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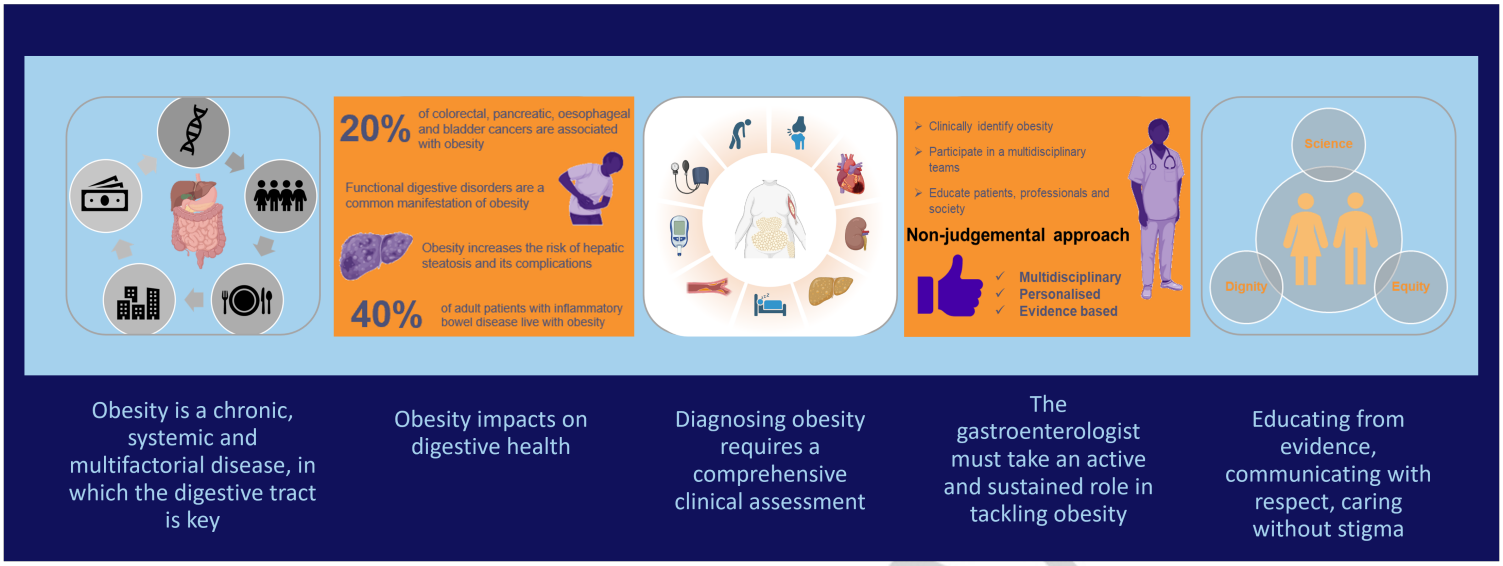

Obesity is a chronic, systemic and multifactorial disease, in which the digestive tract is key

Obesity impacts on digestive health

Diagnosing obesity requires a comprehensive clinical assessment

The gastroenterologist must take an active and sustained role in tackling obesity

Educating from evidence, communicating with respect, caring without stigma

Surname, et al.

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Digestive disease in individuals living with obesity: beyond weight loss

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Keywords: Obesity. Digestive diseases. Metabolic dysfunction. Health Inequities. SLD. MASLD. MetALD. Endoscopic sleeve gastroplasty.

List of abbreviations

ASGE – American Society for Gastrointestinal Endoscopy

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BMI – Body Mass Index

DXA – Dual-energy X-ray Absorptiometry

ESG – Endoscopic Sleeve Gastroplasty

EASO – European Association for the Study of Obesity

GERD – Gastroesophageal Reflux Disease

GIRO – Guía Española del Manejo Integral y Multidisciplinar de la Obesidad

GLP-1 – Glucagon-Like Peptide-1

HCC – Hepatocellular Carcinoma

IBD – Inflammatory Bowel Disease

MASLD – Metabolic Dysfunction-Associated Steatotic Liver Disease

MASH – Metabolic Dysfunction-Associated Steatohepatitis

MetALD – Metabolic Dysfunction-Associated Steatotic Liver Disease with Alcohol Consumption

