BIESZCZAD, Dominika, BARCZEWSKA, Magdalena, KOWALCZYK, Dominika, REIZER, Barbara, KRYSTEK, Klaudia, FELISIAK, Patrycja, ŚLAZYK, Dominik, BOTTO, Zofia, SWOJNÓG, Marzena, and ŚLIWA, Zofia. Lipedema: Current Understanding, Diagnosis and Multimodal Management Approaches. Journal of Education, Health and Sport. 2026;87:67421. eISSN 2391-8306.
https://dx.doi.org/10.12775/JEHS.2026.87.67421
https://apcz.umk.pl/JEHS/article/view/67421

The journal has had 40 points in Minister of Science and Higher Education of Poland parametric evaluation. Annex to the announcement of the Minister of Education and Science of 05.01.2024 No. 32318. Has a Journal's Unique Identifier: 201159. Scientific disciplines assigned: Physical culture sciences (Field of medical and health sciences); Health Sciences (Field of medical and health sciences).
Punkty Ministerialne 40 punktów. Załącznik do komunikatu Ministra Nauki i Szkolnictwa Wyższego z dnia 05.01.2024 Lp. 32318. Posiada Unikatowy Identyfikator Czasopisma: 201159. Przypisane dyscypliny naukowe: Nauki o kulturze fizycznej (Dziedzina nauk medycznych i nauk o zdrowiu); Nauki o zdrowiu (Dziedzina nauk medycznych i nauk o zdrowiu).© The Authors 2024;
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The authors declare that there is no conflict of interests regarding the publication of this paper.
Received: 13.12.2025. Revised: 20.12.2025. Accepted: 16.01.2026. Published: 17.01.2026.

Lipedema: Current Understanding, Diagnosis and Multimodal Management Approaches

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ABSTRACT

Background. Lipedema is a chronic and progressive fat disorder that affects ~11% of the female population. It is characterized by bilateral, disproportionate accumulation of subcutaneous adipose tissue predominantly in the lower body. Symptoms include pain, bruising, swelling, and subcutaneous nodules that are resistant to traditional interventions such as diet and exercise.

Aim. The objective of this review is to summarize recent evidence on the characteristics, pathophysiology, diagnosis and treatment of lipedema.

Materials and Methods. A literature search was conducted using the PubMed database. The inclusion criteria were "full free text" and English scientific articles, published between 2015 and 2025. A total of 74 records were found, of which publications were ultimately included in the review.

Results. Awareness of lipedema in the medical field is increasing, but its differential diagnosis still remains a challenge. Lipedema is often unrecognized or misdiagnosed as obesity or lymphedema.

Conclusion. This narrative review provides a deeper understanding of lipedema as a serious condition, discusses its pathophysiology and treatment options. The data reveal advances in knowledge, particularly in conservative and surgical treatment with a focus on improving quality of life. However, there is a lack of scientific evidence confirming the safety and efficacy of various treatment methods. Further research is required to ensure the safety and increase the efficacy of treatment for this complex condition known as lipedema.

Keywords: lipedema, pathophysiology, diagnosis, treatment

INTRODUCTION

Lipedema is characterized by disproportionate painful accumulation of subcutaneous adipose tissue (SAT) in areas such as the arms, hips, buttocks and thighs, sparing the hands and feet. However, progression of the disease may lead to poor lymphatic circulation (1). Once this occurs, the feet will also become edematous. Lipedema presents symmetrically on both sides and is associated with pain and easy bruising. Typically, there is joint hypermobility and limited mobility of the hands and feet (also known as the "cuff sign"). Patients report feeling of heaviness in the affected areas, with symptoms worsening throughout the day (2). Lipedema occurs almost exclusively in women.

Epidemiologic estimates from the sparse available data suggest an approximately 11% prevalence in the overall female population with a real prevalence potentially higher considering the frequent underdiagnosis (3). Men with lipedema have been reported in the literature only as case reports and tend to have conditions associated with higher estrogen and lower relative testosterone levels, such as male hypogonadism and liver disease (4). Lipedema usually develops during early adulthood due to hormonal changes (e.g pregnancy and menopause), stress, surgery (5). This serious condition is largely understudied and misdiagnosed as classical obesity, delaying appropriate treatment and worsening disease morbidity. Lipedema differs from obesity because it does not respond to diet and exercise. Early diagnosis and treatment are crucial, as the disease is progressive and can lead to immobility as well as a significant decrease in the quality of life (6). This review aims to provide a comprehensive and up-to-date overview of lipedema and also evaluate current treatment modalities ranging from conservative approaches such as compression therapy and manual lymphatic drainage to more invasive options like liposuction.

