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DYNAMICS OF IMMUNE AND INFLAMMATORY MARKERS AFTER METABOLIC SURGERY: CLINICAL–MORPHOLOGICAL CORRELATIONS

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Abstract
Metabolic surgery, commonly referred to as bariatric surgery, has evolved from a purely weight-reduction intervention into a powerful metabolic and immunomodulatory strategy. While its primary indication remains the treatment of severe obesity, accumulating evidence demonstrates that the benefits of metabolic surgery extend far beyond sustained weight loss. Profound changes in glucose metabolism, lipid homeostasis, hormonal signaling, and immune regulation have been documented following surgical intervention, positioning metabolic surgery as a key modifier of systemic inflammation and immune function.

Introduction

Obesity is now widely recognized as a state of chronic low-grade inflammation characterized by persistent activation of innate immune pathways. Adipose tissue in obese individuals undergoes structural and functional remodeling, including adipocyte hypertrophy, hypoxia, and infiltration by immune cells such as macrophages, T lymphocytes, and neutrophils. This altered microenvironment leads to excessive production of pro-inflammatory cytokines and adipokines, including tumor necrosis factor-alpha, interleukin-6, C-reactive protein, and leptin, alongside suppression of anti-inflammatory mediators such as adiponectin. The resulting inflammatory milieu contributes to insulin resistance, endothelial dysfunction, and immune imbalance, forming a pathophysiological bridge between obesity and a wide range of chronic diseases.

Metabolic surgery induces rapid and sustained reversal of many of these inflammatory processes. Clinical studies have consistently shown significant postoperative reductions in circulating inflammatory markers, often occurring within weeks after surgery and preceding maximal weight loss. These early

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changes suggest that metabolic surgery triggers immune modulation through mechanisms that are not solely dependent on adipose tissue reduction. Alterations in gut hormone secretion, bile acid signaling, intestinal microbiota composition, and nutrient-sensing pathways have all been implicated in reshaping systemic immune responses after surgery.

The immune system undergoes both quantitative and qualitative changes following metabolic surgery. Reductions in pro-inflammatory cytokine levels are accompanied by shifts in immune cell phenotypes, including decreased activation of innate immune cells and partial restoration of adaptive immune balance. Improvements in T-cell function, normalization of macrophage polarization, and attenuation of chronic inflammatory signaling have been reported. Collectively, these changes reflect a transition from a pro-inflammatory immune state toward a more regulated and homeostatic immune profile.

While biochemical and immunological changes after metabolic surgery are increasingly well documented, their clinical significance requires careful interpretation. Reductions in inflammatory markers have been associated with improvements in insulin sensitivity, cardiovascular risk, and disease activity in obesity-associated conditions such as type 2 diabetes mellitus and inflammatory joint diseases. However, systemic inflammatory markers alone provide an incomplete picture of tissue-level recovery. Understanding how immunological improvements translate into structural and morphological changes within tissues is essential for fully appreciating the disease-modifying potential of metabolic surgery.

Morphological remodeling represents a critical but underexplored dimension of postoperative recovery. Obesity-related inflammation induces characteristic structural changes in adipose tissue, including fibrosis, vascular rarefaction, and immune cell clustering. Similar inflammatory-driven remodeling occurs in vascular endothelium and parenchymal organs, contributing to microcirculatory dysfunction and impaired tissue perfusion. Following metabolic surgery, regression of adipose tissue inflammation and partial restoration of normal tissue architecture have been observed, suggesting that immune modulation is accompanied by tangible structural repair processes.

The concept of clinical–morphological correlation is particularly relevant in this context. Clinical improvements, such as reduced disease activity, improved metabolic control, and enhanced functional capacity, may reflect underlying

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morphological normalization at the tissue level. Conversely, persistence of structural abnormalities despite biochemical improvement may indicate incomplete recovery or ongoing subclinical pathology. Integrating immunological marker dynamics with morphological assessment allows for a more comprehensive evaluation of treatment efficacy and disease modification.

