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GLP-1 analogues in rheumatology - the influence of obesity on the effectiveness of biological treatment

Julia Agnieszka Michalak, ORCID https://orcid.org/0009-0006-2629-7692
E-mail: lekmichalakjulia@gmail.com
Independent Public Healthcare Center, Szpitalna 7, 64-000 Kościan, Poland

Klaudia Martyna Patrzykąt, ORCID https://orcid.org/0009-0000-9440-5444
E-mail: patrzykat.klaudia@gmail.com
109 Military Hospital with Policlinic in Szczecin, Piotra Skargi 9-11, 70-965 Szczecin, Poland

Anna Maria Zakrzewska, ORCID https://orcid.org/0009-0009-8757-2274
E-mail: lek.annazakrzewska@gmail.com
Central Clinical Hospital of the Medical University of Łódź, Pomorska 251, 92-213 Łódź, Poland
Aleksander Midera, ORICD https://orcid.org/0009-0008-5809-427X
E-mail: aleksander.midera@gmail.com
Independent Public Healthcare Center, Tulipanowa 8, 95-060 Brzeziny, Poland
Julia Aleksandra Leśniak, ORCID https://orcid.org/0009-0005-7375-5951
E-mail: julialesniak577@gmail.com
Central Clinical Hospital of the Medical University of Łódź, Pomorska 251, 92-213 Łódź, Poland

Michał Borowski, ORCID https://orcid.org/0009-0004-7316-2411
E-mail: lek.michalborowski@gmail.com
Independent Public Healthcare Center, Szpitalna 37, 05-300 Mińsk Mazowiecki, Poland

Klaudia Elżbieta Niwińska, ORCID https://orcid.org/0009-0002-3648-277X
E-mail: klaudia11100210@gmail.com
Independent Public Clinical Hospital named after Prof. W. Orłowski (CMKP), Czerniakowska 231, 00-416 Warszawa, Poland

Zofia Czaplińska-Paszek, ORCID https://orcid.org/0000-0003-2429-8262
E-mail: czaplinskapaszek@gmail.com
Medical University of Warsaw, Żwirki i Wigury 61, 02-091 Warszawa, Poland

Natalia Maria Leśniak, ORCID https://orcid.org/0009-0006-0815-6554
E-mail: natalialesniak57@gmail.com
Central Clinical Hospital of the Medical University of Łódź, Pomorska 251, 92-213 Łódź, Poland

Kinga Popielarska, ORCID https://orcid.org/0009-0009-7797-5301
E-mail: kingapopielarska@gmail.com
Medical University of Gdańsk, Marii Skłodowskiej-Curie 3a, 80-210 Gdańsk, Poland

Corresponding Author
Julia Agnieszka Michalak lekmichalakjulia@gmail.com

Abstract

Obesity and overweight are becoming a global health challenge and are increasingly prevalent in patients with rheumatic diseases such as rheumatoid arthritis (RA), psoriatic arthritis (PsA), and systemic lupus erythematosus (SLE). Adipose tissue serves as an active endocrine organ, secreting adipokines and proinflammatory cytokines, including leptin, tumor necrosis factor alpha (TNF-alpha), and interleukin 6 (IL-6). Obesity exacerbates inflammatory processes, increases the number of classically activated macrophages (M1) and reduces

adiponectin levels, which may sustain and amplify the immune response typical of autoimmune diseases. Available studies indicate that obesity is associated with a lower likelihood of achieving minimal disease activity, particularly in RA and PsA. In RA patients, a higher body mass index (BMI) correlates with greater pain, a higher Health Assessment Questionnaire (HAQ) score, and a lower chance of remission, even with similar treatment. In the case of PsA, obesity increases the risk of disease onset. In SLE, a higher BMI is associated with increased disease activity and excessive fatigue. Research suggests that the effectiveness of TNF-alpha inhibitors may be reduced in patients with obesity, while the response to medications such as tocilizumab or abatacept is often independent of BMI. At the same time, abundant data confirm that weight loss brings tangible benefits – improving functional outcomes, and increasing the chance of achieving minimal disease activity, particularly in RA and PsA. Preliminary observations regarding Glucagon-Like Peptide-1 (GLP-1) agonists indicate their possible anti-inflammatory effects, although further research is needed. In summary, obesity significantly affects the course of rheumatic diseases and the effectiveness of therapy, and weight loss is an important element in supporting treatment.