REVIEW METHODS

A systematic literature search was conducted in PubMed and Google Scholar to identify English scientific free full-text access articles on lipedema. Publications between 2015 and 2025 were included. The following search strategy was applied across databases with syntax adapted as needed: *("lipedema" OR "lipoedema") AND ("diagnosis" OR "treatment" OR "pathophysiology" OR "management") AND ("review" OR "clinical study" OR "observational study")*. Eligible studies included reviews, clinical trials, observational studies and larger case series focusing on the diagnosis, pathophysiology, treatment or clinical management of lipedema. Reference lists of included papers were additionally screened. Exclusion criteria involved: non-English publications, lack of free full text and studies published before 2015.

STATE OF KNOWLEDGE

In recent years, lipedema has gained increasing recognition among healthcare professionals and the general public, largely due to expanding online resources and growing medical interest in the condition. Although the exact cause of lipedema remains unknown, several hypotheses have been proposed. Current evidence suggests a multifactorial etiology involving genetic predispositions and hormonal influences, particularly estrogen, which may contribute to abnormalities in adipocyte growth and differentiation (7),(8). These changes are thought to coexist with microvascular and lymphatic dysfunction. Lipedema is therefore considered a heterogeneous, likely genetic disease that often manifests alongside female hormonal transitions. Additionally, emerging research

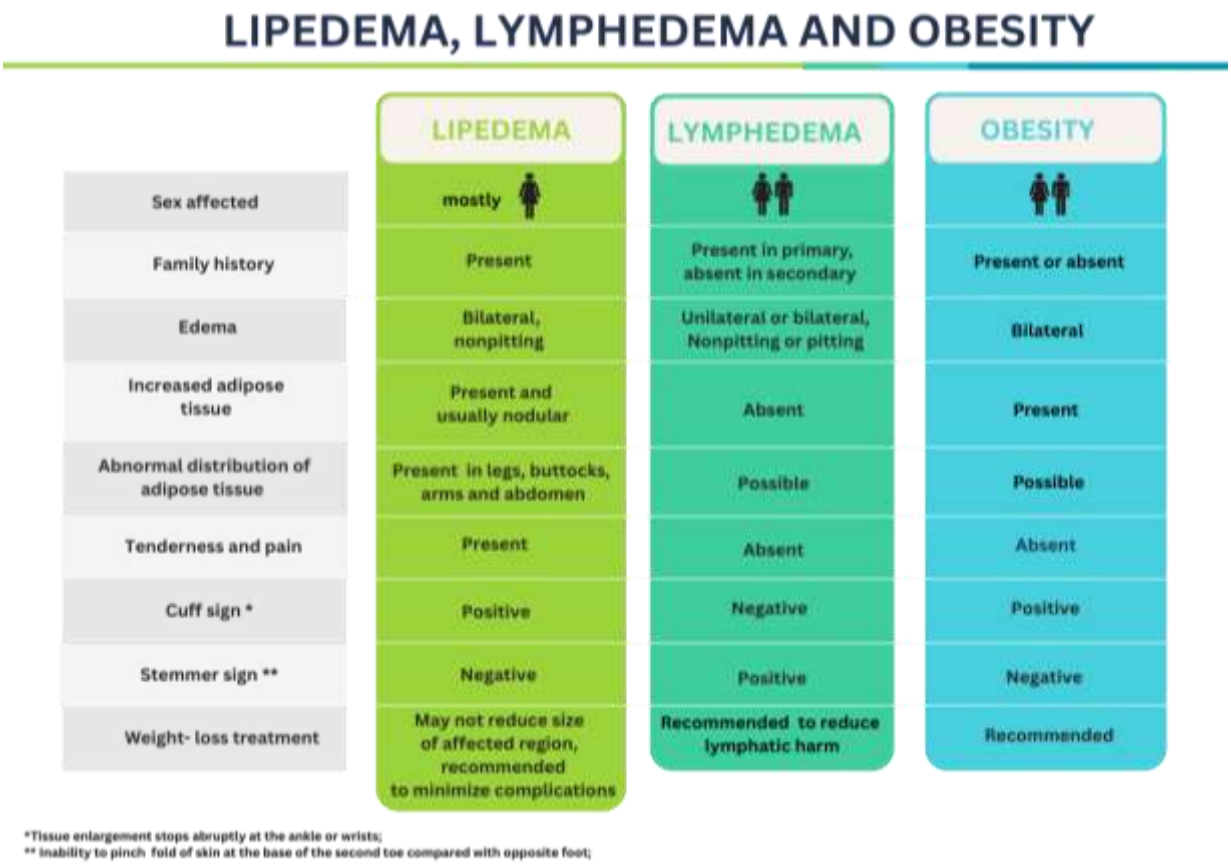
indicates that inflammation of peripheral nerves and disturbances in sympathetic innervation within SAT may contribute to neuropathy, with secondary adipocyte hyperproliferation (9).

