Improvement of metabolic and cardiovascular outcomes after bariatric surgery: from bench to bedside

Mejoría en los resultados metabólicos y cardiovasculares después de la cirugía bariátrica: de la investigación básica a la práctica clínica

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Abstract

Obesity is a complex multifactorial metabolic disorder linked to a myriad of endocrine, cardiovascular, and musculoskeletal, oncologic and neuropsychiatric complications. A large epidemiologic boom has been observed in the epidemiologic trends of obesity in recent decades, paralleling those of cardiovascular disease (CVD). Indeed, the combination of obesity and CVD represents an essential problem for everyday clinical practice; which has prompted a surge in the development of treatment alternatives for obesity. Bariatric surgery (BS) remains a valuable tool in this armamentarium, in particular for complex cases with several comorbidities. BS has been observed to grant particularly valuable advantages, producing profound and durable improvements in metabolic and cardiovascular health, mediated by a multifaceted interplay of mechanisms extending beyond weight loss alone. Anatomical alterations to the gastrointestinal tract trigger rapid changes in enteroendocrine signaling and local and systemic inflammation, which together enhance insulin sensitivity, optimize lipid handling, reduce and restore autonomic balance, and promote beneficial hemodynamic changes. These pathophysiological adaptations translate into clinically meaningful outcomes, including high rates of type 2 diabetes remission, sustained improvements in dyslipidemia and hypertension, reduced incidence of major adverse cardiovascular events, and decreased all-cause and cardiovascular

mortality. In this article, we revise the mechanisms and epidemiology of the amelioration of metabolic and cardiovascular outcomes after BS.

Keywords: Bariatric surgery, obesity, cardiovascular disease, diabetes, dyslipidemia..

Resumen

La obesidad es un trastorno metabólico complejo y multifactorial, asociado a una gran variedad de complicaciones endocrinas, cardiovasculares, musculoesqueléticas, oncológicas y neuropsiquiátricas. En las últimas décadas se ha observado un marcado aumento en las tendencias epidemiológicas de la obesidad, paralelo al de la enfermedad cardiovascular (ECV). De hecho, la combinación de obesidad y ECV representa un problema fundamental en la práctica clínica diaria, lo que ha impulsado un auge en el desarrollo de alternativas terapéuticas para la obesidad. La cirugía bariátrica (CB) sigue siendo una herramienta valiosa dentro de este arsenal, especialmente en casos complejos con múltiples comorbilidades. Se ha observado que la CB ofrece ventajas particularmente importantes, produciendo mejoras profundas y duraderas en la salud metabólica y cardiovascular, mediadas por una interacción multifacética de mecanismos que van más allá de la simple pérdida de peso. Las modificaciones anatómicas del tracto gastrointestinal provocan cambios rápidos en la señalización enteroendocrina y en la inflamación local y sistémica, lo que en conjunto mejora la sensibilidad a la insulina, optimiza el manejo de los lípidos, reduce y restaura el equilibrio autonómico, y promueve cambios hemodinámicos beneficiosos. Estas adaptaciones fisiopatológicas se traducen en resultados clínicamente significativos, que incluyen altas tasas de remisión de la diabetes tipo 2, mejoras sostenidas en dislipidemia e hipertensión, reducción de la incidencia de eventos cardiovasculares adversos mayores, y disminución de la mortalidad total y cardiovascular. En este artículo revisamos los mecanismos y la epidemiología de la mejoría de los resultados metabólicos y cardiovasculares después de la CB.

Palabras clave: Cirugía bariátrica, obesidad, enfermedad cardiovascular, diabetes, dislipidemia.

Introduction

Obesity is a complex multifactorial metabolic disorder characterized by excess body fat, which heightens the risk for a broad series of endocrine, cardiovascular, and musculoskeletal, oncologic and neuropsychiatric complications; among many others. As a result, it is associated with lower quality of life and a shortened life expectancy[1]. Overweight and obesity rates have increase two-fold over the last 40 years, and at present, around one third of the global population are thought to be in one of these categories. This trend is ubiquitous across all ages, but is particularly marked in the elderly, and in women[2]. Indeed, globally, 1 billion adult men and 1.11 adult women are estimated to be overweight or obese[3]. Furthermore, obesity is linked to very high financial costs, due to disability and medical expenses; therefore underlining obesity as a veritable public health problem[4].

In parallel, cardiovascular disease (CVD) remains the main cause of mortality and morbidity worldwide, a trend that has persisted during the last thirty years[5]. Moreover, in the last three decades, CVD incidence has risen from 34.74 million 50 66.81 million, and mortality has increased from 12.33 million to 19.42 million; corresponding to increases of 92.3% and 57.5%, respectively[6]. CVD also represents a heavy burden of disease, with a substantial amount of disability-adjusted life years (DALYs) attributed to it. Projections from 2025 to 2050 indicate an expected 90% increase in prevalence, a 73,4% augmentation in crude mortality, and 54.7% increase in crude DALYs, with approximately 35.6 million CVD deaths in 2050[7].

