

Gastroenterología y Hepatología



REVIEW



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KEYWORDS

Digestive system; Surgery; Complications; Gastrointestinal endoscopy; Morbid obesity **Abstract** Obesity is a highly prevalent disease worldwide, and one in which gastroenterologists can play an important role. Some digestive diseases are more common in obese patients, and preoperative evaluation may be required in some cases. Additionally, bariatric surgery can lead to digestive complications in the short and long term that require intervention, and endoscopic treatment can be an important factor in weight loss. The aim of this review is to highlight the role of the gastroenterologist in the management of obese patients who are either scheduled for or have undergone surgical or endoscopic treatment for obesity. © 2016 Elsevier España, S.L.U., AEEH and AEG. All rights reserved.

PALABRAS CLAVE

Aparato digestivo; Cirugía; Complicaciones; Endoscopia gastrointestinal; Obesidad mórbida

Papel del digestólogo en el manejo del paciente obeso

Resumen La obesidad es una enfermedad con una elevada prevalencia a nivel mundial en la que el digestólogo puede tener una labour importante. Por una parte, algunas enfermedades digestivas son más frecuentes en este grupo de pacientes y es posible que sea necesario evaluarlos antes de la cirugía. Por otra, la cirugía bariátrica puede presentar complicaciones digestivas a corto y largo plazo en las que sea imprescindible nuestra actuación. Además, no podemos olvidar el papel que el tratamiento endoscópico puede tener en la pérdida de peso. El objetivo de la presente revisión es destacar el papel del digestólogo en el manejo del paciente obeso candidato y/o sometido a tratamiento quirúrgico o endoscópico de la obesidad. © 2016 Elsevier España, S.L.U., AEEH y AEG. Todos los derechos reservados.

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Introduction

Obesity is a disease associated with a lower life expectancy. A worldwide prevalence of more than 700 million patients was estimated for 2015; thus it is considered a pandemic. Lifestyle changes are the mainstay of its treatment; however, long-term results are disheartening. To date, only bariatric surgery has achieved suitable, lasting weight loss.¹ Therefore, demand for this therapeutic option is increasing in a large number of countries.² In this context, the gastroenterologist plays an increasingly important role in assessment of gastrointestinal diseases associated with obesity, in endoscopic treatment of obesity in itself and in initial management of certain adverse effects deriving from surgery.

The obese patient with gastrointestinal symptoms

The obese patient with gastrointestinal symptoms requires clinical management similar to that of any other patient; however, some disorders, such as gastro-oesophageal reflux disease (GORD), disorders deriving from gallstones, functional abnormalities, non-alcoholic steatohepatitis (NASH) and colorectal cancer (CRC) require a special approach.

Many factors that promote gastro-oesophageal reflux in obesity have been reported. Multiple studies have shown that obese people are at higher risk of GORD and its complications, such as peptic oesophagitis, Barrett's oesophagus and oesophageal adenocarcinoma.3-5 The factors involved include: (a) visceral fat, with a significant association between oesophagitis and visceral fat measured by computed tomography⁶; (b) increased intra-abdominal pressure, which is associated, on the one hand, with an increased gastro-oesophageal pressure gradient⁷ (thus promoting disruption at the gastro-oesophageal junction and a hiatal hernia) and, on the other hand, a higher pressure gradient through the lower oesophageal sphincter, all directly related to body mass index (BMI) and waist circumference⁸⁻¹⁰; (c) decreased anti-inflammatory factors (such as adiponectin, tumour necrosis factor alpha and interleukin 6) and increased pro-inflammatory factors (such as leptin), which may promote the development of cancer at the gastro-oesophageal junction^{11,12}; (d) exposure to oestrogens produced by adipose tissue; (e) dietary habits, such as diets that are predominantly high in calories, fats and simple carbohydrates and low in fibre¹³; (f) use of proton-pump inhibitors (PPIs), which cause an increase in non-acid reflux episodes in obese patients¹⁴; and (g) Helicobacter pylori (Hp) infection, although its role in GORD is controversial.^{15,16} The therapeutic approach to GORD in the obese patient does not differ from that of the general population, although weight loss, as well as hygienic and dietary recommendations, do take on a particular importance. Among drug treatment options, PPIs are the drugs of choice.17

Obesity also promotes the formation of *gallstones*, thereby increasing the incidence of events (such as biliary colic, cholangitis and cholecystitis) that will require a cholecystectomy^{18,19} and sometimes endoscopic retrograde

cholangiopancreatography (ERCP) to treat possible choledocholithiasis. In this context, obesity has been associated with an increased risk of post-ERCP pancreatitis, probably due to excess subcutaneous adipose tissue.²⁰ Factors such as abnormal lipid metabolism help to form conglomerates of crystals of cholesterol monohydrate, mucin, calcium bilirubinate and proteins.²¹ High-calorie, low-fibre diets; sedentary lifestyle; and metabolic syndrome also increase the risk of gallstones,²² primarily due to secretion of bile supersaturated with cholesterol.²³ In addition, rapid weight loss (>1.5 kg/week) secondary to very low-calorie diets or bariatric surgery also promotes the development of gallstones. In fact, when weight loss exceeds 25% of the original weight, the likelihood of developing gallstones increases to 48%. Consequently, some authors have proposed preventive treatment with ursodeoxycholic acid.²⁴

Gallstones are the main aetiological factor for acute pancreatitis (up to 40% of cases). Consequently, the likelihood of developing acute pancreatitis is higher in obese people.²⁵ In addition, obesity acts as an independent risk factor for seriousness of pancreatitis. Different hypotheses have been formulated as to its pathogenesis: a chronic mild pro-inflammatory situation in the obese patient²⁶; increased peripancreatic fat, which may promote the onset of necrosis; slight deterioration of the microcirculation; and decreased respiratory capacity, which may lead to a higher likelihood of hypoxaemia.²⁷

Studies linking obesity to gastrointestinal *functional dis*orders, such as functional dyspepsia and irritable bowel syndrome, have shown contradictory results, but have agreed that diarrhoea and pain in the upper half of the abdomen are more prevalent in obese patients.^{28,29} One hypothesis for pathogenesis linking diarrhoea to obesity is an increase in simple sugars in the diet, which may promote the development of osmotic diarrhoea. However, to date, no concrete studies have been conducted on specific dietary variables.³⁰ In addition, using PPIs for GORD could promote bacterial overgrowth and related symptoms such as tympanites, abdominal pain and diarrhoea.³¹ More contradictory is a possible relationship between obesity and other common symptoms such as nausea, abdominal distension and rectal tenesmus.³²

NASH and metabolic syndrome, often present in the obese patient, increase the risk of developing several types of gastrointestinal cancer,³³ including CRC, predominantly in males.³⁴ An increased incidence of adenomatous polyps and CRC in people with NASH,³⁵ especially in the right colon, has been confirmed, thereby demonstrating the need for strict monitoring in screening programmes in this type of patient. In addition, colonoscopy may be more complex in these patients, especially women, with a longer-than-usual caecal intubation time.³⁶ At present, the exact pathogenic mechanism that predisposes obese patients to developing neoplasms is unknown, although a state of chronic inflammation, with an imbalance in cytokine production, could play a significant role.³⁷ Low blood levels of adiponectin (an inhibitor of the growth of tumour cells in the colon) are associated with an increased risk of developing arteriosclerosis and NASH in patients with a high BMI or hypertriglyceridaemia, ^{38,39} as well as a higher risk of CRC.⁴⁰

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