Pathophysiology

Lipedema is thought to have a strong genetic basis, potentially following an X-linked dominant or autosomal dominant inheritance pattern. According to Paolacci et al., a self-reported positive family history is present in approximately 64% of affected women, which is consistent with other findings identifying lipedema among first-degree relatives (10). A cross-sectional study demonstrated that the prevalence of lipedema increases with rising body weight and body mass index (BMI). Obesity is considered an aggravating factor that exacerbates lymphatic dysfunction and edema, ultimately contributing to lymphatic overload (11). Notably, up to 50% of patients with lipedema are overweight or obese, yet they exhibit a relatively low prevalence of metabolic comorbidities such as diabetes, dyslipidemia and hypertension (5), (12). Lipedema typically manifests during periods of hormonal fluctuation, particularly at puberty and symptoms may lessen after menopause, suggesting an endocrine influence on disease progression. Additional mechanisms proposed in the pathogenesis of lipedema include increased vascular permeability, microvascular damage, excessive lipid peroxidation and disturbances in adipocyte metabolism with altered cytokine production (10). Inflammation of the peripheral nerves and sympathetic innervation abnormalities of the SAT may be responsible for neuropathy with adipocyte hyperproliferation as a secondary phenomenon. Obesity and polycystic ovary syndrome can exacerbate lipid edema. This is believed to be caused by increased concentrations of adipokines, tumor necrosis factor alpha (TNFα) and leptin, which often accompany these conditions associated with venous disease (13),(14). The most relevant differential diagnoses of lipedema include obesity and lymphedema. In cases of more advanced edema of the lower legs e.g chronic venous insufficiency, idiopathic cyclic edema should be considered (15).

Table 1 shows the differences between lipedema, lymphedema and obesity (1),(16).

Table 1.



Classification

There are many classification systems used to characterize fat distribution in lipedema.

It can be classified in 5 types depending on the fat distribution. Type IV is often combined with Type II or III.

• Type I: increased deposit of fat in gluteus, hips and thighs;

• Type II: lipedema extends to knees with a fat pad in the internal zone of the knees;