NICE – National Institute for Health and Care Excellence

SIRS – Systemic Inflammatory Response Syndrome

T2DM – Type 2 Diabetes Mellitus

UEG – United European Gastroenterology

WHO – World Health Organization

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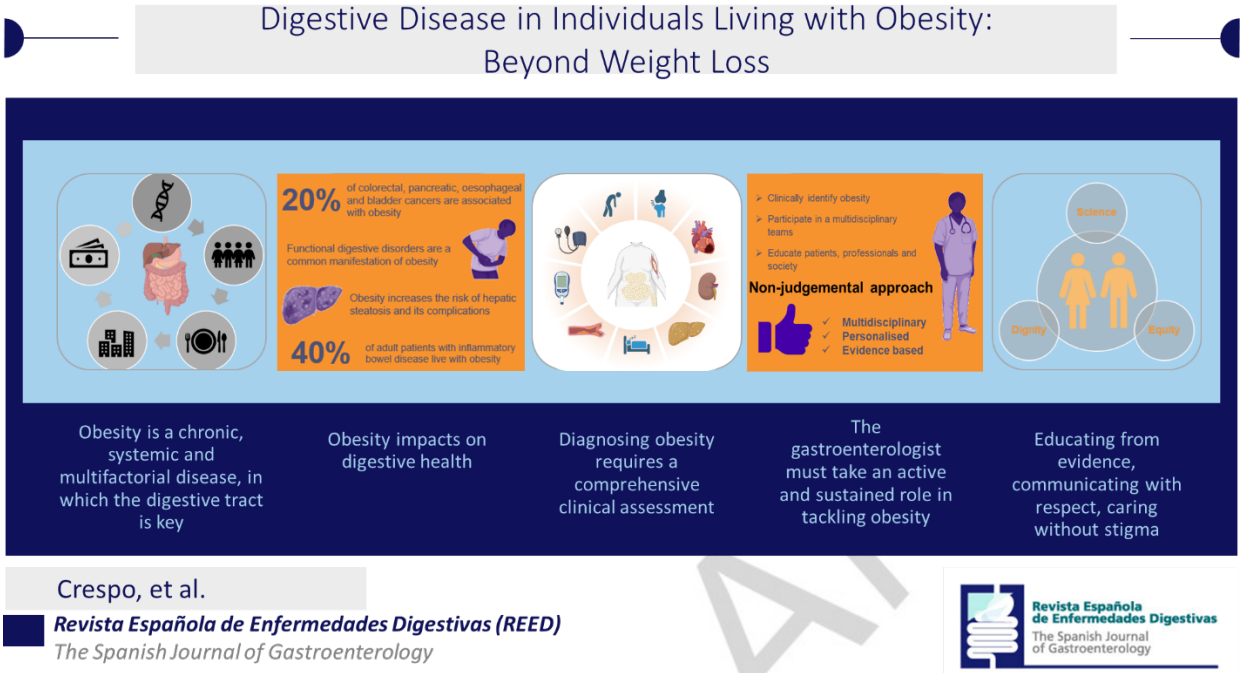
Visual abstract



ABSTRACT

Obesity is a chronic, progressive, and systemic disease that profoundly reshapes the landscape of digestive health. Beyond excess adiposity, it constitutes a multifactorial condition driven by complex interactions among genetic, metabolic, microbiological, and social determinants. This narrative review delineates the extensive and heterogeneous impact of obesity across the digestive tract, with a particular emphasis on metabolic dysfunction-associated steatotic liver disease, now the most prevalent chronic liver condition globally and a leading indication for liver transplantation. Obesity also modulates the pathophysiology, severity, and prognosis of functional gastrointestinal disorders, biliopancreatic diseases, inflammatory bowel disease, and digestive cancers, through mechanisms involving visceral adiposity, low-grade systemic inflammation, gut–brain axis disruption, and intestinal dysbiosis. The review highlights the emerging role of endoscopic bariatric therapies —especially endoscopic sleeve gastroplasty— as minimally invasive interventions that modulate enteroendocrine signaling, improve liver histology, and achieve sustained metabolic benefits. Beyond

the clinical realm, the article addresses the ethical imperative of destigmatizing obesity, the impact of food insecurity and health inequities on digestive outcomes, and the critical need for a multidisciplinary, patient-centered, and precision-based approach to digestive care. Gastroenterologists are called to transcend traditional boundaries, assuming leadership in prevention, early detection, and integrated management of obesity and its digestive sequelae. In doing so, digestive medicine becomes not only a responder to the consequences of obesity, but an active agent in transforming one of the most urgent public health challenges of the 21st century.

INTRODUCTION

Obesity is currently regarded as a chronic, progressive, multifactorial, and systemic disease, with clinical, legal, and social implications (1). In recent years, it has undergone a profound conceptual revision that has reshaped both its clinical definition and healthcare approach. According to the recent work of a global Commission on Obesity published in The Lancet Diabetes & Endocrinology, obesity is defined as an excess of adiposity, with or without abnormal distribution or function of adipose tissue. This definition transcends the BMI, as BMI does not differentiate between fat and lean mass, nor does it reflect adipose tissue distribution or its metabolic function. (2). These novel concepts have yet to be formally incorporated into the Spanish GIRO Guideline or the European Association for the Study of Obesity (EASO) guideline.

Globally, over 890 million adults currently live with obesity, a figure projected to surpass 1.5 billion by 2035 if current trends are not reversed (3, 4). In Spain, the data are equally alarming: 37.8% of adults are overweight and 16.5% are obese, with particularly concerning upward trends among children, where projections estimate that one in two children will have excess weight by 2035 (5).

Beyond the growing number of affected individuals, obesity imposes a significant burden of disease. Over 200 associated pathologies have been described, grouped into four major categories: cardiometabolic disorders, mechanical diseases, psychosocial conditions, and obesity-related cancers (2, 6). Obesity is not solely the outcome of

individual choices but is profoundly shaped by social determinants of health, including educational level, socioeconomic status, access to fresh and healthy foods, availability of safe spaces for physical activity, and the quality of healthcare services. These factors act as structural barriers to effective prevention, early diagnosis, and treatment of obesity (7).

