



## ORIGINAL ARTICLE

# A comparison of medical versus surgical treatment in Barrett's oesophagus acid control<sup>☆</sup>



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### KEYWORDS

Barrett's oesophagus;  
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Nissen  
fundoplication;  
Acid control;  
Proton pump  
inhibitors;  
Gastroesophageal  
reflux disease

### Abstract

**Introduction:** Barrett's oesophagus (BE) is an oesophageal injury caused by gastroesophageal acid reflux. One of the main aims of treatment in BE is to achieve adequate acid reflux control.

**Objective:** To assess acid reflux control in patients with BE based on the therapy employed: medical or surgical.

**Methods:** A retrospective study was performed in patients with an endoscopic and histological diagnosis of BE. Medical therapy with proton pump inhibitors (PPI) was compared with surgical treatment (Nissen fundoplication). Epidemiological data and the results of pH monitoring (pH time <4, prolonged reflux >5 min, DeMeester score) were evaluated in each group. Treatment failure was defined as a pH lower than 4 for more than 5% of the recording time.

**Results:** A total of 128 patients with BE were included (75 PPI-treated and 53 surgically-treated patients). Patients included in the two comparison groups were homogeneous in terms of demographic characteristics. DeMeester scores, fraction of time pH <4 and the number of prolonged refluxes were significantly lower in patients with fundoplication versus those receiving PPIs ( $p < .001$ ). Treatment failure occurred in 29% of patients and was significantly higher in those receiving medical therapy (40% vs 13%;  $p < .001$ ).

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**PALABRAS CLAVE**

Esófago de Barrett;  
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ácido;  
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bomba de protones;  
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*Conclusions:* Treatment results were significantly worse with medical treatment than with anti-reflux surgery and should be optimised to improve acid reflux control in BE. Additional evidence is needed to fully elucidate the utility of PPI in this disease.

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### Estudio comparativo del tratamiento médico frente al quirúrgico en el control ácido del esófago de Barrett

#### Resumen

*Introducción:* El esófago de Barrett (EB) es una lesión esofágica ocasionada mayoritariamente por reflujo gastroesofágico ácido. El control del reflujo ácido es uno de los principales objetivos del tratamiento de esta patología.

*Objetivo:* Evaluar en nuestra área de salud el grado de control del reflujo ácido en los pacientes con EB en función del tratamiento de mantenimiento recibido, médico o quirúrgico.

*Métodos:* Estudio retrospectivo de pacientes con diagnóstico endoscópico e histológico de EB. Un grupo de pacientes recibió tratamiento médico con inhibidores de la bomba de protones (IBP) y otro grupo fue sometido a intervención quirúrgica (funduplicatura de Nissen). Se compararon datos epidemiológicos y resultados de pHmetría (tiempo de pH < 4, reflujo prolongado > 5 min, puntuación de DeMeester) de cada grupo. La pH-metría se realizó con IBP en el grupo de tratamiento médico y en el grupo de cirugía sin consumo de antisecretores ácidos. Se definió fracaso del tratamiento como un pH < 4 total superior al 5%.

*Resultados:* Fueron incluidos 128 pacientes con EB (tratamiento médico 75, tratamiento quirúrgico 53). Ambas cohortes eran homogéneas respecto a sus características demográficas. Las puntuaciones de DeMeester, fracción de tiempo de pH < 4 y cantidad de reflujo prolongado fueron significativamente inferiores en los pacientes con funduplicatura frente a los que recibían IBP ( $p < 0,001$ ). De forma global se apreció un fracaso de tratamiento en el 29% de los pacientes, que fue significativamente mayor en el grupo de tratamiento médico (40% vs 13%;  $p < 0,001$ ).

*Conclusiones:* El grado de control del reflujo ácido gastroesofágico es subóptimo en un elevado porcentaje de pacientes con EB. El tratamiento médico ofrece resultados inferiores a la cirugía antirreflujo y se debería intentar optimizar sus resultados.

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## Introduction

Barrett's oesophagus (BO) is characterised by replacement of the oesophageal squamous epithelium by metaplastic columnar epithelium. BO, an oesophageal injury caused by severe, chronic gastro-oesophageal reflux,<sup>1</sup> is the primary risk factor for developing oesophageal adenocarcinoma (OAC), with a 30- to 50-fold increased risk compared to the general population.<sup>2</sup> Nevertheless, only a small proportion of patients with BO develop OAC; annual incidence is estimated at 0.27%, or 0.61% for a combined risk of high-grade dysplasia and OAC.<sup>3,4</sup> For this reason, screening and surveillance programmes for high-risk patients have been implemented in developed countries.<sup>5</sup> Some studies have reported a considerable increase in the incidence of OAC over the past 30 years in countries such as the USA.<sup>6</sup> This is evidence of both shortcomings in existing screening programmes and the existence of other external factors, apart from BO, involved in the carcinogenic process. Studies have shown obesity and an unhealthy diet to be independent risk factors for OAC, even in the absence of gastro-oesophageal reflux disease (GORD).<sup>7</sup>

Strategies for treating gastro-oesophageal reflux include medical therapy, to inhibit gastric acid secretion, and surgery. Both have been shown to initially improve symptoms, reduce the acid reflux that damages the oesophageal mucosa, and halt the carcinogenic process.<sup>4,8,9</sup> Among the drugs available to treat BO, proton pump inhibitors (PPI) have been shown to be the most effective in reducing gastric acid production, controlling GORD symptoms, and increasing mucosal healing in patients with oesophagitis. Acid suppressants are not usually entirely successful in controlling either symptoms or acid secretion, above all in long-segment BO (>3 cm), which is more refractory to conventional dosing regimens.<sup>10</sup> Evidence suggests that control of symptoms is not always associated with control of acid reflux measured by pH monitoring.<sup>11</sup> Some clinical trials have reported a lower incidence of dysplasia and cancer in patients with BO undergoing Nissen fundoplication compared with those receiving medical treatment, although these preliminary findings were not confirmed in later, more methodologically sound studies.<sup>12,13</sup>

BO is the primary risk factor for OAC, and clinical symptoms do not reflect pH acidity levels, therefore, controlling

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