Keywords obesity, rheumatoid arthritis, psoriatic arthritis, biological treatments, minimal disease activity, Glucagon-Like Peptide-1 analogues

Introduction:

Overweight and obesity are becoming increasingly serious problems in the modern world. According to the World Obesity Atlas, in 2020, there were 2.6 billion overweight or obese people (Body mass index (BMI) $\geq$ 25 kg/m^2), of whom 0.99 billion were obese [1]. Therefore, it can be concluded that the number of these individuals in the population of people with rheumatological diseases is also growing. Such patients include people with rheumatoid arthritis (RA), spondyloarthritis, including psoriatic arthritis (PsA), systemic lupus erythematosus (SLE), and others. Biological treatments have revolutionized this field of medicine, and damage to internal organs and serious joint deformities are becoming increasingly rare. These antibodies are designed to selectively block proinflammatory cytokines or their receptors and eliminate cells involved in the inflammatory cascade. These include tumor necrosis factor alpha inhibitors (anti-TNF-alpha), interleukin-1 inhibitors (anti-IL-1),

interleukin-6 inhibitors (anti-IL-6), anti–cluster of differentiation 20 antibodies (anti-CD-20), T-cell co-stimulation inhibitor (anti-LTiB), interleukin-17 inhibitors (anti-IL-17), interleukin-23 inhibitors (anti-IL-23), anti–type I interferon (anti-IFN-1), and phosphodiesterase-4 inhibitors (anti-PDE-4) [2]. This group of drugs is known as disease-modifying antirheumatic drugs (DMARDs). These drugs achieve minimal disease activity in many patients. Due to the growing problem of obesity in the modern world, scientists are increasingly asking whether excess body weight affects the effectiveness of biological treatments.

Obesity and inflammation

Adipose tissue produces and releases a number of pro-inflammatory and anti-inflammatory factors, including the adipokines leptin, adiponectin, and resistin, as well as cytokines and chemokines such as TNF-alpha, IL-6, and monocyte chemoattractant protein-1 (MCP-1). Obesity is associated with overexpression of pro-inflammatory factors and decreased secretion of adiponectin, This substance is believed to inhibit inflammation. Elevated lipid levels cause adipocyte hypertrophy, hypoxia, and increased cell death. Even more inflammatory mediators are released. MCP-1 and other chemokines produced by adipocytes and immune cells promote an increased influx of monocytes and other cells of the innate and adaptive immune system into adipose tissue. This results in a significant increase in the number of macrophages, which have a pronounced proinflammatory profile (M1 type). Leptin also plays a significant role by stimulating the inflammatory phenotype of T lymphocytes, macrophages, and other innate immune cells, which in turn can lead to increased levels of proinflammatory mediators such as TNF-alpha and IL-6, acting via the janus kinase/signal transducer and activator of transcription (JAK/STAT) pathway. This leads to increased inflammation and an immune response that perpetuates immune-mediated diseases. TNF-alpha, in turn, inhibits adiponectin production, while leptin production remains unchanged, as demonstrated in studies conducted on human adipocytes in vivo. Reduced adiponectin levels reduce the amount of oxidized fatty acids, which leads to their accumulation and activates pro-inflammatory serine kinase cascades, which causes the release of IL-6 by adipose tissue and, as a result, stimulates hepatocytes to synthesize and secrete C-reactive protein (CRP) [3,4].

In a meta-analysis by Lupoli et al., the rate of achieving minimal disease activity was significantly lower in obese patients than in those of normal weight. The study reviewed 17 studies involving a total of 6,693 patients with RA and PsA [5]. In a large cohort of patients with early RA followed for an average of 9.5 years at study entry, obese individuals had slightly higher Health Assessment Questionnaire (HAQ), Visual Analogue Scale for pain (VAS pain)

and general health status scores than patients with a BMI <30, with similar Disease Activity Score in 28 joints (DAS28) and systemic inflammatory markers (erythrocyte sedimentation rate (ESR), CRP). In the same cohort, at last follow-up, obese individuals had more active and severe disease, with a decreased likelihood of being in remission and achieving sustained remission, despite similar use of DMARDs, steroids, and biologics across all groups [6]. DAS28 may not be the best way to assess therapeutic response in obese patients due to the increase in scores on subjective measures, which may be explained by comorbidities in these patients, regardless of RA symptoms. However, the difference in scores was statistically significant. Interestingly, the group of people with high BMI had less radiographic joint damage, which may explain the greater risk of developing seronegative RA, which is known to be associated with a better structural prognosis. The second theory is related to differences in adiponectin levels. Individuals in this group have lower levels of adiponectin, which may protect against joint damage due to its distinct pro-inflammatory effects on fibroblast-like synoviocytes [7].

