FROM THE EXPERTS IN ENDOCRINOLOGY

2026 ENDOCRINE CASE MANAGEMENT: MEET THE PROFESSOR

ENDOCRINE SOCIETY

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ENDO 2026
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ENDOCRINE
CASE MANAGEMENT

ENDO 2026

ENDOCRINE SOCIETY

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The Society has earned an international reputation for excellence in the quality of its peer-reviewed journals, educational resources, meetings, and programs that improve public health through the practice and science of endocrinology.

Clinical Practice Chair, ENDO 2026
Ana Paula Abreu Metzger, MD, PhD

ISBN: 978-1-936704-72-9
eISBN: 978-1-936704-73-6

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ENDO 2026
OVERVIEW

OVERVIEW

Endocrine Case Management: Meet the Professor is designed to provide physicians with a concise, high-quality review of 40 common and rare endocrine disorders to help you keep your practice current. It features case-based clinical vignettes and rationales written by experts across all areas of endocrinology, diabetes, and metabolism.

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LEARNING OBJECTIVES

Endocrine Case Management: Meet the Professor enables learners to assess their knowledge of all aspects of endocrinology, diabetes, and metabolism.

Upon completion of this educational activity, learners will be able to:

- Recognize the clinical manifestations of endocrine and metabolic disorders and select among current diagnostic, management, and therapeutic options.
- Identify risk factors for endocrine and metabolic disorders and develop prevention strategies.
- Evaluate endocrine and metabolic manifestations of systemic disorders.
- Use existing resources on clinical guidelines and treatment recommendations for endocrine and related metabolic disorders to guide diagnosis and treatment.

TARGET AUDIENCE

Endocrine Case Management: Meet the Professor provides case-based education for clinicians seeking to improve patient care.

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As a CME provider accredited by the Accreditation Council for Continuing Medical Education, the Endocrine Society has a policy to ensure that the content of this educational activity is high-quality, balanced, independent, objective, and scientifically rigorous. The scientific content of this activity was developed under the supervision of the Endocrine Society's Annual Meeting Steering Committee.

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LAST REVIEW: May 2026

ACTIVITY RELEASE: June 13, 2026

ACTIVITY EXPIRATION DATE: December 31, 2028 (date after which this enduring material is no longer certified for *AMA PRA Category 1 Credits*™ and ABIM Medical Knowledge MOC points)

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COMMON ABBREVIATIONS IN MEET THE PROFESSOR

ACTH = corticotropin

ACE inhibitor = angiotensin-converting enzyme inhibitor

ALT = alanine aminotransferase

AST = aspartate aminotransferase

BMI = body mass index

CNS = central nervous system

CT = computed tomography

DHEA = dehydroepiandrosterone

DHEA-S = dehydroepiandrosterone sulfate

DNA = deoxyribonucleic acid

DPP-4 inhibitor = dipeptidyl-peptidase 4 inhibitor

DXA = dual-energy x-ray absorptiometry

FDA = Food and Drug Administration

FGF-23 = fibroblast growth factor 23

FNA = fine-needle aspiration

FSH = follicle-stimulating hormone

GH = growth hormone

GHRH = growth hormone–releasing hormone

GLP-1 receptor agonist = glucagonlike peptide 1 receptor agonist

GnRH = gonadotropin-releasing hormone

hCG = human chorionic gonadotropin

HDL = high-density lipoprotein

HIV = human immunodeficiency virus

HMG-CoA reductase inhibitor = 3-hydroxy-3-methylglutaryl coenzyme A reductase inhibitor

IGF-1 = insulinlike growth factor 1

LDL = low-density lipoprotein

LH = luteinizing hormone

MCV = mean corpuscular volume

MIBG = *meta*-iodobenzylguanidine

MRI = magnetic resonance imaging

NPH insulin = neutral protamine Hagedorn insulin

PCSK9 inhibitor = proprotein convertase subtilisin/kexin 9 inhibitor

PET = positron emission tomography

PSA = prostate-specific antigen

PTH = parathyroid hormone

PTHrP = parathyroid hormone–related protein

SGLT-2 inhibitor = sodium-glucose cotransporter 2 inhibitor

SHBG = sex hormone–binding globulin

T_3 = triiodothyronine

T_4 = thyroxine

TPO antibodies = thyroperoxidase antibodies

TRH = thyrotropin-releasing hormone

TRAb = TSH-receptor antibodies

TSH = thyrotropin

VLDL = very low-density lipoprotein

ADIPOSE TISSUE, APPETITE, OBESITY, AND LIPIDS

Lipedema at the Intersection of Endocrinology and Adipose Biology

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Educational Objectives

After reviewing this chapter, learners should be able to:

