INTRODUCTION

Eosinophilic esophagitis (EE) was described in 1977 by Landres (1) in a patient with achalasia, but it has been regarded a proper entity over the last decade. It is charac-
characterized by the presence of eosinophils in the esophageal wall, not involving other sections of the gastrointestinal tract. EE has been more frequently reported in the pediatric population but is currently becoming increasingly recognized in the adult population.

In young males the most common presenting symptoms are intermittent dysphagia and food impaction episodes (2-4); it is estimated that dysphagia attacks develop for more than a week in 60% of patients, and in young adults half of food impaction episodes are caused by EE (4,5). Eosinophilic leukocyte infiltration in the esophageal wall and their degranulation in nervous and muscle tissues could play a role in the motor and structural disorders of EE (2,6,7).

All studies agree that 80-90% of patients have endoscopic features, most of them non-specific (8,9) and none of them are pathognomonic of EE (10).

Sometimes the endoscopic patterns do not explain food impaction episodes, and in these cases a manometric study could be reasonable.

Motility studies in patients with EE are documented in The American Gastroenterological Association Review, where peristaltic abnormalities were reported in 30 of 77 adult patients with EE (10). Later, Lucendo et al. (3) described 57 of 30 patients with a hypoperistaltic motor pattern and 30% with a hyperkinetic pattern.

The aim of this study was to evaluate the clinical aspects, endoscopic features, pH-metric, and manometric disorders in EE, and to analyze if there was a correlation between them.

MATERIAL AND METHODS

Patients

During 12 months we included prospectively 11 adult patients with EE diagnosed by histological criteria in the proper clinical context and in the absence of other eosinophilic infiltration diseases, as the American Gastroenterological Association recommended. The presence of 15 or more intraepithelial eosinophils per high-power field (x400) in biopsy specimens was diagnostic of EE. We suspected EE in young adults with food impaction episodes or recurrent dysphagia. Consent was obtained from all patients.

Every patient was interviewed about age, personal or familiar history of allergies, duration and frequency of dysphagia, and number of urgent endoscopic explorations to resolve food impaction.

The degree of peripheral eosinophilia was documented, and it was considered abnormal if greater than 7% of the total leucocyte count.

Stationary esophageal manometry and 24-hour pH-metry were performed in all patients. Proton pump inhibitors were stopped a week before the procedure, and other treatments that could alter intragastric acidity or esophageal motility were interrupted 72 hours before.

Manometric study

Stationary esophageal manometry performed with a pneumatic hydraulic capillary perfusion system using an eight-channel multilumen catheter with 2-cm separations was performed, each channel hooked up to a PC using the program System-MMS (Sistema Solar GI Manometry). To carry out the manometry we followed the recommendations by Castell (11), and for an interpretation of results we referred to recommendations by Leite, Ho, and Tutuian (12-14).

PH-metry

A twenty-four-hour ambulatory pH-metry was performed using a Holter Mark III (Synectics Medical, Irving, Tx) with a frequency of 4-second intervals hooked up to an antimony electrode and specific software (Polygraf version 5.0. Synectics Medical, Stockholm, Sweden). The antimony electrode was placed 5 centimeters proximal to the lower esophageal sphincter (localized by manometric study) after calibrating the catheter for pH 1.07 and 7.01. We considered a study pathological when DeMeester’s score was more than 14.24 and/or total acid exposure in the lower third of the esophagus was more than 4% (15,16).

RESULTS

Clinical aspects

Eleven adult patients were diagnosed with EE. All of them were males with a mean age of 35 ± 3 years. Six patients (54%) had no history of allergies. Three patients were allergic to pollen, 2 to grass, 3 to dust mites (one of them kiwi and beer too), and 2 had asthma. Four (36%) patients had a family history of allergies. Three (27%) patients had slightly high levels of eosinophils in their blood: 0.5 x 10^9/microliter, 0.7 x 10^9/microliter and 1 x 10^9/microliter (normal values lower than 0.5 x 10^9/microliter).

Symptom duration was 1 to 20 years. One patient had specific attacks of dysphagia after eating egg and pulses; other patient referred the starting of symptoms after a pneumonia.

The number of dysphagia attacks was between 1 and 12 per month. In 7 (64%) cases an emergency endoscopy was needed to resolve food impaction.
Endoscopic findings

All endoscopies showed pathological features. The most prevalent finding was “felinization esophagus” (45%). Four (36%) endoscopies showed mucosal abnormalities like white exudates, 3 (27%) patients had Schatzky’s rings, and in 2 cases there was luminal stenosis (in middle and distal esophagus) that did not block the passage of the endoscope. Two (27%) mucosal tearings during the procedure were described: one of them in a middle-esophageal stenosis and one in the gastroesophageal junction.

