duodenogastric reflux was also proved by upper digestive endoscopy (bile inside the stomach).

The abdominal ultrasound examination was used to diagnose the gallbladder diseases and to evaluate the gallbladder motility; in this way we calculated gallbladder volume (*V*) using the formula $V = 0.5 \times L \times l \times g$ where *L* is the gallbladder length, *l* is the width of gallbladder and *g* is the thickness of gallbladder wall [3]. The ejection fraction (EF) was then calculated using the formula $EF = V_1\text{-}V_2 / V_1 \times 100$ were V_1 is the fasting gallbladder volume and V_2 is the volume after the ingestion of Boyden meal. Normal EF values are 50 to 60%; the cases with higher values were noted as „hyperkinesia". The duodenal motility was also evaluated using abdominal ultrasound exam and it was noted as „normal" or „increased".

In order to evaluate the GERD we calculated the GERD Q-score [4-6]. The Q-score contains 6 questions: 4 items about symptoms and 2 items about the impact of symptoms on the patient's life (health related quality of life) (Table I). The answers are filled in by the patient and than evaluated by the doctor. The total score was calculated by adding the point values for each corresponding answer. Increasing scores correlated with GERD severity (Table I).

Data were included in a MS Excel database and statistically analyzed; bivariate analysis (*t* test and χ^2 test) was used. A $P < 0.05$ was considered statistically significant.

Table I GERD Q score questionnaire [4]

Symptoms and health related quality of life (HQLR)	How many times does this occur per week?			
	0 Days	1 Day	2 to 3 Days	4 to 7 Days
How often have you had burning sensation in the chest (heatburn) ?	0 p	1 p	2 p	3 p
How often have you felt food or liqiud stomach contents turning it in the mouth or throat (regurgitation) ?	0 p	1 p	2 p	3 p
How often have you had pain in the upper abdomenum (epigastric pain) ?	3 p	2 p	1 p	0 p
How often have you had nausea ?	3 p	2 p	1 p	0 p
How often have you had trouble sleeping due to heartburn or regurgitation ? (HQLR - item)	0 p	1 p	2 p	3 p
How often have you taken medication for heartburn and regurgitation further other than those prescribed by a doctor (ranitidine, maalox, dicarbocalm) (HQLR - item)	0 p	1 p	2 p	3 p

The total score is interpretated as follows: *0 to 2*: no probability of GERD; *3 to 7*: Low probability of GERD (50% likelihood of GERD); *8 to 10:* GERD (discomfort ± disturbing symptoms); *11 to 18*: severe GERD (discomfort ± disturbing symptoms)

RESULTS

The women to men ratio was 30 to 29 and the mean age was 42 ±2 years.

Cholesterolosis was found in 45.7% (*n*=27) from the patients; 54.3% (*n*=32) did not have any gallbladder diseases. From the patients with cholesterolosis, 92.6% (*n*=25) had a ejection fraction (EF) above normal (hyperkinesia) and only two patients had normal gallbladder motility (EF 50 to 60%). From the patients with cholesterolosis, twenty (74%), had duodenogastric reflux. The other 32 patients without cholesterolosis had no duodeno-gastric reflux neither.

We noted a strong statistical correlation between the cholesterolosis and

duodenogastric reflux (χ^2 test; $\boldsymbol{P = 1{,}115 \times 10^{-8}}$).

The duodenogastric reflux was found in 33.89% (*n*=20) from all the patients and half of them (*n*=10) had associated esophagitis; we found a strong statistical correlation between duodenogastric reflux and esophagitis: χ^2 test; $\boldsymbol{P = 1{,}543 \times 10^{-13}}$.

All the patients with duodenogastric reflux (100%; *n*=20) had increased duodenal motility too. Among the other 39 patients without duodenogastric reflux, only 12.8% (*n*=5) had increased duodenal motility; this correlation is also statistically significant (χ^2 test; $\boldsymbol{P = 8{,}439 \times 10^{-10}}$). We noted similar results for galbladder hyperkinesia: all the patients with duodenogastric reflux (100%; *n*=20) versus five patients (12.8%) without duodenogastric reflux (χ^2 test; $\boldsymbol{P = 8{,}439 \times 10^{-10}}$).