- Recognize lipedema as a distinct disorder of loose connective tissue under the umbrella of obesity, distinct from vascular disease–related lymphedema.
- Describe endocrinology and adipose biology relevant to the development and future treatment of lipedema.
- Discuss current and emerging treatment options to reduce morbidity from lipedema.

Significance of the Clinical Problem

What Is Lipedema?

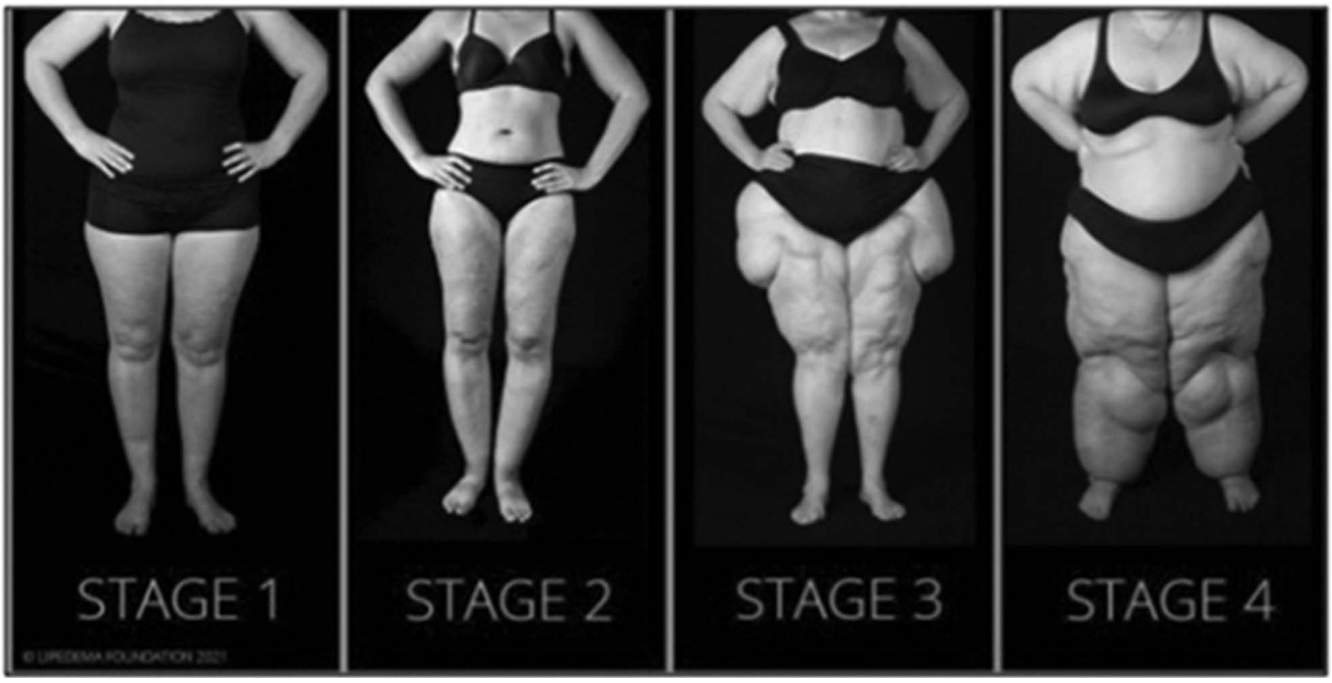
Lipedema is a chronic disorder of subcutaneous adipose tissue characterized by disproportionate fat accumulation in the limbs (usually the legs, sometimes the arms) and almost exclusively affects women. First described in 1940 by Allen and Hines, lipedema is distinct from general obesity and lymphedema, with morbidity decreasing quality of life.[1,2] In lipedema, excessive fat deposition causes symmetric enlargement of the legs and/or arms, often presenting as a columnlike leg shape, while hands and feet remain unaffected until late stages. A hallmark is that the affected fat tissue is painful to palpation and prone to bruising with minor trauma, indicative of capillary fragility. Unlike common obesity, the fat in lipedema is diet-resistant, meaning patients often report little improvement in limb size even after significant weight loss. This can help distinguish lipedema from simple obesity. Importantly, lipedema fat deposition is usually bilateral and symmetric, differentiating it from lymphedema, in which swelling is often unilateral.[3] In summary, lipedema is recognized as a loose connective tissue disease of fat, involving nodular, fibrotic fat accumulation on the hips, buttocks, and limbs, arising in the context of hormonal and developmental triggers (eg, puberty), and causing progressive disproportionate limb enlargement with pain.