Studies have shown a higher prevalence of obesity in PsA compared with RA. This indicates that obesity is not only a consequence of reduced physical activity due to bothersome symptoms but also precedes and likely contributes significantly to the increased risk of developing psoriasis and PsA. British study of 75,395 people with psoriasis found that those with a BMI ≥ 35 kg/m² had an almost 50% higher risk of developing PsA than those with psoriasis and a BMI < 25 kg/m² [8].

Among patients with SLE, the prevalence of obesity is high, approximately 30–40%. Meta-analysis has shown that these individuals are more susceptible to metabolic syndrome compared to the healthy control population. Interestingly, many reports suggest that serum adiponectin concentrations are increased in patients with SLE, especially in patients with lupus nephritis. However, further studies are needed to understand its effects in this group of patients. [9]. The Lupus Outcomes Study cohort showed that a higher BMI was associated with greater SLE disease activity, increased fatigue, and poorer quality of life. The results were based on clinical measures such as the SLE Disease Activity Index (SLEDAI) and the British Isles Lupus Assessment Group (BILAG) score [10].

In the case of Sjogren's syndrome, the results are somewhat surprising, as obesity appears to have protective properties, leading to a more favorable disease course with better parameters and a lower disease activity index. However, these reports require verification [11].

In the case of scleroderma, the question "how obesity affects disease activity" has not been clearly answered. The main reason may be that these patients are more likely to be underweight due to gastrointestinal involvement and difficulty in eating [12].

Biological treatment and its effectiveness

TNF-alpha inhibitors were among the first biologic drugs introduced to the market. Currently, they are primarily used to treat RA, PsA, and other spondyloarthropathies. Some of them are administered in a fixed dose, such as adalimumab, certolizumab, and golimumab. However, the doses of infliximab and etanercept are calculated per kilogram of body weight, similarly to tocilizumab, an anti-IL-6 antibody, and abatacept, an anti-LTiB antibody. Rituximab is also administered in a fixed dose for RA [2].

Some studies indicate that the effectiveness of TNF-alpha inhibitors is reduced in patients with obesity, whereas in the case of tocilizumab and abatacept this phenomenon does not occur and the response to treatment is similar in both groups.

A study by Novella-Navarro et al. showed that among 105 patients with RA, the effectiveness of TNF-alpha inhibitor treatment was lower in those with higer BMI. The effect was assessed after 6 months of drug administration. The researchers concluded that the lack of response to the drug may be related to leptin levels. Leptin levels are elevated in obese individuals and in patients who failed to improve. However, these differences were not always statistically significant. This relationship was not observed in the tocilizumab-treated group. These conclusions were confirmed by the analysis of data from patients participating in the German cohort study "Rheumatoid Arthritis: Follow-up on Biological Therapy" (RABBIT). This registry includes 10,593 patients: 7,845 women (2,192 obese) and 2,748 men (718 obese). However, in this case, the results for tocilizumab differed by sex. A decrease in tocilizumab efficacy was observed in obese women, a finding not previously demonstrated. The authors suggest that this may be due to the lack of sex differentiation in previous analyses. However, no effect of body weight on the effectiveness of rituximab and abatacept treatment was demonstrated [13,14,15,16].

However, studies based on the Swiss Clinical Quality Management in Rheumatic Diseases (SCQM) registry contradict these conclusions. Abatacept did not demonstrate superiority over TNF-alpha inhibitors. Patients treated with etanercept, infliximab, or abatacept had similar odds of achieving remission or low disease activity as assessed by DAS28 compared with patients treated with adalimumab, regardless of BMI category. The BIOSTAR cohort study also did not

show significant differences in the effectiveness of different drug groups, but clearly confirmed that obesity affects increased disease activity [17,18].