From the perspective of the digestive system, the impact of obesity is especially relevant. First and foremost is metabolic-associated steatotic liver disease (MASLD), a condition that affects approximately 38% of the global population and up to 90% of individuals with obesity (8). Additionally, obesity increases the risk of digestive cancers (9).

Against this backdrop, the role of the gastroenterologist extends beyond the management of digestive complications. It is not only about addressing the gastrointestinal consequences of obesity, but also about adopting an expanded, proactive, and cross-disciplinary clinical role—one capable of early disease detection, intervention in underlying mechanisms, and contribution to prevention from an integrated perspective. (10).

In this context, the present narrative review aims to provide digestive specialists with an updated, rigorous, and empathetic understanding of the clinical, pathophysiological, therapeutic strategies, and social impact of obesity within their field. Additionally, it includes a practical decalogue (Table 1), as well as a graphical summary synthesizing the implicated mechanisms, their clinical consequences, and intervention opportunities within the specialty (Figure 1).

OBESITY, GASTROESOPHAGEAL REFLUX DISEASE, AND FUNCTIONAL DIGESTIVE DISORDERS

Obesity is significantly associated with a broad spectrum of functional gastrointestinal disorders, whose pathophysiology involves complex mechanisms such as increased intra-abdominal pressure, gastrointestinal motility dysfunction, disruption of digestive hormonal axes, and low-grade systemic inflammation. This relationship is particularly

evident in gastroesophageal reflux disease (GERD), a condition with greater prevalence and severity among individuals living with obesity. The accumulation of visceral fat increases intra-abdominal pressure and promotes transient relaxation of the lower esophageal sphincter, facilitating acid and non-acid reflux into the distal esophagus. Moreover, a higher prevalence of hiatal hernia and esophageal dysmotility has been observed in these patients (11-14). This esophageal dysfunction not only exacerbates typical GERD symptoms such as heartburn and regurgitation but is also associated with an increased risk of erosive esophagitis, Barrett's esophagus, and gastroesophageal junction adenocarcinoma. Clinical management requires an individualized approach, in which even modest weight loss can improve both functional and clinical parameters, such as lower esophageal sphincter pressure and esophageal acid exposure time (15).

Beyond the esophagus, other functional gastrointestinal disorders such as functional dyspepsia, gastroparesis, and irritable bowel syndrome (IBS) are significantly linked to obesity. Functional dyspepsia—particularly the postprandial distress subtype—has been associated with central obesity, likely mediated by impaired gastric accommodation, delayed gastric emptying, or antral hypersensitivity (16). Motor disturbances such as gastroparesis, especially prevalent in patients with obesity and T2DM, may lead to persistent symptoms and nutritional compromise (17). In the case of IBS, multiple studies have shown a higher prevalence among individuals with obesity, involving mechanisms such as visceral hypersensitivity, intestinal dysbiosis, accelerated colonic transit, chronic inflammation, and gut-brain axis dysfunction (18).

Intestinal dysbiosis contributes to both the onset and perpetuation of functional gastrointestinal symptoms, acting as a central pathophysiological axis linking obesity and digestive dysfunction (19). Individuals with obesity typically exhibit lower bacterial diversity and an imbalanced ratio of Firmicutes to Bacteroidetes, which is associated with enhanced energy harvest from food, altered intestinal transit, and immunometabolic dysfunction. This dysbiosis has been proposed as a therapeutic target via probiotics, prebiotics, or personalized nutritional interventions (20).

The diagnosis and treatment of these functional disorders are often complicated by symptom overlap with other prevalent gastrointestinal diseases, limited access to

specific diagnostic tests, and the frequent coexistence of anxiety-depressive symptoms. The latter is particularly relevant in the context of weight-related stigma, which increases psychological burden, alters symptom perception, and may limit access to or adherence to therapeutic interventions (21).

OBESITY AND INFLAMMATORY BOWEL DISEASE

Although IBD has traditionally been associated with weight loss and malnutrition, current obesity rates among patients with IBD range from 15% to 40%, depending on disease type, geographic region, and diagnostic criteria used (22). This epidemiological shift reflects not only the global nutritional transition but also the influence of shared environmental factors on gut biology and immune homeostasis, as well as the use of corticosteroids and biologics during prolonged phases of disease. From a pathophysiological standpoint, obesity induces a state of chronic low-grade inflammation mediated by proinflammatory adipokines such as TNF-α, IL-6, and leptin, which enhance activation of Th1 and Th17 immune pathways—key mediators in IBD pathogenesis—while reduced adiponectin levels dampen anti-inflammatory signaling. This systemic inflammation is accompanied by alterations in the gut microbiota (23), increased intestinal permeability, and epithelial barrier disruption, which together promote innate immune activation and perpetuate mucosal inflammation (24).

In this dysbiotic and immunoactivated environment, there is a breakdown in oral tolerance, which, along with the expansion of visceral adipose tissue, fosters persistently proinflammatory milieu. In Crohn's disease, in particular, mesenteric fat infiltration (“creeping fat”) is a morphological manifestation of this interaction and is associated with an increased risk of intestinal strictures and postoperative recurrence, suggesting that adipose tissue not only reflects but amplifies intestinal inflammation (25).