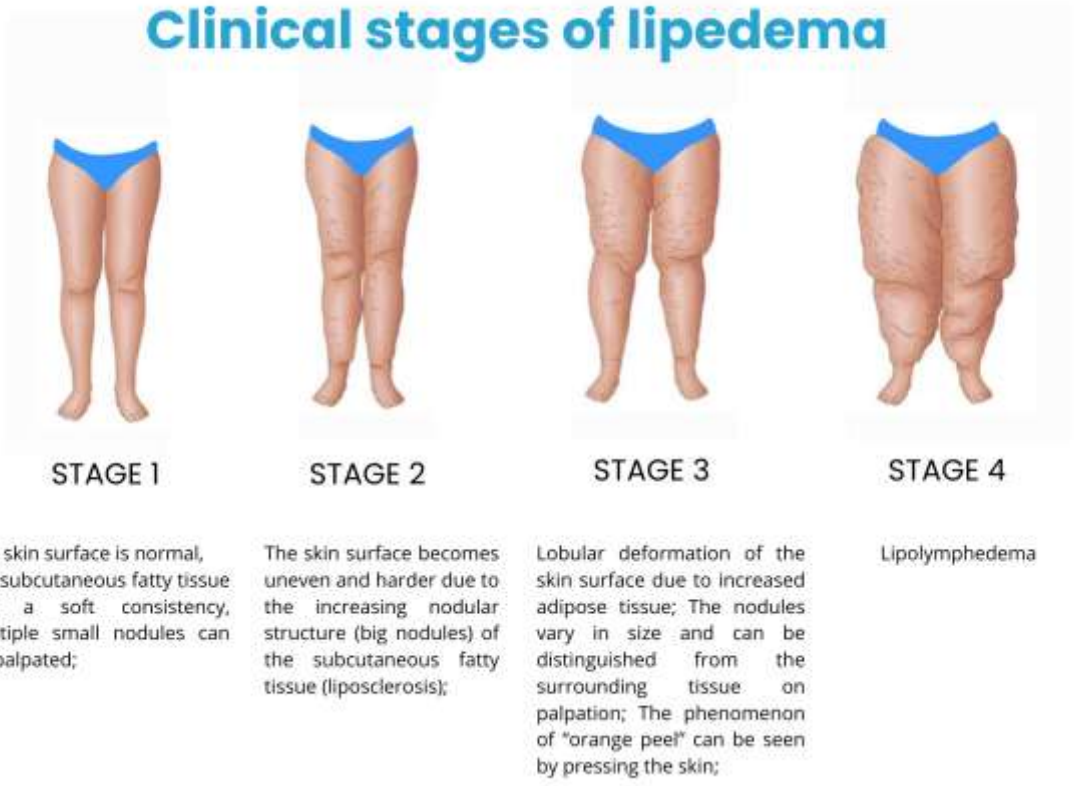
• Type III: lipedema extends from hips to ankles;

• Type IV: upper limbs are affected;

• Type V: only lower part of the legs are affected;

Based on inspection and palpation, lipedema can be classified in 4 clinical stages of severity (5). The symptoms and subjective degree of suffering are not necessarily correlated with the disease stage.

Figure 1 shows the stages of lipedema.