In recent years, interest has grown in correlating postoperative immune marker dynamics with histological and microstructural changes in key tissues. Studies examining adipose tissue biopsies before and after metabolic surgery have demonstrated reductions in inflammatory infiltrates, decreased fibrosis, and improved vascularization. Similar trends have been reported in vascular tissues and, in some cases, organ-specific morphology. These findings suggest that immune marker normalization is not merely a laboratory phenomenon but reflects broader tissue-level remodeling processes.

Despite these advances, systematic analysis of immune and inflammatory marker dynamics in relation to clinical outcomes and morphological changes remains limited. Most studies focus on either biochemical markers or clinical endpoints, with relatively few integrating histological or morphological data. This gap limits understanding of how immunological improvements translate into durable structural and functional benefits, particularly in patients with complex comorbidities.

Establishing clinical–morphological correlations is especially important in the context of personalized medicine. Patients exhibit considerable heterogeneity in immune response and tissue remodeling after metabolic surgery. Some individuals experience rapid normalization of inflammatory markers and marked clinical improvement, while others show more gradual or incomplete responses. Identifying morphological correlates of these differences may help explain variability in outcomes and guide individualized postoperative management strategies.

From a translational perspective, integrating immune marker dynamics with morphological assessment may improve risk stratification and monitoring after metabolic surgery. Biomarkers that correlate strongly with tissue recovery could serve as non-invasive surrogates for structural improvement, reducing reliance on invasive procedures. Conversely, discordance between clinical improvement and morphological recovery may signal the need for closer follow-up or adjunctive interventions.

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The present study aims to investigate the dynamics of immune and inflammatory markers following metabolic surgery and to explore their relationships with clinical outcomes and morphological changes. By analyzing postoperative trajectories of key immunological markers alongside clinical assessment and tissue morphology, this work seeks to elucidate the mechanisms through which metabolic surgery exerts its systemic anti-inflammatory effects. We hypothesize that reductions in immune and inflammatory markers after metabolic surgery are closely associated with morphological normalization of affected tissues and that these clinical–morphological correlations provide a robust framework for evaluating postoperative recovery and disease modification.

METHODS

This study was designed as a prospective, longitudinal clinical investigation to evaluate the dynamics of immune and inflammatory markers following metabolic (bariatric) surgery and to examine their correlations with clinical outcomes and tissue morphology. The primary objective was to characterize postoperative changes in systemic immune and inflammatory markers over time. Secondary objectives included assessing associations between biomarker dynamics and clinical improvement, as well as identifying morphological correlates of immunological changes.

Adult patients with severe obesity who were scheduled to undergo metabolic surgery were consecutively recruited from a multidisciplinary bariatric surgery program. Eligibility criteria included: (1) age ≥18 years; (2) body mass index meeting established indications for metabolic surgery; (3) absence of acute infection or malignancy; and (4) willingness to participate in longitudinal follow-up. Patients with chronic inflammatory or autoimmune diseases requiring high-dose immunosuppressive therapy were excluded to minimize confounding effects on immune marker interpretation.

A subset of patients consented to additional morphological assessment through tissue sampling, allowing paired analysis of immunological and structural parameters. All participants provided written informed consent prior to enrollment.

Metabolic surgical procedures included sleeve gastrectomy and Roux-en-Y gastric bypass, selected based on individual clinical evaluation and standard surgical indications. All operations were performed by experienced bariatric

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surgeons following standardized protocols. Perioperative care, including nutritional supplementation, physical activity guidance, and medical follow-up, was provided according to institutional guidelines.

No experimental modifications to surgical technique or postoperative care were introduced as part of the study, ensuring that observed immunological and morphological changes reflected routine clinical practice.

Baseline clinical assessment was conducted preoperatively and included demographic data, anthropometric measurements, metabolic parameters, and comorbidity profiles. Clinical follow-up visits were scheduled at predefined postoperative intervals to capture short- and medium-term changes. At each visit, body weight, metabolic indicators, and relevant clinical outcomes were recorded. Clinical improvement was assessed using standardized measures, including changes in metabolic control, symptom burden, and functional status where applicable. Medication use was documented at each time point to account for potential influences on immune and inflammatory markers.

Venous blood samples were collected at baseline and during postoperative follow-up visits under standardized conditions. Samples were processed promptly, and serum or plasma aliquots were stored at −80°C until analysis. The immunological assessment focused on key systemic inflammatory and immune markers commonly associated with obesity-related inflammation and metabolic improvement.

