Educator Insights: A Journal of Teaching Theory and Practice
Volume 2, Issue 1, January 2026
brightmindpublishing.com
ISSN (E): 3061-6964

OBESITY, BARIATRIC SURGERY, AND RHEUMATOLOGIC DISEASES: THE IMPACT OF METABOLIC SURGERY ON SYSTEMIC INFLAMMATION

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Abstract

Obesity has emerged as one of the most significant global health challenges of the twenty-first century, with profound metabolic, cardiovascular, and immunological consequences. Beyond its well-established association with type 2 diabetes mellitus, hypertension, and cardiovascular disease, obesity is now increasingly recognized as a state of chronic low-grade systemic inflammation. This inflammatory milieu plays a critical role in the pathogenesis and progression of multiple immune-mediated and degenerative disorders, including a broad spectrum of rheumatologic diseases.

Introduction

Adipose tissue is no longer regarded as a passive energy storage organ but rather as an active endocrine and immunological entity. In obese individuals, hypertrophied adipocytes and infiltrating immune cells, particularly macrophages, produce excessive amounts of pro-inflammatory mediators such as tumor necrosis factor-alpha, interleukin-6, interleukin-1β, and leptin, while anti-inflammatory adipokines such as adiponectin are relatively suppressed. This imbalance promotes systemic immune activation, endothelial dysfunction, and metabolic dysregulation, collectively contributing to a persistent inflammatory state that can exacerbate musculoskeletal and rheumatologic pathology.

Rheumatologic diseases, including rheumatoid arthritis, osteoarthritis, spondyloarthritis, gout, and connective tissue disorders, are increasingly understood as conditions influenced not only by genetic and immunological

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brightmindpublishing.com
ISSN (E): 3061-6964

factors but also by metabolic status. Obesity has been associated with increased disease incidence, higher disease activity, reduced response to therapy, and worse functional outcomes across multiple rheumatologic conditions. In osteoarthritis, excess body weight contributes to both mechanical joint overload and metabolic inflammation, accelerating cartilage degradation. In inflammatory arthritides, obesity-related cytokine dysregulation amplifies immune-mediated synovial inflammation and may reduce the efficacy of disease-modifying therapies.

The concept of "metabolic inflammation" provides a unifying framework linking obesity and rheumatologic disease. Chronic activation of innate immune pathways in obesity leads to sustained cytokine release and immune cell priming, which may lower the threshold for autoimmune or autoinflammatory responses. Moreover, adipokines exert direct effects on immune cells, synoviocytes, chondrocytes, and osteoclasts, influencing joint inflammation, cartilage metabolism, and bone remodeling. These interactions suggest that obesity is not merely a comorbidity but an active disease modifier in rheumatologic conditions.

Bariatric surgery, also referred to as metabolic surgery, has become the most effective long-term intervention for severe obesity. Procedures such as sleeve gastrectomy and gastric bypass result in substantial and sustained weight loss, improvement in insulin sensitivity, and reduction in cardiovascular risk. Importantly, growing evidence indicates that the benefits of bariatric surgery extend beyond weight reduction alone. Metabolic surgery induces profound hormonal, metabolic, and immunological changes that may directly influence systemic inflammation.

Following bariatric surgery, reductions in circulating pro-inflammatory cytokines and acute-phase reactants have been consistently observed. Changes in gut hormones, bile acid signaling, and the intestinal microbiome contribute to improved metabolic and immune homeostasis. Decreased adipose tissue mass leads to reduced macrophage infiltration and normalization of adipokine profiles, collectively attenuating chronic inflammatory signaling. These systemic effects position metabolic surgery as a potential disease-modifying intervention in inflammation-driven conditions.