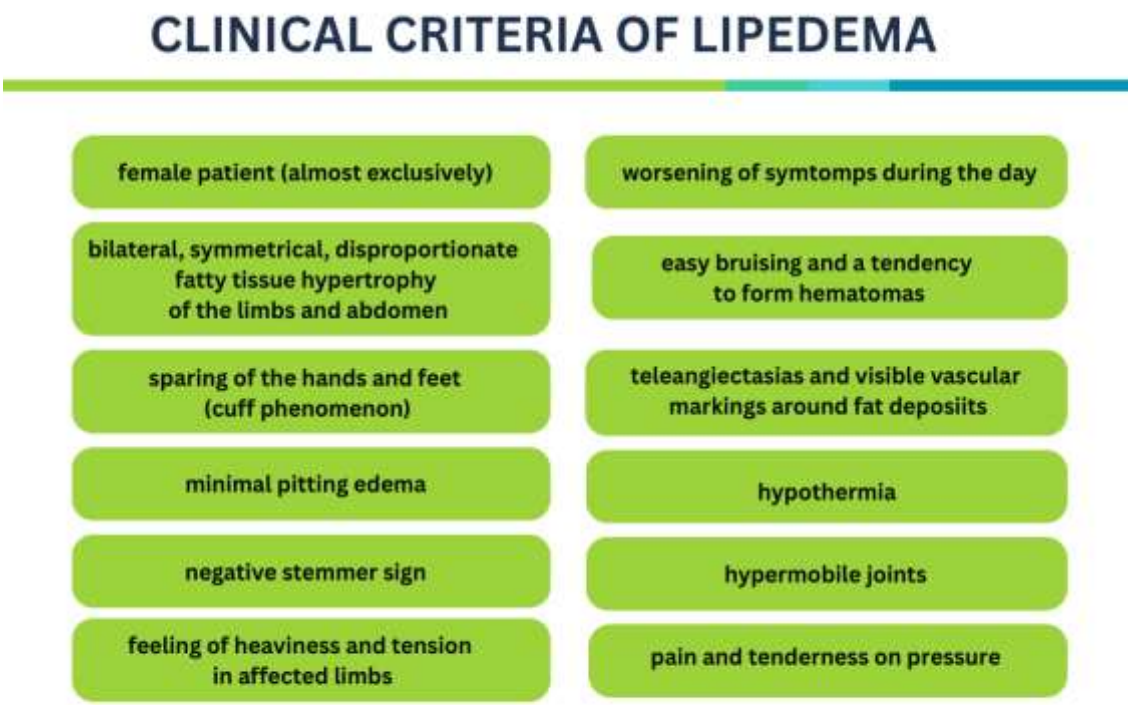
Diagnosis

A detailed medical history and a physical examination often allows for establishing the correct diagnosis of lipedema (1), (16), (17). After considering the possibility of lipedema, the medical interview should include an assessment of diet, physical activity patterns and the chronology of lipedema symptom development, with particular attention to hormonal changes in women, including puberty, pregnancy and menopause. It is also essential to document pain, easy bruising in the affected areas and any family history of similar features in female relatives (18). The guideline committee advises the use of defined criteria to make a medical diagnosis of lipedema. Clinical criteria for the diagnosis of lipedema, proposed by Wold et al, and amended by Herbst and Kruppa et al., are as follows by Figure 2. Initially, a visual examination should be performed to determine the disproportion between the fat tissue in the upper and lower body and the waist-to-hip ratio should be measured, which is also helpful in diagnosing abdominal obesity. Laboratory tests should be obtained to exclude heart, kidney, liver, thyroid (hypothyroidism), hormonal or edema-promoting disturbances such as secondary effects of medications (eg, calcium channel blockers, gabapentin and oral corticosteroids). Serum

selenium levels are often checked because selenium deficiency due to oxidative stress can lead to tissue injury by inflammation, apoptosis or necroptosis (19).

Ma et al., identified platelet factor 4 as a promising diagnostic marker of lymphatic malfunction that could help in diagnosing and clinically differentiating lipedema, lymphedema and obesity. Furthermore, it was found at higher levels in women with lipedema even if they were not overweight or obese. Thus, elevated levels of platelet factor 4 may provide evidence of underlying lymphatic structural and functional vasculature dysfunction in the pathogenesis of lipedema (20). It is not routinely used in practice as a diagnostic marker, but research continues on this topic. There are currently no imaging exams that can be used to definitively differentiate lipedema fat from non-lipedemadous adipose tissue. However, some imaging studies may be useful. Imaging tests such as ultrasonography, CT, or MRI can be used to study the skin and subcutaneous tissue. Ultrasonography can show thinner skin and increased thickness and hypoechogenicity of subcutaneous fat toward the medial calf and distal extremities (21). CT can show fatty hypertrophy in the lower extremities and MRI can show dilation of lymphatic vessels in the legs (18). Indirect lymphography, functional lymphatic scintigraphy and fluorescence microlymphography can be used to evaluate the structure and function of the lymphatic system (14).

Figure 2.



Treatment

The goal of treatment is to reduce pain and improve function and quality of life. Management of lipedema can be classified into conservative and surgical methods.

Conservative management should be delivered through a multidisciplinary approach and incorporate regular physical activity, compression therapy, psychosocial support, weight control strategies, patient education and guidance in self-management (22). Although the overall level of evidence remains limited, multiple therapeutic interventions have demonstrated potential clinical benefit. Dietary modification does not directly reverse lipedema-associated adipose tissue, which is characteristically resistant to weight loss. However, it plays an important role in supporting metabolic homeostasis and modulating systemic inflammation. Nutritional patterns characterized by a low intake of processed carbohydrates and added sugars, which contribute to reduced insulin secretion and adipogenic activity, are associated with the most beneficial outcomes. Additionally, minimizing food intake between meals may further enhance metabolic regulation (23). Additionally, Low Carb High Fat (LCHF) ketogenic diet may offer therapeutic benefit in the conservative management of lipedema, with reported reductions in body weight, BMI, pain and selected anthropometric parameters, alongside improvements in quality of life. However, given the limited number of available studies, these findings should be interpreted with caution and further research is required to confirm efficacy and long-term outcomes (24),(25). Pharmacological and supplemental approaches focus on reducing inflammation, edema, fibrosis progression and adiposity (26). Metformin shows potential to limit fibrosis development in adipose tissue, particularly in patients with metabolic syndrome or later-stage lipedema (27). Physical activity plays a key role in promoting venous and lymphatic circulation. Individuals with lipedema commonly demonstrate reduced muscle strength and may experience exacerbation of pain or edema in response to certain forms of exercise. Consequently, graded and individualized exercise programs are recommended. Aquatic therapy, in particular, appears beneficial due to the hydrostatic pressure of water, which provides uniform external compression, facilitates fluid mobilization and improves exercise tolerance and functional mobility (28). Compression garments play a crucial role in mitigating symptoms by providing support to adipose tissue and alleviating discomfort during physical activity. Both pneumatic compression devices and manual lymphatic drainage are frequently employed in the management of lipedema, with many patients reporting symptomatic improvement (29). Given that lipedema is primarily characterized by abnormal adipose tissue rather than pathological edema, the therapeutic benefit of lymphatic drainage beyond the reduction of orthostatic edema remains unclear. Additionally, there is a notable absence of randomized controlled