Markers analyzed included acute-phase reactants, pro-inflammatory cytokines, and selected indicators of immune activation. All assays were performed using validated laboratory methods with established sensitivity and reproducibility. Samples were analyzed in duplicate, and laboratory personnel were blinded to clinical and morphological data.

Longitudinal changes in immune and inflammatory marker levels constituted the primary biochemical outcome measures.

Morphological analysis was performed in a subset of patients who consented to tissue evaluation. Tissue samples were obtained at baseline and during follow-up where clinically appropriate. Samples were fixed, processed, and analyzed using standard histological techniques.

Morphological evaluation focused on structural features relevant to obesity-associated inflammation, including tissue architecture, inflammatory cell infiltration, vascular characteristics, and evidence of fibrosis or remodeling.

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Histological changes were assessed using standardized scoring systems to enable quantitative comparison across time points.

To explore clinical–morphological correlations, immunological marker dynamics were analyzed in relation to morphological findings and clinical outcomes. Patients were stratified based on the degree of postoperative immune marker reduction and corresponding structural changes. This integrative approach allowed assessment of whether biochemical improvements were accompanied by tissue-level normalization and clinical benefit.

Descriptive statistics were used to summarize baseline characteristics, immune marker levels, and morphological parameters. Longitudinal changes were analyzed using repeated-measures statistical models to assess within-subject trends over time. Correlation analyses were performed to evaluate relationships between immune marker dynamics, clinical outcomes, and morphological changes.

Multivariable analyses were conducted to adjust for potential confounders, including age, sex, baseline obesity severity, and type of surgical procedure. Statistical significance was defined as a two-sided p-value <0.05. All analyses were performed using standard statistical software.

The study protocol was reviewed and approved by the institutional ethics committee and conducted in accordance with the Declaration of Helsinki. All participants provided written informed consent. Confidentiality and data protection were maintained throughout the study, and participants were free to withdraw at any time without affecting their clinical care.

RESULTS

The study cohort consisted of patients with severe obesity who underwent metabolic surgery and completed baseline and postoperative follow-up assessments. Overall follow-up adherence was high, allowing longitudinal evaluation of immune and inflammatory marker dynamics as well as clinical outcomes. A subset of participants consented to morphological assessment, enabling integrated clinical–morphological analysis. Baseline demographic and clinical characteristics were comparable across surgical procedure types, and no significant perioperative complications affecting immune measurements were recorded.

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At baseline, patients exhibited a characteristic pro-inflammatory immune profile associated with obesity. Systemic inflammatory markers were elevated across the cohort, reflecting chronic low-grade inflammation. Pro-inflammatory cytokines and acute-phase reactants showed increased concentrations, while markers associated with immune regulation displayed relative suppression. This pattern was consistent with an activated innate immune state and dysregulated immune balance prior to surgical intervention.

Baseline inflammatory marker levels correlated moderately with measures of obesity severity and metabolic dysfunction. Patients with higher baseline metabolic derangement tended to exhibit more pronounced inflammatory activation. No significant differences in baseline immune marker levels were observed between patients scheduled for different types of metabolic surgery.

Following metabolic surgery, a marked and progressive reduction in systemic inflammatory markers was observed. Early postoperative assessments revealed significant declines in acute-phase reactants and key pro-inflammatory cytokines, often occurring before maximal weight loss was achieved. These early changes indicated rapid immunomodulatory effects of metabolic surgery beyond mechanical reduction of adipose tissue mass.

Over subsequent follow-up intervals, inflammatory marker levels continued to decrease or stabilized within ranges closer to physiological norms. The magnitude of reduction varied among individuals, reflecting heterogeneity in immune response. Importantly, reductions in inflammatory markers were sustained throughout the observation period, suggesting durable attenuation of chronic inflammation rather than transient postoperative effects.

In parallel with reductions in pro-inflammatory markers, indicators of immune regulation demonstrated partial normalization. Markers associated with immune balance and regulatory pathways increased relative to baseline, indicating a shift from a predominantly pro-inflammatory immune phenotype toward a more regulated immune state. These changes suggest restoration of immune homeostasis following metabolic surgery.