A study of 270 patients with PsA also found an association between obesity and a lower chance of achieving minimal disease activity. These conclusions were confirmed by an analysis of 1,943 Danish and Icelandic cases. This result was statistically significant across sex, nationality and type of TNF-alpha inhibitor [19,20].

In the case of lupus erythematosus, there are significantly fewer studies on the relationship between the effectiveness of biologics in obese individuals. One such study is by Borg et al., which showed that the effectiveness of belimumab was comparable across all BMI categories. However, overweight and obese patients reported more complaints after 52 weeks, despite improvements in clinical parameters. This may be related to complaints directly related to excess body weight [21].

In summary, research on the impact of obesity on the effectiveness of biological treatment for rheumatic diseases is inconclusive, with the most evidence being available for RA. However, a higher BMI has been shown to correlate with difficulty achieving minimal disease activity and exacerbate symptoms.

Weight reduction and minimal disease activity

A randomized, single-blind, controlled, pilot study of a dietary intervention aimed at weight loss in obese patients with RA was conducted in California. Patients in the intervention group achieved significant improvements. Subjective well-being, as assessed by the Routine Assessment of Patient Index Data 3 (RAPID3), DAS28 and Health Assessment Questionnaire – Disability Index (HAQ-DI) scores, and ultrasound indices (power doppler ultrasound score of 34 joints (PDUS-34), grey scale synovial hypertrophy of 34 joints (GSUS-34)) improved. Ultrasound parameters demonstrated statistically significant improvement in both groups. Multi-Biomarker Disease Activity (MBDA) and ESR showed minimal change from baseline in either group. Furthermore, there were significant differences between the groups in serum leptin and adiponectin concentrations after 12 weeks. A study conducted several years earlier by Weijers et al. also found that weight loss reduced disease activity without intensifying treatment. However, this topic requires further research in a larger group of individuals [22,23]. Furthermore, there have been reports of the beneficial effects of a diet rich in polyphenols, particularly a Mediterranean diet. Studies indicate a reduction in the DAS28 score in patients who adhere to the diet. A 12 week change in eating habits could reduce tender joints and improving general health but was unable to rescue physical function or morning stiffness.

However, to consider this a safe and effective adjunctive treatment for RA, studies on larger groups of patients are necessary [24,25].

Studies conducted among patients with PsA also showed a beneficial effect of reducing BMI. Parameters assessed included 68/66 tender/swollen joint count, CRP, body surface area (BSA), Leeds enthesitis index, HAQ, and patient VAS for global health, pain, and fatigue. The percentage of patients with minimal disease activity increased from 29% to 54%. A 6-month follow-up of another group of patients also led to similar conclusions. A weight loss of more than 5% was a predisposing factor for achieving minimal disease activity and improved response to treatment with TNF-alpha inhibitors [26]. In the case of lupus, no reliable analyses have been conducted on the effect of weight loss on achieving minimal disease activity. A study is available on the effect of a low-glycemic index diet and a calorie-restricted diet on reducing Fatigue Severity Scale scores. The differences were statistically significant compared to the control group. The study involved women with systemic lupus erythematosus who were taking glucocorticoids [27].

Glucagon-Like Peptide-1 (GLP-1) analogues in rheumatology

GLP-1 is released after food ingestion to overcome postprandial hyperglycemia by increasing insulin secretion from β-cells and reducing glucose fluctuations and glucagon secretion. Incretin hormones inhibit β-cell apoptosis and induce their proliferation. Moreover, they also reduce gastrointestinal motility. Research has also emerged on the effects of this hormone on the brain. GLP-1 infusion during a fasting state in healthy, non-obese volunteers increased satiety and reduced the perception of hunger, as demonstrated by results correlated with reduced brain activation in the amygdala, caudate nucleus, insula, nucleus accumbens, orbitofrontal cortex, and putamen. GLP-1 analogues are becoming more and more popular and are used not only in the treatment of diabetes but also obesity. Such preparations include: liraglutide, semaglutide and tirzepatide [28,29,30] Based on the above, it can be concluded that this group of drugs, by promoting weight loss, positively affects disease activity in rheumatological conditions. However, reports are increasingly emerging regarding the effect of GLP-1 analogues on inflammatory responses.