trials evaluating the efficacy of compression therapy in this population. Nonetheless, compression remains an essential component of treatment for overweight patients who have developed secondary lymphedema (30). Conservative treatments like manual lymphatic drainage, multi-layer short-stretch bandaging, and medical compression stockings can help relieve pain in lipedema patients and prevent associated edema. However, these methods do not reduce the pathological adipose tissue (31).

Liposuction is an established surgical option for lipedema, particularly when conservative treatments are inadequate or unsuccessful (32),(33). Among available interventions, liposuction that preserves lymphatic vessels using tumescent, water-assisted or laser-assisted techniques is the only modality capable of physically removing pathological adipose tissue, thereby improving mobility and reducing patient symptoms in the affected limbs (34),(35). There are few studies on whether weight gain after surgical treatment for lipedema leads to a recurrence of symptoms. Knowledge about the long-term outcomes of liposuction is also limited, but a published longitudinal study with data at 4, 8 and 12 years suggests that the pain-relieving effect persists despite weight gain (36). Patients with lipedema experience considerable psychosocial challenges, including appearance-related distress, depression, and eating disorders. These factors contribute to a diminished quality of life, elevated risk of suicide, impaired mobility, and social isolation. Pain, coexisting mental health conditions, and reduced physical capacity further compromise daily functioning. Provision of psychological support and the establishment of affirming clinical environments are essential to enhance patient well-being and to address issues of stigma and misdiagnosis (37). Effective disease management is essential and focuses on two primary objectives: first, alleviating subjective symptoms such as pain, and second, preventing the development of additional complications, including lipo-lymphedema, skin infections, psychological comorbidities, and mechanically induced complaints such as gait disturbances and joint deformities.

SUMMARY

In recent years, lipedema research has expanded beyond predominantly clinical observations to encompass molecular and cellular investigations, significantly advancing the understanding of this complex disorder. In light of the expanding understanding of genetic contributions to related adipose and lymphatic disorders, together with rapid progress in genomic screening technologies, the development of a dedicated genetic test for lipedema is increasingly warranted. Despite these advances, substantial gaps in knowledge remain. Many of the available data are preliminary and require further validation through well-designed and reproducible experimental approaches. An additional and long-standing limitation in lipedema research is the insufficient consideration of

coexisting obesity as a confounding factor. Addressing this issue is essential for the accurate interpretation of experimental and clinical data and does not necessitate a complete revision of existing knowledge, but rather a critical reassessment of unverified assumptions surrounding the disease. Therefore, further research is needed to advance the understanding of lipedema, establish specific and standardized diagnostic criteria, and elucidate the relationships between lipedema, lifestyle factors, comorbidities, and quality of life, all of which may contribute to reducing diagnostic delay. The primary goals of lipedema management include alleviation of lower extremity symptoms, reduction of functional limitations, and prevention of disease progression. Early and accurate identification of lipedema is essential in order to implement timely therapeutic interventions and reduce the risk of associated comorbidities. As etiology-based management strategies for lipedema are not yet available, effective therapeutic approaches should adopt a comprehensive and individualized framework. Such strategies must also address coexisting conditions, including venous and lymphatic edema, reduced levels of physical activity, obesity, and other pathological factors that may exacerbate the clinical manifestations of lipedema.

Disclosure

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All authors have read and agreed with the published version of the manuscript.

Funding
The study did not receive special funding.

Institutional Review Board Statement
Not applicable.

Informed Consent Statement
Not applicable.

Data Availability Statement
All relevant data are within the manuscript.

Acknowledgements
None.

Conflicts of Interest
The authors declare no conflict of interest.

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