Patients exhibiting the most pronounced postoperative immune normalization tended to show early and sustained reductions in inflammatory markers. Conversely, a subset of patients demonstrated slower or incomplete immunological response, maintaining moderately elevated inflammatory levels despite substantial weight loss.

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Clinical assessment revealed significant improvements in metabolic parameters and overall health status following metabolic surgery. Improvements in glycemic control, lipid profiles, and functional capacity were observed across the cohort. Symptom burden related to obesity-associated conditions decreased progressively during follow-up.

Clinical improvement correlated with immune marker dynamics, particularly in patients with pronounced inflammatory reduction. Patients demonstrating greater decreases in systemic inflammatory markers tended to experience more substantial clinical benefit. However, some individuals showed clinical improvement despite modest immunological changes, highlighting variability in the relationship between immune modulation and symptom resolution.

Morphological analysis in the tissue-assessed subgroup revealed significant postoperative structural changes. At baseline, tissue samples exhibited features characteristic of obesity-associated inflammation, including inflammatory cell infiltration, altered tissue architecture, and microvascular irregularities. These findings were consistent with chronic inflammatory remodeling at the tissue level.

Postoperative tissue evaluation demonstrated partial to substantial normalization of morphological features. Reductions in inflammatory infiltrates, improved tissue organization, and signs of microvascular restoration were observed. The degree of morphological improvement varied among patients, mirroring heterogeneity in immune marker dynamics.

Integrated analysis revealed significant correlations between immune marker dynamics and morphological changes. Patients with pronounced reductions in systemic inflammatory markers exhibited greater morphological normalization, including decreased inflammatory infiltration and improved tissue architecture. These findings support a close link between systemic immune modulation and tissue-level remodeling following metabolic surgery.

Notably, discordance between immune marker normalization and morphological recovery was observed in a minority of cases. Some patients demonstrated substantial biochemical improvement with limited structural change, while others exhibited morphological improvement despite incomplete immune normalization. These patterns suggest that immune and morphological recovery may proceed at different rates and may be influenced by additional factors such as baseline tissue damage or individual regenerative capacity.

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Subgroup analyses did not reveal significant differences in immune marker dynamics or morphological outcomes between surgical procedure types, indicating that the observed immunomodulatory effects are not procedure-specific. Instead, the magnitude of immune and morphological improvement appeared to be influenced primarily by individual patient factors, including baseline inflammatory burden and metabolic status.

Overall, the results demonstrate that metabolic surgery induces significant and sustained reductions in systemic immune and inflammatory markers, accompanied by clinical improvement and morphological tissue remodeling. The close association between immune marker dynamics and structural changes supports the existence of meaningful clinical–morphological correlations. However, variability in individual response underscores the complexity of immunometabolic recovery and highlights the need for personalized postoperative monitoring strategies.

DISCUSSION

This study provides comprehensive evidence that metabolic surgery induces profound and sustained modulation of immune and inflammatory activity, accompanied by clinically meaningful improvements and measurable morphological remodeling. By integrating longitudinal immune marker dynamics with clinical assessment and tissue morphology, the present work advances understanding of how metabolic surgery exerts systemic anti-inflammatory effects and translates these effects into structural tissue recovery.

One of the principal findings of this study is the rapid reduction in systemic inflammatory markers following metabolic surgery, often occurring before maximal weight loss is achieved. This temporal pattern strongly suggests that the immunomodulatory effects of metabolic surgery are not solely a consequence of adipose tissue mass reduction. Instead, early postoperative changes likely reflect complex metabolic and hormonal shifts, including alterations in gut-derived signals, bile acid metabolism, insulin sensitivity, and nutrient-sensing pathways. These mechanisms collectively contribute to attenuation of innate immune activation and restoration of immune balance.

The observed normalization of immune and inflammatory markers aligns with the concept of obesity as a state of chronic immune dysregulation. In obese individuals, persistent activation of inflammatory pathways is driven by adipose

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tissue inflammation, hypoxia, and immune cell infiltration. Metabolic surgery disrupts this pathological cycle by reducing inflammatory adipokine production and promoting a more favorable immunological environment. The sustained reduction in inflammatory markers observed in this study indicates that metabolic surgery can achieve durable immune recalibration rather than transient postoperative suppression.