In light of this panorama, the combination of obesity and CVD represents an essential problem for everyday clinical practice. In particular, there has been a surge in the development of treatment alternatives for obesity[8]. However, bariatric surgery (BS) remains a valuable tool in this armamentarium, in particular for complex cases with several comorbidities[9]. BS has been observed to grant particularly valuable advantages, such as improvement of the course of CVD[10]. In this article, we revise the mechanisms and epidemiology of the amelioration of metabolic and cardiovascular outcomes after BS.

BARIATRIC SURGERY: UNDERLYING CARDIOVASCULAR AND METABOLIC MECHANISMS

Bariatric surgery induces profound and sustained improvements in metabolic and cardiovascular health through a complex interplay of anatomical, hormonal, inflammatory, and neuroendocrine adaptations[11]. These effects extend beyond weight loss alone and often oc-

cur within days to weeks postoperatively, suggesting that mechanisms independent of adiposity reduction are also at play[12].

The primary driver of improved cardiovascular outcomes remains substantial and sustained weight loss, leading to decreased total and visceral adipose tissue. Visceral fat reduction alleviates ectopic lipid deposition in the liver, pancreas, and myocardium, thereby improving insulin sensitivity, reducing myocardial steatosis, and decreasing cardiac workload[13]. Adipose tissue remodeling after BS also reduces adipocyte hypertrophy and restores a more favorable adipokine profile, characterized by increased adiponectin and decreased leptin and resistin levels[14].

Alterations in gut anatomy can also profoundly affect enteroendocrine signaling after procedures such as Roux-en-Y gastric bypass and sleeve gastrectomy. The incretin effect is often potentiated: Enhanced secretion of glucagon-like peptide-1 (GLP-1) and peptide YY (PYY) improves postprandial insulin secretion, suppresses glucagon, and slows gastric emptying, contributing to rapid glycemic control[15]. Likewise, reduced ghrelin levels following fundus resection in SG attenuate appetite and may improve insulin sensitivity[16]. Fibroblast growth factor 19 (FGF19) and bile acid–mediated signaling via the farnesoid X receptor (FXR) and TGR5 pathways have also been observed to be increased after BS, further enhancing glucose metabolism and lipid handling[17]. These hormonal adaptations may contribute to why remission of type 2 diabetes mellitus (T2DM) frequently precedes significant weight loss in patients who have undergone BS[18].

Anti-inflammatory effects also appear to follow BS. Obesity is characterized by a chronic pro-inflammatory state mediated by increased release of interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF-α), and C-reactive protein (CRP)—among several other mediators—from dysfunctional adipose tissue[19]. BS reduces systemic inflammation through decreased adipose macrophage infiltration and improved gut microbiota composition[20]. This anti-inflammatory shift contributes to improved endothelial function, stabilization of atherosclerotic plaques, and reduced risk of cardiovascular events[21].

Lipid metabolism has also been highlighted to change significantly after BS. Postoperative changes in lipid handling include reduced hepatic very-low-density lipoprotein (VLDL) production, increased clearance of triglyceride-rich particles, and improved high-density lipoprotein (HDL) functionality[22]. Enhanced bile acid signaling also modulates cholesterol metabolism, contributing to favorable lipid profiles that reduce atherogenic risk[23].

After BS, several other important changes occur regarding hemodynamic and cardiac functional adaptations. Sustained weight loss decreases total blood volume and systemic vascular resistance, thereby reducing left ventricular (LV) afterload. Improvements in LV geometry, diastolic function, and myocardial strain have been observed as early as six months after surgery[24]. Additionally, reductions in obstructive sleep apnea and improved respiratory mechanics decrease pulmonary artery pressures and right ventricular strain[25].

Likewise, obesity-related sympathetic hyperactivity is attenuated post-BS, partly due to improved insulin sensitivity, reduced leptin resistance, and lower systemic inflammation. Enhanced parasympathetic tone supports better heart rate variability, lower resting heart rate, and improved blood pressure control[26]. Lastly, an emerging body of evidence links BS to favorable alterations in gut microbiota composition, with increased abundance of species associated with improved glucose and lipid metabolism. These microbial changes influence short-chain fatty acid production, bile acid metabolism, and systemic inflammation, further contributing to cardiovascular and metabolic benefits[27].

In summary, BS initiates a cascade of interrelated physiological changes that extend beyond caloric restriction and weight reduction. Hormonal, inflammatory, and neuroendocrine adaptations synergistically improve insulin sensitivity, lipid metabolism, vascular health, and cardiac function, providing a mechanistic basis for the robust reduction in metabolic disorders and cardiovascular morbidity observed after surgery[28].