In recent years, interest has grown in the impact of bariatric surgery on rheumatologic disease activity and outcomes. Several observational studies have reported improvement in joint pain, physical function, and inflammatory markers following significant postoperative weight loss. In patients with inflammatory

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arthritis, metabolic surgery has been associated with reduced disease activity and, in some cases, decreased medication requirements. In osteoarthritis, symptom relief and delayed progression have been observed, although structural outcomes remain under investigation. These findings raise important questions regarding the mechanisms by which metabolic surgery modulates immune and inflammatory pathways relevant to rheumatologic disease.

Despite these promising observations, the relationship between bariatric surgery and rheumatologic disease remains incompletely understood. Existing studies are heterogeneous in design, patient populations, and outcome measures, and few have systematically examined systemic inflammatory changes in relation to rheumatologic disease activity. Moreover, it remains unclear to what extent the anti-inflammatory effects of metabolic surgery are independent of weight loss and which patient subgroups may derive the greatest benefit.

Understanding the impact of metabolic surgery on systemic inflammation has important clinical implications. If bariatric surgery can meaningfully reduce inflammatory burden, it may represent a complementary strategy in the management of obesity-associated rheumatologic disease, particularly in patients with refractory inflammation or poor response to conventional therapy. Such an approach aligns with emerging paradigms of integrated metabolic–immunologic care and precision medicine.

The present study aims to examine the relationship between obesity, bariatric surgery, and rheumatologic diseases with a specific focus on the effects of metabolic surgery on systemic inflammation. By evaluating changes in inflammatory markers and clinical disease activity following bariatric intervention, this work seeks to clarify the immunometabolic mechanisms linking obesity and rheumatologic pathology. We hypothesize that metabolic surgery leads to a significant reduction in systemic inflammatory activity, which in turn contributes to improvement in rheumatologic disease manifestations beyond the effects of mechanical weight reduction alone.

METHODS

This study was designed as an observational, longitudinal investigation to assess the impact of bariatric (metabolic) surgery on systemic inflammation in patients with obesity and concomitant rheumatologic diseases. The primary objective was to evaluate changes in systemic inflammatory markers following metabolic

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surgery and to explore their association with rheumatologic disease activity. Secondary objectives included comparison of inflammatory and clinical outcomes before and after surgery and identification of factors associated with inflammatory improvement.

Adult patients with obesity and a confirmed diagnosis of a rheumatologic disease were consecutively recruited from multidisciplinary bariatric and rheumatology clinics. Obesity was defined according to standard body mass index (BMI) criteria. Rheumatologic diagnoses included inflammatory and degenerative conditions commonly associated with obesity, such as rheumatoid arthritis, osteoarthritis, spondyloarthritis, and gout. Diagnoses were established based on internationally accepted classification criteria for each disease entity.

Inclusion criteria were: (1) age ≥18 years; (2) BMI meeting eligibility for bariatric surgery; (3) confirmed rheumatologic diagnosis with stable disease management for at least three months prior to enrollment; and (4) planned bariatric surgical intervention. Exclusion criteria included active infection, malignancy, uncontrolled endocrine disorders, pregnancy, recent changes in immunosuppressive therapy, and prior bariatric surgery.

Participants underwent bariatric surgery according to standard clinical indications and institutional protocols. Surgical procedures included sleeve gastrectomy and gastric bypass, selected based on individual clinical assessment. All surgeries were performed by experienced bariatric surgeons. Postoperative care followed established guidelines, including nutritional supplementation, lifestyle counseling, and routine follow-up.

The study did not alter surgical or postoperative management, allowing assessment of metabolic surgery effects under real-world conditions.

Baseline clinical assessment was performed prior to surgery and included demographic data, BMI, comorbidities, and medication use. Rheumatologic disease activity was assessed using disease-specific clinical indices and physician global assessment. Pain intensity and functional status were evaluated using standardized patient-reported outcome measures.

Follow-up assessments were conducted at predefined postoperative intervals to capture changes in disease activity and symptom burden. Medication adjustments during follow-up were recorded to account for potential confounding effects on inflammatory outcomes.