Clinically, patients with both IBD and obesity tend to present with more refractory disease phenotypes, reduced responsiveness to biologic therapies, increased need for therapeutic intensification, earlier loss of response, and higher hospitalization rates (26). Moreover, altered pharmacokinetics of drugs such as infliximab and adalimumab

in patients with elevated BMI may necessitate dose adjustments and closer therapeutic monitoring (27). Obesity also increases the technical complexity and risk of surgical procedures, with a higher incidence of surgical site infections, fistulas, thrombotic events, and conversions to open surgery. In this context, elevated BMI has been identified as an independent predictor of postoperative complications in colectomies and ileoanal anastomoses (28, 29). Nutritional assessment, prescription of tailored physical activity, monitoring of body composition beyond BMI, and early intervention strategies should be incorporated into routine IBD management (30).

OBESITY AND BILIOPANCREATIC DISEASE

Cholelithiasis is significantly more frequent among individuals living with obesity, with a two- to three-fold higher risk compared to the general population. This is attributed to increased cholesterol saturation in bile, reduced gallbladder motility, and heightened mucin secretion that facilitates nucleation and crystal growth (31). Intestinal dysbiosis and alterations in the enterohepatic bile acid cycle also play an important role in lithogenesis (32). In the context of bariatric surgery the risk of gallstone formation increases. This justifies the prophylactic use of ursodeoxycholic acid in selected patients, although systematic prophylactic cholecystectomy is not recommended (33). Acute cholecystitis and other biliary complications carry higher surgical risk in patients with obesity, requiring individualized approaches that account for altered anatomy and metabolic comorbidities (34).

Obesity is a well-established risk factor for acute pancreatitis, increasing both its incidence and clinical severity. This includes a higher risk of pancreatic necrosis, local complications, systemic inflammatory response syndrome (SIRS), multiorgan failure, and mortality (35-39). This increased severity is related to the activation of lipases in peripancreatic adipose tissue, which release free fatty acids with cytotoxic effects that amplify the inflammatory response.

Obesity may also contribute to the pathogenesis of chronic pancreatitis, whether as an evolution of acute episodes or through metabolic and immunological mechanisms not yet fully elucidated. It is more prevalent among young males with central adiposity and

metabolic syndrome (38). Exocrine pancreatic insufficiency is more common in this context and contributes to steatorrhea, micronutrient malabsorption, and secondary malnutrition, all of which have significant clinical implications (40, 41). Histologically, pancreatic steatosis has been documented in autopsies of individuals with obesity, suggesting increased susceptibility to chronic injury (42). This fat infiltration, along with imaging findings such as glandular atrophy, ductal dilation, and signs of chronic pancreatitis, is more frequent in this population and should be considered in the differential diagnosis of chronic abdominal pain and malabsorption (43, 44).

OBESITY AND LIVER DISEASE

Liver disease in individuals living with obesity primarily manifests as MASLD, currently the most prevalent form of chronic liver disease worldwide, with an estimated global adult prevalence of nearly 38% and over 85% among those with obesity (3, 45). Approximately one in five individuals with MASLD progresses to steatohepatitis, a more severe inflammatory phenotype that may evolve into cirrhosis and HCC (46).

In overweight or obese individuals, such progression is even more common: it is estimated that about 20% present with significant fibrosis, and up to 7% develop advanced fibrosis (47). This high burden of liver disease justifies the implementation of proactive screening strategies in populations with obesity, even in the absence of overt biochemical abnormalities. MASLD is now the second leading cause of end-stage liver disease and primary liver cancer in adults, and the most common indication for liver transplantation among HCC patients on waiting lists in the United States (48, 49).

Importantly, the clinical impact of MASLD extends beyond liver-specific complications. It plays a major role in systemic morbidity and mortality by contributing to cardiovascular, metabolic, neoplastic, and extrahepatic diseases (50). Therapeutic management is based primarily on lifestyle modification, with progressive weight loss being the most influential factor in histological regression of steatohepatitis and fibrosis. However, achieving sustained weight loss remains particularly challenging for individuals with severe obesity.

Pharmacologically, resmetirom is currently the only drug approved by the FDA for use in metabolic steatohepatitis with significant fibrosis (F2-F3) (51). In addition, GLP-1 receptor agonists have shown promising results in improving hepatic fat content, lipid and glycemic profiles, and regression of histologic lesions, and are recommended by national and international guidelines for patients with MASLD and obesity with or without T2DM (52). New agents under investigation, such as dual GLP-1/GIP and triple GLP-1/GIP/glucagon receptor agonists, are being evaluated for their potential to induce fibrosis regression and alter the disease course (53, 54).

At the population level, the burden of liver disease is not equitably distributed; nutritional inequity and socioeconomic vulnerability exacerbate the progression of MASLD by limiting access to healthy food, impeding therapeutic adherence, and restricting access to advanced diagnostic tools. This highlights the urgent need to incorporate a social dimension into health planning (55).