Manometric and pH-metric studies

The manometric study of the upper esophageal sphincter was normal in all patients, including pressures and pharyngoesophageal motor coordination. A lower esophageal sphincter study was normal in 9 (81%), and in 2 (18%) the esophagus was hypotensive but with a normal dynamic behavior and with no pathological gastroesophageal reflux in pH-metric study. The esophageal body study showed 6 (54%) motility disorders: 5 (83%) were hypomotility motor patterns like ineffective esophageal motility, and 1 case showed a hyperdynamic abnormality (distal esophageal spasm). Ineffective esophageal motility was defined as more than 30% of lower amplitude contractions (< 30 mmHg) or nontransmitted contractions (12,13). Distal esophageal spasm was described as more than 20% of simultaneous contractions following wet swallows (14). Two (18%) patients showed pathological reflux during 24-hours pH monitoring. Table I shows the findings described.

CONCLUSION

EE is a disease of increasing interest mainly affecting young adult males with an unknown real prevalence (1,2,4,6). The pathophysiology could be explained as due to a hypersensitivity response to different allergens (1,2,4,8,9). In fact, 50% of pediatric patients and up to 29-60% of the adult population have atopic symptoms, and a strong family history of atopy is also demonstrated (17-20). EE suggest that the esophagus could be immunologically active because of the presence of eosinophils in the mucosal layer that induce esophageal injury (2,20). As Amisen et al, noted an allergy study could help understand EE (21). All our patients were young males aged between 22 and 43 years. Forty-five percent of them were atopic, and in 36% of cases familial forms were present too.

Between 10% and 50% of adults with EE have a high peripheral eosinophil count (2,7), but it is not diagnostic and its correlation with disease activity is unknown (10). We found 3 cases with peripheral eosinophilia.

Besides an allergic etiology, some authors reported a relationship between gastroesophageal reflux disease and EE, but controversy continues at the present moment (6,22). Data regarding pH monitoring were reported in 91 adults, with gastroesophageal pathological reflux in 18% (10). In our series 2 (18%) patients showed pathological reflux in agreement with other authors (10,23).

A manometric study reported two hypotense lower esophageal sphincters but none of them associated with pathological reflux in the pH-metric study. The upper esophageal sphincter was normal in all patients. The esophageal body study showed 54% of motility disorders, greater than the percentage described by other authors (10). All motility disorders were hypomotility-like (ineffective peristalsis); even in the gastroesophageal reflux disease population, the prevalence of ineffective peristalsis is about 30% (24), lower than in our series.

At endoscopy, felinization, distal rings, narrow esophagus, longitudinal shearings, mucosal exudates or friability have been identified, but none of them are pathognomonic of EE (10,11). Most of the listed findings have been reported in our patients. Some studies describe a high-risk of complications during the endoscopic procedure in patients with EE, but do not report exact percentages. We had two mucosal tearings (in a mid-length stenosis and the gastroesophageal junction).

The main symptoms in adults with EE are dysphagia and recurrent food impaction episodes, but sometimes endoscopic findings do not explain these symptoms; moreover, Prasad et al. (25) described 10% of normal endoscopic examinations in patients with EE and food impaction episodes. In our study the number of dysphagia attacks was between 0 and 12 a month, and patients with more dysphagia episodes did not report a longer time of disease or more food impaction episodes. Moreover, the patient who needed more emergency endoscopies to resolve food impactions referred only two years of symptom’s disease, and the endoscopic examination was normal, but in the manometric study we found a hyperkinetic disorder, distal esophageal spasm-like. Of the three patients with short-term symptoms (one year), 2 showed mucosal features in the endoscopic examination and other had esophageal felinization; none of them required emergency endoscopy to resolve food impactions, and a manometric study was normal in all three cases.

In conclusion, although endoscopic findings are common in EE, they do not explain dysphagia and food impaction episodes in all cases. In our paper, the most frequent motility disorder found was ineffective peristalsis, but it was not possible to establish a relationship between this motor pattern and symptoms. Because of the low number of cases diagnosed with eosinophilic esophagitis, it is difficult to establish the natural history of this illness: we do not know if structural findings predispose to a motor esophageal disorder or otherwise the eosinophilic infiltration of the esophageal wall produces motor abnor-
malities that after a long time induce alterations in the caliber of the esophagus.

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REFERENCES