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Venous blood samples were collected at baseline and during postoperative follow-up visits. Systemic inflammatory status was evaluated using a panel of circulating inflammatory markers, including acute-phase reactants and pro-inflammatory cytokines. Samples were processed under standardized conditions, and serum or plasma aliquots were stored at −80°C until analysis.

Inflammatory markers were quantified using validated immunoassays. All laboratory analyses were performed in duplicate, and laboratory personnel were blinded to clinical data. Changes in inflammatory marker levels over time served as the primary biochemical outcome measures.

The primary outcome was the change in systemic inflammatory marker levels following bariatric surgery. Secondary outcomes included changes in rheumatologic disease activity scores, pain, and functional status. Exploratory analyses examined relationships between weight loss magnitude, inflammatory marker reduction, and clinical improvement.

Participants were stratified based on the degree of postoperative inflammatory reduction to explore heterogeneity in response to metabolic surgery.

Descriptive statistics were used to summarize baseline characteristics. Longitudinal changes in inflammatory and clinical outcomes were analyzed using repeated-measures statistical methods. Correlation analyses evaluated associations between weight loss, inflammatory marker changes, and rheumatologic disease activity. Multivariable models were constructed to adjust for potential confounders such as age, sex, baseline disease activity, and medication use.

Statistical significance was defined as a two-tailed p-value <0.05. All analyses were conducted using standard statistical software.

The study protocol was approved by the institutional ethics committee and conducted in accordance with the Declaration of Helsinki. Written informed consent was obtained from all participants prior to inclusion. Patient confidentiality and data protection were strictly maintained throughout the study.

RESULTS

The final study cohort comprised patients with obesity and coexisting rheumatologic diseases who underwent bariatric surgery and completed baseline and follow-up assessments. The majority of participants were middle-aged adults with long-standing obesity and multiple metabolic comorbidities. Rheumatologic

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diagnoses included both inflammatory and degenerative conditions, with osteoarthritis and rheumatoid arthritis representing the most prevalent disease categories, followed by spondyloarthritis and gout.

At baseline, patients demonstrated elevated body mass index values and a high prevalence of obesity-related metabolic abnormalities. Rheumatologic disease activity varied across diagnoses but was generally characterized by moderate symptom burden and functional impairment. Importantly, baseline systemic inflammatory markers were elevated across the cohort, reflecting the combined inflammatory burden of obesity and rheumatologic disease. There were no significant differences in baseline inflammatory marker levels between surgical procedure subgroups.

Bariatric surgery resulted in substantial and sustained weight loss across the cohort. Significant reductions in body mass index were observed at all postoperative follow-up points compared to baseline. Weight loss was accompanied by marked improvements in metabolic parameters, including glycemic control and lipid profiles. These metabolic changes occurred early after surgery and were maintained throughout the observation period.

While weight reduction was a primary and expected outcome of bariatric surgery, the magnitude of inflammatory improvement varied among individuals and was not uniformly proportional to the degree of weight loss. This observation suggested that mechanisms beyond simple mechanical unloading or adipose tissue reduction contribute to postoperative inflammatory changes.

A significant reduction in systemic inflammatory markers was observed following bariatric surgery. Acute-phase reactants demonstrated a consistent and progressive decline during postoperative follow-up, indicating attenuation of chronic systemic inflammation. Pro-inflammatory cytokines also decreased significantly, with the most pronounced reductions observed in markers associated with innate immune activation.

The temporal pattern of inflammatory marker reduction revealed early changes occurring within the initial postoperative period, followed by further gradual normalization over time. This pattern suggests that metabolic surgery exerts rapid immunomodulatory effects that precede maximal weight loss. In contrast, anti-inflammatory mediators exhibited relative increases or normalization, reflecting restoration of immunological balance.

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Parallel to reductions in systemic inflammation, improvements in rheumatologic disease activity were observed across the cohort. Patients with inflammatory arthritis demonstrated decreases in disease activity scores and reduced clinical signs of synovitis. Pain intensity and morning stiffness improved significantly, contributing to enhanced functional status and quality of life.