All the patients from the study group had GERD Q-scores higher than 7 points; the mean score was 11.25±4.04 points (range 8-18). 67.8% (*n*=40) had a Q-score of 8 to 10 (mean: 9±1.13; range: 8-10) and 32.2% (*n*=19) had a score of 11 to 18 (mean: 16±3.27; range: 11-18).

The mean Q-score value for the cases with duodenogastric reflux was 16.2±3.37; range: 8-18; out of 20 cases with duodenogastric reflux, 35% (*n*=7) had high GERD Q-scores (11 to 18) and associate esophagitis demonstrated by endoscopy. The cases without duodenogastric reflux (*n*=39) had a mean Q-score of 9.2±1.23 (range 8-18): The Q-score was statistically higher in duodenogastric reflux group (*t* test; $\boldsymbol{P = 3{,}138 \times 10^{-9}}$).

We also noted significant higher Q-scores values for the patients with gallbladder hyperkinesia (14.16±4.06 vs 9±1.26; *t* test; $\boldsymbol{P = 1{,}688 \times 10^{-9}}$) and for the patients with increased duodenal motility (14.5±4.38 vs 9.2±1.02; *t* test; $\boldsymbol{P=2{,}832 \times 10^{-8}}$).

DISCUSSION

The duodenogastric reflux was described in about 25% cases of patients with GERD according to the paper of Brillantino A et al. [1]. They described duodenogastric reflux associated with gastric ulcer, gallbladder lithiasis and esophagitis and they pointed that the duodenogastric reflux can damage the gastric and esophageal mucosa due to the fact that it contains biliary acids and pancreatic juice [7-8]. It is not clear if the duodenogastric reflux can sometimes be found with normal people as a physiological event or is a pathological dismotily associated with gastroesophageal reflux.

In our paper, we noticed that duodenogastric reflux is found in 34% of patients with GERD versus 25% mentioned in Brillantino's paper [1].

The duodenogastric reflux differs from transpiloric flow in the fact that it operates the bile and pancreatic juice inside the stomach and it happens in the interdigestive phase. Johnson AG noticed that none of these waves of transpiloric retrograde flow is due to antiperistalsis waves from D_2 [9].

In experimental study done on volunteers by intubation technique Keane FB et al. [8] have been measured the antral and duodenal pressure, the pH, the levels of bicarbonate, biliary acids and trypsin in gastric and duodenal juice. The polientilenglicol (PEG) was used as a marker which indicates the direction of fluid movement. All the data were recorded simultaneously with the phases of interdigestive motor complexes (IMC).

The authors found that the duodenogastric reflux has two components: the secretory phases which occur before phase III of IMC and the motor phase which occurs in the II phase of IMC. Due to the fact that after the phase III of IMC the contents of duodenogastric reflux is evacuated from the stomach we can say that III phase acts as a gastric clearance.

In our study we found that all cases of gallbladder cholesterolosis have high values of EF meaning hyperkinesia. We also noticed that cholesterolosis is associated with enhanced duodenal motility.

These facts allow us to speculate that the gallbladder bile which contains a large amount of cholesterol and which is

evacuated quickly in the duodenum can induce an enhanced duodenal motility.

The duodegastric reflux requires two conditions: the pylorus opening and the retrograde duodenal motor wave. We think that enhanced duodenal motility can fulfill these two conditions. So, the gallbladder cholesterolosis can be one of the etiological factors which produce duodenogastric reflux.We did not exclude a lot of other patological conditions wich can produce duodenogastric reflux alsa well as a methods for its diagnosise [10-16].

All cases with duodenogastric reflux have high GERDQ score that means the duodenogastric reflux can aggravate gastroesophageal reflux; in this way, from the 20 cases with duodenogastric reflux, 10 cases had esophagitis.

CONCLUSIONS

The duodenogastric reflux is associated with GERD in 34% from cases. The association of duodenogastric reflux with gallbladder cholesterolosis can induce the supposition that duodenal bile rich in cholesterol can be the trigger for enhanced duodenal motility and duodenogastric reflux which may aggravate gastroesophageal reflux.

CONFLICT OF INTERESTS

Authors have no conflict of interests to declare.

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