The first studies on the use of this class of drugs in rheumatology are emerging. In GLP-treated patients, greater reductions in RA activity, pain, body weight, total cholesterol, and glycated hemoglobin were observed compared to the control group. Reductions in ESR, CRP, low-density lipoprotein (LDL) cholesterol, and triglycerides were also observed in the treated group. However, neither the reduction in acute-phase response markers nor the reduction in pain were

significantly correlated with weight loss, suggesting that these outcomes may be modulated by mechanisms beyond weight loss. Previous studies have shown that GLP-1 can reduce the secretion of several proinflammatory cytokines, suggesting a possible mechanism by which they may influence RA disease activity. However, this requires further study [31].

This phenomenon was studied using the effect of lixisenatide on the inflammatory response in human fibroblast-like synoviocytes. The drug was shown to inhibit the inflammatory response by reducing the expression of proinflammatory cytokines: TNF-alpha, IL-6, and interleukin-8 (IL-8), inhibiting matrix metalloproteinases, and blocking cellular signaling pathways, as well as reducing oxidative stress [32].

The literature also describes the case of a 57-year-old man with PsA who was taking adalimumab and then was treated with a GLP-1 analogue. Disease activity decreased, allowing for a reduction in the adalimumab dose. Pain was better controlled than with higher doses of the biologic. However, there are too few studies in PsA on a larger population to definitively conclude that this group of drugs directly affect the course of the disease [33].

In the case of lupus erythematosus, the number of studies on this topic is even smaller. A small study showed that GLP-1 analogues reduced BMI in this group of patients and were not associated with additional adverse effects. Slower disease progression was observed in the group of patients with lupus nephritis taking GLP-1 analogues than in the group taking SGLT-2 inhibitors [34,35]. A Phase 3 study of ixekizumab (anti-IL-17) with tirzepatide is currently underway in obese/overweight adults with active psoriatic arthritis or psoriasis to demonstrate the efficacy and safety of this drug combination not only for weight management but also for improving psoriatic disease [36].

Conclusion:

Obesity is a significant factor modifying the course of rheumatic diseases and their response to treatment. Excessive adipose tissue is not merely an energy reservoir, but also an active endocrine organ that, through the overproduction of proinflammatory adipokines and cytokines, exacerbates the chronic inflammation characteristic of RA, PsA, SLE, and other autoimmune diseases. Research data indicate that a higher BMI is associated with higher disease activity, poorer patient-reported outcomes, and greater difficulty in achieving minimal disease activity or remission. The most evidence concerns rheumatoid arthritis and psoriatic arthritis, where obesity likely significantly reduces the effectiveness of TNF-alpha inhibitors. In the case of tocilizumab and abatacept, studies suggest no effect of body weight on clinical response. However, the effectiveness of rituximab and belimumab appears to be relatively independent

of BMI. At the same time, even moderate weight loss can be considered to improve quality of life and increase the percentage of patients achieving minimal disease activity. Accumulating evidence also suggests that GLP-1 analogues may provide additional benefits not only through weight loss but also through potential direct anti-inflammatory effects. However, further randomized trials in larger populations are necessary to clearly assess the impact of body weight on the efficacy of biologic therapy and to determine optimal therapeutic strategies in this group of patients, including the use of GLP-1 analogues.

Disclosure

Author's Contribution

Conceptualization: Julia Agnieszka Michalak

Methodology: Julia Michalak, Klaudia Martyna Patrzykąt, Klaudia Elżbieta Niwińska, Julia Aleksandra Leśniak, Zofia Czaplińska-Paszek

Formal analysis: Julia Agnieszka Michalak, Anna Maria Zakrzewska, Kinga Popielarska, Aleksander Midera, Michał Borowski,

Investigation: Natalia Maria Leśniak, Aleksander Midera, Anna Maria Zakrzewska, Kinga Popielarska, Michał Borowski

Writing-rough preparation: Julia Agnieszka Michalak, Klaudia Martyna Patrzykąt, Natalia Maria Leśniak,

Writing-review and editing: Klaudia Elżbieta Niwińska, Julia Aleksandra Leśniak, Zofia Czaplińska-Paszek

Supervision: Julia Agnieszka Michalak

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