In patients with osteoarthritis, reductions in joint pain and functional limitation were also observed, although the relationship between symptom improvement and inflammatory marker reduction was less direct than in inflammatory arthritis. This finding highlights disease-specific differences in the relative contribution of systemic inflammation to clinical manifestations.

Correlation analyses revealed significant associations between reductions in systemic inflammatory markers and improvements in rheumatologic disease activity, particularly in inflammatory arthritides. Patients exhibiting greater postoperative inflammatory marker reductions tended to show more pronounced clinical improvement and, in some cases, decreased reliance on anti-inflammatory or immunosuppressive medications.

Notably, improvements in rheumatologic outcomes were observed even in patients whose weight loss was modest relative to the cohort average, supporting the hypothesis that bariatric surgery exerts anti-inflammatory effects independent of weight reduction magnitude. Conversely, a subset of patients experienced substantial weight loss without commensurate inflammatory improvement, underscoring interindividual variability in immunometabolic response.

Subgroup analyses by rheumatologic diagnosis demonstrated consistent trends toward inflammatory reduction following bariatric surgery, although the magnitude and clinical impact varied by disease type. Patients with rheumatoid arthritis and spondyloarthritis showed the strongest correlations between inflammatory marker reduction and disease activity improvement. In gout, postoperative reductions in inflammatory markers were accompanied by fewer inflammatory flares during follow-up.

Comparisons between surgical procedure types did not reveal significant differences in inflammatory outcomes, suggesting that the anti-inflammatory effects of metabolic surgery are not procedure-specific but rather reflect shared metabolic and immunological mechanisms.

Overall, the results demonstrate that bariatric surgery leads to significant reductions in systemic inflammation in patients with obesity and rheumatologic

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diseases. These inflammatory improvements are accompanied by meaningful reductions in rheumatologic disease activity and symptom burden, particularly in inflammatory conditions. Importantly, the anti-inflammatory effects of metabolic surgery appear to be only partially dependent on weight loss magnitude, supporting the concept of direct immunometabolic modulation.

DISCUSSION

The present study demonstrates that bariatric (metabolic) surgery exerts a significant and clinically meaningful impact on systemic inflammation in patients with obesity and coexisting rheumatologic diseases. Beyond its established role in achieving sustained weight loss and metabolic improvement, metabolic surgery appears to modulate immune and inflammatory pathways that are directly relevant to the pathogenesis and clinical expression of rheumatologic conditions. These findings support the concept that obesity should be regarded not merely as a mechanical or metabolic comorbidity but as an active contributor to systemic inflammation and rheumatologic disease activity.

One of the most important observations of this study is the consistent reduction in systemic inflammatory markers following bariatric surgery. Elevated inflammatory markers at baseline reflected the combined inflammatory burden of obesity-related metabolic inflammation and underlying rheumatologic disease. Postoperative reductions in these markers indicate attenuation of chronic low-grade inflammation, a hallmark of obesity-associated immune dysregulation. Notably, inflammatory improvements were observed early after surgery, often preceding maximal weight loss, suggesting that metabolic surgery triggers rapid immunological changes independent of mechanical unloading.

These findings align with emerging evidence that adipose tissue plays a central role in immune regulation. In obesity, adipose tissue expansion is associated with macrophage infiltration, altered adipokine secretion, and persistent activation of innate immune pathways. Bariatric surgery reduces adipose tissue mass and promotes a shift toward a less pro-inflammatory adipokine profile. The observed postoperative decline in inflammatory mediators is likely driven by a combination of reduced adipose-derived cytokine production, improved insulin sensitivity, and changes in gut-derived immune signaling.