An increasing proportion of individuals with obesity also consume alcohol at moderate levels, not reaching criteria for classical alcohol-related liver disease, but sufficient to accelerate liver damage progression. This subgroup, termed MetALD (metabolic dysfunction-associated steatotic liver disease with alcohol consumption), represents a mixed phenotype of clinical interest, as the synergy between metabolic and ethanol-induced injury leads to accelerated progression toward advanced fibrosis and increased HCC risk. This justifies its identification and specific monitoring within the broader spectrum of steatotic liver disease (56). Even low levels of alcohol consumption can be highly detrimental to liver health, increasing the risk of advanced fibrosis up to fivefold (57).

Obesity also significantly affects both the indication for and outcomes after liver transplantation. Patients with class III obesity (BMI ≥ 40) have higher waitlist mortality, more postoperative complications, increased readmission rates, and reduced overall survival, due to both intraoperative technical difficulties and the persistence of a metabolically adverse environment post-transplantation (58-60). Moreover, post-transplant metabolic syndrome is associated with a high probability of MASLD recurrence, *de novo* diabetes, dyslipidemia, and increased cardiovascular risk,

necessitating pre-transplant risk stratification and long-term metabolic optimization programs (61).

OBESITY AND DIGESTIVE CANCER

Obesity is a well-established risk factor for the development of multiple neoplasms of the digestive system, contributing directly or indirectly to over 20% of all gastrointestinal cancers in the general population. This association includes esophageal, gastric, colorectal, pancreatic, and hepatocellular tumors, with a linear risk gradient that increases with BMI (62).

From a pathophysiological perspective, obesity fosters a proinflammatory tumor microenvironment characterized by dysfunctional adipose tissue expansion, local hypoxia, sustained release of adipokines (leptin, resistin), proinflammatory cytokines (TNF-α, IL-6), hyperinsulinemia, elevated insulin-like growth factor 1 (IGF-1), oxidative stress, and activation of oncogenic signaling pathways such as mTOR, JAK/STAT, and NF-κB. These processes not only promote carcinogenesis but also influence tumor progression and therapeutic response (63). In addition, obesity-associated gut dysbiosis contributes to the development of a protumorigenic microenvironment through the production of proinflammatory metabolites, disruption of the epithelial barrier, and stimulation of innate immune pathways (64).

In the esophagus, obesity significantly increases the risk of distal adenocarcinoma, partly mediated by the higher prevalence and severity of GERD and Barrett's esophagus in these patients. This association persists independently of acid reflux (65).

The risk of colorectal cancer is also elevated in individuals with obesity, especially among men, with a 30% increase in risk for every 5 kg/m² rise in BMI. This is attributed to a combination of dysbiosis, insulin resistance, and impaired intestinal immunosurveillance (66). In this type of cancer, abdominal obesity has been linked to a higher density of cancer stem cells, chemotherapy resistance, and specific somatic mutations involving the Wnt/β-catenin and PI3K/AKT pathways.

In the liver, MASLD and its advanced form, steatohepatitis, are independent risk factors for HCC, even in the absence of cirrhosis, with growing incidence rates attributable to the global rise in obesity (67).

Similarly, in the pancreas, an increased risk of ductal adenocarcinoma has been reported in individuals with obesity, driven by mechanisms such as chronic inflammation, dysregulated lipid metabolism, and immune dysfunction (68).

Beyond cancer incidence, obesity also negatively impacts prognosis. Digestive tumors in patients with obesity are associated with poorer responses to immunotherapy, attributed to innate immune dysregulation, $CD8^+$ T-cell exhaustion, and overexpression of PD-1/PD-L1 in the obesity-related tumor microenvironment (69). Obesity increases surgical difficulty and treatment-related toxicity, limits access to clinical trials, and reduces overall survival (50).

Ultimately, obesity not only acts as a tumorigenic risk factor but also modulates disease progression, therapeutic response, and equity in oncologic care. This oncologic vulnerability is further compounded by social determinants of health. Nutritional inequity, food insecurity, and low educational attainment amplify cancer risk by limiting access to healthy diets, screening programs, and precision therapies (55).

The digestive physician, therefore, must take a proactive role in the early identification of oncological risk associated with obesity. This implies implementing primary prevention strategies as well as participation in specific screening programmes, particularly in colorectal and oesophageal cancer. The integration of precision medicine tools allows personalisation of clinical decisions based on metabolic risk phenotypes and tumour prognosis. In short, obesity not only acts as a tumor risk factor, but also as a modulator of progression, response and equity in cancer care.

OBESITY AND DIGESTIVE ENDOSCOPY

Digestive endoscopy in individuals living with obesity requires a tailored approach that addresses both logistical and technical challenges as well as emerging therapeutic opportunities in this patient population. From a procedural standpoint, obesity affects

multiple aspects of endoscopic practice: it increases anesthetic risk and requires advanced monitoring during sedation, presents positional difficulties, reduces the quality of colonic preparation, and may interfere with access and visualization during both upper endoscopy and colonoscopy—particularly in cases of massive visceral adiposity or previous abdominal surgery (70-73).

Despite these limitations, endoscopy remains an irreplaceable tool for the structural and functional diagnosis of digestive diseases that disproportionately affect individuals with obesity. However, the most relevant transformation has been the development and consolidation of metabolic endoscopy, a minimally invasive strategy that targets key pathophysiological mechanisms of obesity such as satiety regulation, gastric emptying, enteroendocrine hormone secretion, and insulin sensitivity.