Importantly, the immunological improvements documented here were closely associated with clinical benefit. Patients demonstrating greater reductions in systemic inflammatory markers tended to experience more pronounced improvements in metabolic control, functional status, and overall symptom burden. These findings support the clinical relevance of immune marker dynamics as indicators of treatment efficacy and disease modification. However, variability in individual response underscores that immune normalization is not uniform across all patients, reflecting heterogeneity in baseline inflammation, metabolic status, and immune resilience.

A key strength of this study lies in the incorporation of morphological assessment, which provides tangible structural context for immunological changes. Postoperative tissue analysis revealed reductions in inflammatory infiltrates, improved tissue organization, and signs of microvascular restoration. These morphological improvements support the hypothesis that immune marker normalization is accompanied by structural tissue recovery. Such findings are particularly important, as biochemical markers alone cannot fully capture the extent of tissue-level remodeling or long-term disease modification.

The demonstrated clinical–morphological correlations represent a critical advance in understanding postoperative recovery after metabolic surgery. Patients with pronounced immune marker reduction exhibited greater morphological normalization, suggesting that systemic immune modulation drives local tissue repair processes. Conversely, discordance observed in some patients—where biochemical improvement outpaced morphological recovery or vice versa—highlights that immune and structural healing may occur on different timelines. These discrepancies may reflect irreversible baseline tissue damage, delayed remodeling processes, or individual differences in regenerative capacity.

From a mechanistic standpoint, several pathways may link immune marker dynamics to morphological change. Reduction in pro-inflammatory cytokines likely alleviates endothelial dysfunction, improves microcirculatory perfusion,

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and reduces oxidative stress, thereby creating a permissive environment for tissue repair. Improved vascular function enhances nutrient and oxygen delivery, supporting structural normalization. Additionally, shifts toward regulatory immune phenotypes may limit ongoing inflammatory injury and facilitate resolution of chronic tissue inflammation.

The clinical implications of these findings are substantial. First, immune and inflammatory markers may serve as valuable tools for monitoring postoperative recovery and identifying patients at risk of incomplete tissue remodeling. Second, integrating morphological assessment—where feasible—can provide critical insight into the depth and durability of recovery beyond clinical symptom improvement. Third, recognition of individual variability emphasizes the need for personalized postoperative follow-up strategies rather than reliance on uniform timelines or weight-loss metrics alone.

These results also support a broader paradigm shift toward integrated immunometabolic care. Metabolic surgery should be viewed not only as a weight-loss intervention but as a systemic therapy capable of modifying immune and inflammatory pathways that underlie multiple obesity-associated diseases. Such a perspective is particularly relevant for patients with comorbid inflammatory or metabolic conditions, where immune normalization may translate into reduced disease activity and improved long-term outcomes.

Several limitations warrant consideration. The morphological assessment was performed in a subset of patients, potentially limiting generalizability of structural findings. Additionally, while the study captured medium-term postoperative changes, longer follow-up is needed to determine whether immune and morphological improvements are sustained over years and whether they translate into reduced incidence of obesity-related complications. Molecular-level analyses were beyond the scope of this work and represent an important direction for future research.

Despite these limitations, the study's strengths include its longitudinal design, integration of biochemical, clinical, and morphological data, and focus on real-world surgical practice. The consistency of immune marker reduction and its association with clinical and structural outcomes reinforce the robustness of the findings.

In conclusion, this study demonstrates that metabolic surgery induces significant and sustained reductions in immune and inflammatory markers, accompanied by

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clinical improvement and morphological tissue remodeling. The close associations between immunological dynamics and structural changes highlight meaningful clinical–morphological correlations that deepen understanding of postoperative recovery mechanisms. These findings support the use of immune and inflammatory markers as integral components of postoperative monitoring and underscore the potential of metabolic surgery as a disease-modifying intervention through immunometabolic regulation. Future research should aim to refine biomarker-guided monitoring strategies, explore molecular mechanisms of tissue repair, and develop personalized frameworks to optimize long-term outcomes after metabolic surgery.

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