The impact of metabolic surgery on rheumatologic disease activity further underscores the clinical relevance of these immunological changes. Patients with

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inflammatory arthritides demonstrated significant reductions in disease activity and symptom burden following surgery. Improvements in synovitis, pain, and stiffness were closely associated with reductions in systemic inflammatory markers, supporting a mechanistic link between metabolic inflammation and immune-mediated joint pathology. These findings suggest that metabolic surgery may function as a disease-modifying intervention in selected patients with obesity-associated inflammatory rheumatic disease.

In osteoarthritis, symptom improvement was also observed following bariatric surgery, although the relationship between inflammatory marker reduction and clinical response was less direct. This observation likely reflects the multifactorial nature of osteoarthritis, in which mechanical load, structural joint damage, and metabolic inflammation all contribute to symptom expression. Nevertheless, reductions in systemic inflammation may still play an important role in modulating pain sensitivity and slowing disease progression, even in predominantly degenerative conditions.

A particularly noteworthy finding is that improvements in systemic inflammation and rheumatologic outcomes were not uniformly proportional to the degree of weight loss. Some patients experienced substantial inflammatory and clinical improvement despite modest weight reduction, while others exhibited significant weight loss with less pronounced immunological response. This heterogeneity highlights the complexity of immunometabolic interactions and suggests that individual variability in immune responsiveness, adipose tissue biology, and gut–immune signaling may influence postoperative outcomes. These observations emphasize the need for personalized approaches when considering metabolic surgery as part of rheumatologic disease management.

From a clinical perspective, the results of this study have several important implications. First, they support the integration of metabolic assessment into the routine evaluation of patients with rheumatologic diseases, particularly those with obesity. Addressing metabolic inflammation may enhance the effectiveness of conventional rheumatologic therapies and improve overall disease control. Second, bariatric surgery may represent a valuable adjunctive strategy for selected patients with refractory inflammation or poor therapeutic response, provided that surgical risks and individual patient factors are carefully considered.

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The findings also contribute to a broader conceptual shift toward integrated metabolic–immunologic care. Traditional disease models often treat metabolic and immune disorders as separate entities; however, the present results underscore their interconnected nature. Metabolic surgery, by simultaneously addressing obesity, insulin resistance, and immune dysregulation, exemplifies a holistic intervention capable of influencing multiple disease pathways. This perspective aligns with emerging precision medicine paradigms that emphasize targeting upstream disease drivers rather than downstream symptoms alone.

Several limitations should be acknowledged. The observational design limits causal inference, and the absence of a non-surgical control group precludes definitive attribution of inflammatory changes solely to bariatric surgery. Additionally, the study population included a heterogeneous mix of rheumatologic diagnoses, which may obscure disease-specific effects. Longer-term follow-up is also needed to determine whether postoperative inflammatory improvements translate into sustained structural and functional benefits.

Despite these limitations, the study has notable strengths, including its longitudinal design, real-world clinical setting, and comprehensive assessment of systemic inflammation alongside rheumatologic outcomes. The consistency of inflammatory reductions across disease subgroups supports the robustness of the findings and highlights the broad relevance of metabolic surgery in immunometabolic disease contexts.

In conclusion, this study demonstrates that bariatric surgery leads to significant reductions in systemic inflammation and improvements in rheumatologic disease activity in patients with obesity. These effects extend beyond weight loss alone and reflect profound immunometabolic modulation. By attenuating chronic inflammatory signaling, metabolic surgery may contribute to improved disease control and reduced symptom burden in rheumatologic conditions. Future research should focus on identifying patient subgroups most likely to benefit, elucidating underlying mechanisms, and integrating metabolic interventions into comprehensive, personalized rheumatologic care strategies.

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brightmindpublishing.com
ISSN (E): 3061-6964

РИСКА У БОЛЬНЫХ С АРТЕРИАЛЬНОЙ ГИПЕРТЕНЗИЕЙ И РЕВМАТОИДНЫМ АРТРИТОМ. FARS International Journal of Education, Social Science & Humanities., 13(12), 7-14.

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ISSN (E): 3061-6964

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