Among the most established techniques is endoscopic sleeve gastroplasty (ESG), which has proven to be safe, reproducible, and effective not only in achieving sustained weight loss, but also in improving cardiometabolic, hepatic, and quality-of-life parameters. In the ESG-MASH clinical trial, ESG combined with lifestyle intervention led to greater improvement in liver fibrosis and remission of steatohepatitis in patients with MASLD (74) compared to the control group (75).

Beyond ESG, other techniques—such as POSE 2.0, MEGA, etc—have shown variable efficacy depending on clinical profiles. These options are particularly useful in patients with contraindications, high surgical risk, or reluctance to undergo bariatric surgery. Such alternatives should be considered within a stepwise therapeutic strategy that integrates clinical judgment, patient preferences, and available resources (74, 76-79).

The impact of these procedures extends beyond the liver. Studies have shown significant reductions in BMI, improved insulin sensitivity, normalized glycemic parameters, reduced liver volume, decreased triglycerides and blood pressure, and sustained improvements in health-related quality of life (77).

In comparative studies, ESG outcomes are comparable to or even exceed those of pharmacotherapies such as semaglutide, and remain favorable even when compared to tirzepatide in certain subgroups of patients with severe obesity and multiple metabolic comorbidities (80). In 2024, a new clinical guideline broadened the

indication for BETs, issuing a conditional recommendation—with low evidence quality—for patients with BMI >30 kg/m² without comorbidities and even those with BMI between 27 and 30 kg/m² when a relevant metabolic comorbidity is present (81). That same year, the UK's National Institute for Health and Care Excellence recognized ESG as a valid therapeutic option against obesity (82). The Spanish GIRO 2.0 Guideline recommends, with moderate strength and evidence, considering ESG in the following scenarios (10):

1. Patients with BMI 30–39.9 kg/m² in whom medical treatment fails to meet weight loss or maintenance goals.
2. Patients with suboptimal weight response following bariatric surgery where combined non-surgical therapies have proven inadequate.
3. Patients with BMI ≥50 kg/m² requiring preoperative weight loss to reduce surgical morbidity and mortality.

This expanding therapeutic role of the endoscopist requires specialized training in obesity pathophysiology, candidate selection, comorbidity management, and interdisciplinary collaboration with endocrinology, nutrition, and hepatology. Additionally, it necessitates adapting endoscopy units to accommodate longer procedures of greater technical complexity, as well as structured longitudinal follow-up.

In summary, digestive endoscopy—from its diagnostic dimension to its potential as metabolic endoscopy—is a strategic tool enabling gastroenterologists to assume a comprehensive role in the clinical management of individuals living with obesity.

OBESITY AND PUBLIC HEALTH

Obesity must be understood as a chronic, multifactorial disease whose roots and perpetuating mechanisms are profoundly shaped by structural, social, economic, and cultural factors. It is not merely the sum of individual choices, but a condition that develops and persists in obesogenic environments molded by the so-called social determinants of health. These determinants include educational attainment,

employment status, housing quality, access to healthcare services, food security, and the broader community environment, all of which critically influence individuals' ability to adopt healthy behaviors, access early diagnoses, and benefit from effective treatments (2). These factors operate at both the individual and population levels, determining opportunities for health and perpetuating inequalities. As with other non-communicable diseases, obesity is particularly prevalent in populations experiencing economic and educational disadvantage, which in turn undermines health-promoting behaviors and treatment adherence (7). Limited comprehension of medical or nutritional recommendations, combined with barriers to accessing healthy products, perpetuates cycles of disease and inequity.

Moreover, food insecurity is associated with higher consumption of ultraprocessed products, which are economically accessible and widely available in areas where fresh food is scarce or expensive (83). This vulnerability is further exacerbated by so-called financial toxicity: the direct and indirect economic burden of diagnostic tests, medical consultations, and innovative treatments, which contributes to poor adherence and denies the most vulnerable patients access to proven interventions (84).

In this context, obesity has reached epidemic proportions. This situation constitutes a public health crisis with both systemic and individual repercussions. At the systemic level, obesity increases healthcare expenditures, overburdens health systems, and generates significant economic losses (2). At the individual level, it leads to a higher burden of chronic diseases such as MASLD, type 2 diabetes, and cardiovascular disease, as well as other consequences including depression (55).

The emerging concept of "social nutrition" helps elucidate the interaction between structural determinants and personal support networks as key factors in both the etiology and management of obesity (85). This holistic perspective demands the active and sustained involvement of multiple stakeholders: legislators, healthcare professionals, the food industry, educators, community leaders, mental health experts, and religious representatives. Only through a systemic, intersectoral, and coordinated approach can we advance toward effective, equitable, and sustainable obesity control.

Addressing this challenge requires a robust ecosystem of scientific, technological, and educational innovation. The global obesity market mobilizes over $80 billion annually, which explains the dynamism in research on new therapeutic approaches (86). Since the 1970s, research has linked obesity to poverty, ethnicity, sleep disorders, lipid abnormalities, and even prenatal exposures (87). Sleep, for instance, has been associated with lipid markers and cardiovascular risk in specific ethnic groups (88).