METABOLIC AND CARDIOVASCULAR OUTCOMES AFTER BARIATRIC SURGERY: CLINICAL EVIDENCE

The clinical benefits of BS extend beyond weight reduction, encompassing substantial and sustained improvements in metabolic homeostasis and cardiovascular health. Evidence from randomized controlled trials (RCTs), prospective cohorts, and large registry-based studies consistently demonstrates reductions in the incidence and severity of obesity-related comorbidities, along with decreased all-cause and cardiovascular mortality[29].

Multiple RCTs, including the STAMPEDE trial, have shown that bariatric surgery is superior to intensive medical therapy for achieving T2DM remission, with remission rates of 30–80% depending on surgical type, baseline duration of diabetes, and β-cell reserve. Improvements in fasting glucose, HbA1c, and postprandial glycemia often occur within days to weeks postoperatively, preceding significant weight loss[30]. Long-term follow-up demonstrates that while some patients experience relapse, glycemic control remains significantly better than in non-surgical cohorts[31].

On the other hand, meta-analyses consistently demonstrate significant and sustained improvements in lipid profiles following BS. In a comprehensive analysis of 178 studies involving over 25,000 patients, Heffron et

al.[32] reported mean reductions at one year of –28.5 mg/dL in total cholesterol, –22.0 mg/dL in LDL cholesterol, and –61.6 mg/dL in triglycerides, alongside an increase in HDL cholesterol of +6.9 mg/dL. These changes are significantly greater than those observed in non-surgical controls1. Similarly, the landmark meta-analysis by Buchwald et al. demonstrated sustained benefits at two years, with mean changes of –33 mg/dL in total cholesterol, –29 mg/dL in LDL cholesterol, –80 mg/dL in triglycerides, and a notable rise in HDL cholesterol, particularly after Roux-en-Y gastric bypass and vertical banded gastroplasty[33]. These lipid improvements are associated with reduced atherogenic risk and contribute to the long-term cardiovascular benefits observed after BS[34].

Bariatric surgery also appears to lead to remission or improvement of hypertension in approximately 40–70% of patients. The GATEWAY trial demonstrated that surgical intervention significantly reduced antihypertensive medication requirements and improved 24-hour ambulatory blood pressure compared to medical therapy alone[35]. These effects are mediated by weight loss, reduced sympathetic activity, and improved vascular compliance[36].

Moreover, BS is associated with improvements in indicators of heart failure. In a systematic review and meta-analysis by Espaarham et al.[37], a significant amelioration of LV ejection fraction was observed after BS, with an average difference of 7.78%. In addition, the NYHA class notably decreased following BS, with an average difference of –0.40. Lastly large-scale observational studies, such as the Swedish Obese Subjects (SOS) study, report significant decreases in cardiovascular mortality. In this study, the unadjusted overall hazard ratio was 0.76 in the surgery group (P=0.04) in comparison with the control group. The hazard ratio adjusted for sex, age, and risk factors was 0.71 (P=0.01). The benefits persist for over two decades, even after partial weight regain, suggesting lasting metabolic and vascular adaptations[38].

Overall, the clinical evidence robustly supports BS as a powerful intervention for improving metabolic parameters, reducing cardiovascular risk factors, and preventing major adverse cardiovascular events. The combination of sustained weight loss and surgery-specific physiological effects provides a durable cardiometabolic benefit unmatched by lifestyle or pharmacological interventions alone[39].

Conclusions

Overall, it is evident that BS produces profound and durable improvements in metabolic and cardiovascular health, mediated by a multifaceted interplay of mechanisms extending beyond weight loss alone. Anatomical alterations to the gastrointestinal tract trigger rapid changes in enteroendocrine signaling and local and systemic inflammation, which together enhance insulin sensitivity, optimize lipid handling, reduce and restore autonomic balance, and promote beneficial hemodynamic changes. These pathophysiological adaptations translate into clinically meaningful outcomes, including high rates of type 2 diabetes remission, sustained improvements in dyslipidemia and hypertension, reduced incidence of major adverse cardiovascular events, and decreased all-cause and cardiovascular mortality.

Looking forward, the field is poised to expand its scope in several directions. Advances in surgical techniques, perioperative care, and patient selection algorithms promise to further improve safety and accessibility. Integration with emerging pharmacotherapies for obesity and diabetes may yield synergistic effects, potentially allowing tailored combination approaches. In parallel, mechanistic research—leveraging metabolomics, microbiome science, and precision imaging—will deepen understanding of the molecular pathways linking BS to cardiovascular and metabolic protection. In conclusion, BS represents both a therapeutic intervention and a powerful model for studying the interplay between the gut, metabolism, and cardiovascular health. As the evidence base grows and technology advances, a more personalized, mechanism-driven approach will likely emerge, maximizing patient benefit while minimizing risk.

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