There are also behavioral treatments rooted in neuroscience, such as the ORBIT model, designed to address psychological factors in pediatric and adolescent obesity (89). Current advances include GLP-1 receptor agonists (semaglutide), dual agonists (tirzepatide), functional foods, and pioneering gene therapy trials using adipocytes engineered to express CPT1AM, a key enzyme in lipid oxidation (90).

On the educational front, the proliferation of obesity-specific scientific journals and WHO initiatives—such as the 2021 Acceleration Plan—underscore the urgency of intervention through continuing medical education (91). Initiatives like Project Globe in Russia have shown that training healthcare professionals improves clinical outcomes (92). Moreover, the use of artificial intelligence in self-monitoring and educational tools has demonstrated benefits in metabolic patients (93).

In this ecosystem, the digestive specialist plays a strategic clinical role. Obesity is linked to highly prevalent and severe digestive diseases (94, 95). Coordination with endocrinology, bariatric surgery, and psychiatry is already routine, but the digestive role also extends to endoscopic interventions (44). Systematic involvement of gastroenterologists enhances diagnosis, therapeutic planning, and postoperative follow-up, including the use of tools such as hepatic ultrasound, elastography, or preoperative endoscopy (34).

However, this clinical commitment must also extend to the ethical domain. Obesity has traditionally been perceived as a personal failure, giving rise to a medical and societal stigma that undermines the well-being and care of affected individuals. Reconceptualizing health as “the ability to adapt and self-manage in the face of physical, emotional, and social challenges,” as proposed by Huber, helps to affirm the right to well-being and autonomy even in the presence of disease (96).

Weight stigma has been shown to diminish the quality of physician-patient communication, reduce treatment adherence, and decrease follow-up frequency (97). It also exerts deleterious effects on physical and mental health (98) and functions as a structural determinant of health inequity that must be actively eradicated through public policy and clinical practice (99). As the World Health Organization emphasizes, eliminating stigma is an essential condition for effective, humane, and inclusive obesity care (100).

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Statement of Generative AI and AI-assisted technologies in the writing process

During the preparation of this paper, the authors used ChatGPT [https://chat.openai.com/] and DeepL Translate [DeepL.com] for language editing porpoises. After using these tools, the authors reviewed and edited the content as necessary and take full responsibility for the content of the publication.

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Table 1: Ten principles for transformative digestive care in individuals living with obesity.

1. **Obesity is a chronic, complex, and multifactorial disease.** It should not be viewed as the result of failed personal choices. Rather, it is a chronic and relapsing condition caused by dynamic interactions among genetic, biological, social, environmental, and commercial factors. Over 890 million adults worldwide are affected, and projections estimate more than 1.02 billion by 2030. In Spain, over 22 million adults live with excess weight, and more than half the population is expected to have overweight or obesity by 2035.
2. **Diagnosing obesity requires comprehensive clinical assessment.** Body mass index (BMI) is a useful but insufficient tool. Proper diagnosis involves evaluating fat quantity and distribution, as well as the metabolic, organ-specific, and functional impact of adiposity. Waist circumference, waist-to-height ratio, and body composition assessment via bioimpedance or dual-energy X-ray absorptiometry (DXA) are all valuable techniques.
3. **Obesity increases the risk of hepatic steatosis and its complications.** Metabolic-associated steatotic liver disease (MASLD) is now the most common form of chronic liver disease. Approximately two-thirds of people with obesity develop MASLD, and one-third may progress to steatohepatitis with risk of advanced fibrosis, cirrhosis, hepatocellular carcinoma, and liver failure. In the U.S., MASLD is already the leading indication for liver transplantation. While obesity may affect post-transplant outcomes, it should not be viewed as a contraindication, but rather as an opportunity to design multidisciplinary approaches. The impact of MASLD extends beyond the liver, increasing cardiovascular risk and overall mortality.
4. **Obesity increases the risk of digestive cancers via systemic mechanisms.** Up to 20% of colorectal, pancreatic, esophageal, and gallbladder cancers are associated with obesity. This elevated risk is mediated by chronic low-grade inflammation, insulin resistance, hormonal and immune dysregulation—representing a network of interactions that transcend

individual lifestyle factors.

5. **Functional gastrointestinal disorders are frequent and potentially disabling manifestations of obesity.** GERD, dyspepsia, diarrhea, constipation, and functional abdominal pain are common in people living with obesity and may compromise physical and emotional well-being. These conditions are shaped by microbiota alterations, systemic inflammation, dietary environment, and gut–brain axis dysfunction.
6. **Obesity can worsen the clinical course of inflammatory bowel disease (IBD).** Up to 40% of adult IBD patients live with obesity. This coexistence is associated with higher surgical risk, increased hospitalization rates, greater postoperative recurrence, and lower therapeutic response. Recognizing obesity as a relevant comorbidity in IBD calls for a coordinated, complexity-adapted clinical approach.
7. **The digestive system plays a central role in the pathophysiology, prevention, and treatment of obesity.** The digestive tract regulates satiety, energy metabolism, and hormonal signaling. Incretin-based drugs, endoscopic sleeve gastroplasty, and metabolic surgery target these pathways, achieving 10–25% weight loss and preventing clinical complications. Digestive health promotion—through fiber-rich diets, regular mealtimes, and mindful chewing—also contribute to sustainable obesity prevention and management.
8. **Obesity care requires a rigorous, bias-free clinical approach.** As with any chronic disease, obesity must be treated without prejudice or stigma. Treatment should be individualized and multidisciplinary, combining nutritional, pharmacologic, psychological, endoscopic, or surgical strategies tailored to the patient's clinical profile. Therapeutic planning must be guided by evidence and aimed at functional improvement and quality of life, rather than shaped by personal, social, or non-clinical judgments.
9. **Gastroenterologists must assume an active and sustained role in obesity care.** Digestive specialists have a responsibility to clinically identify obesity, participate in its multidisciplinary management —including pharmacologic and endoscopic interventions— and contribute to the education of patients,

healthcare professionals, and society. Incorporating obesity care into gastroenterology is not optional: it is a clinical, ethical, and public health obligation.

10. **Educate based on evidence, communicate with respect, and care without stigma.** From clinical practice to public discourse, it is imperative to transform how we speak and act regarding obesity. Evidence-based training, non-stigmatizing language, and person-centered care are essential to building a healthcare model grounded in science, equity, and human dignity.

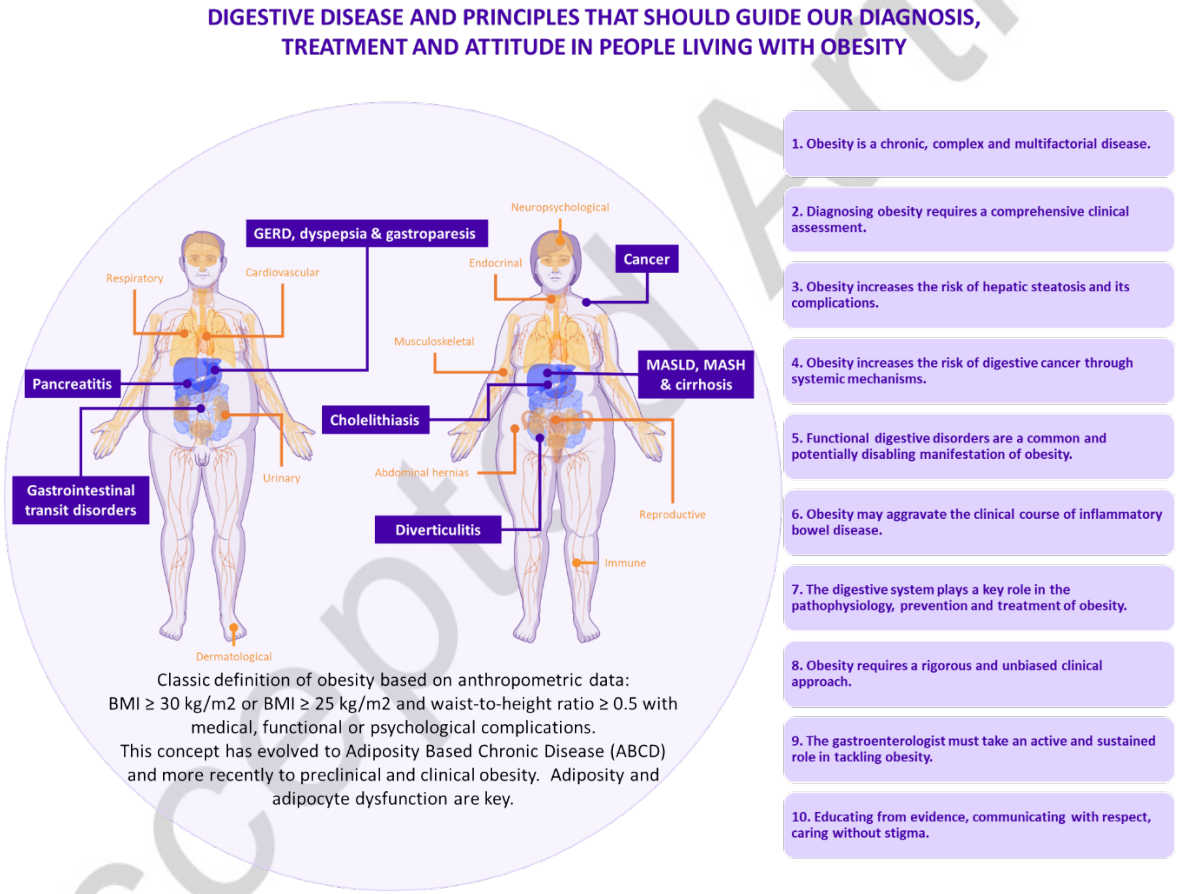

Figure 1: Digestive diseases and comorbities in patients living with obesity and guidelines for diagnosis, treatment and management. GERD: Gastroesophageal Reflux Disease; MASLD: Metabolic Dysfunction-Associated Steatotic Liver Disease; MASH: Metabolic Dysfunction-Associated Steatohepatitis; BMI: Body Mass Index. *Created in https://BioRender.com.*