Diagnostic Criteria

The diagnosis of lipedema is clinical, based on a thorough patient history, characteristic physical exam findings, and exclusion of other conditions. Formal diagnostic criteria were established by the US Standard of Care consensus,[4] which outlined key features for identifying lipedema in practice. There is no specific laboratory test or biomarker for lipedema; instead, clinicians rely on recognizing a constellation of signs and symptoms. Major criteria include: (1) a bilateral, symmetric distribution of abnormal subcutaneous fat, typically affecting the legs (from the hips down to the ankles) and sometimes the arms, with sparing of the feet and hands (producing an

abrupt cutoff or "cuffing" at the ankles or wrists); (2) a negative Stemmer sign, meaning the skin at the base of the toes and fingers can be pinched and lifted (in contrast to lymphedema, where this is usually impossible due to swelling); (3) little or no pitting edema on pressure, at least in early stages (any swelling in lipedema is nonpitting and generally minimal unless lymphatic compromise develops). Additional supportive criteria include pain, tenderness, and easy bruising in the affected fat tissue; patients often report that even slight pressure or minor bumps cause pain and bruising in the legs. Onset or worsening during hormonal changes, such as around puberty or pregnancy, is another common historical clue. Importantly, disproportionate fat distribution is a key feature; the lower body is much more enlarged than the upper body, sometimes accompanied by palpable fat nodules in the subcutis. In contrast, the trunk remains comparatively slender, and the feet are not swollen, distinguishing lipedema from generalized obesity or lymphedema. Patients typically do not experience significant improvement in limb size with calorie restriction or exercise (diet-resistant fat), which can aid in diagnosis. Clinical examination may also reveal textural changes in the skin, depending on the stage of the disease. In early stages, the skin may feel rubbery with fine nodularity, progressing to larger nodules and an irregular "mattress-like" texture in later stages as fibrotic nodules form. Formal staging systems grade the skin and tissue nodularity: stage 1, smooth skin, soft nodules; stage 2, uneven skin surface, large nodules (apple- to walnut-sized), fat thickening, and fibrosis; stage 3, large extrusion of fat tissue, indurated masses and lobules of fat; and stage 4, sometimes used to denote lipedema with accompanying lymphedema (lipo-lymphedema), as shown in the *Figure*. Additionally, lipedema is classified by type based on regional distribution: type I: buttocks/hips; type II: buttocks to the knees; type III: down to the ankles; type IV: arms; type V: calves. These staging classifications, illustrated in the *Figure*, help document the extent of disease for clinical and research purposes.

Figure. Stages of Lipedema According to Progression of Fat Accumulation and Changes to Skin and Lymphatic System



Adapted from the Lipedema Foundation, lipedema.org

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Prevalence

Lipedema is believed to be far more common than historically recognized, affecting a substantial percentage of the female population. Precise prevalence figures remain uncertain because of underdiagnosis and past confusion with obesity, but recent estimates suggest that roughly 11% (up to nearly 1 in 9) of adult women worldwide may have lipedema. In specific settings, even higher rates have been reported; for example, a German literature review reported that 6% to 8% of women in the general population could have lipedema, and 15% to 19% of women seen in specialty vascular clinics were diagnosed with lipedema.[5] In contrast, lipedema is exceedingly rare in men, with only isolated case reports, usually in men with extreme hormonal disorders, such as testosterone deficiency or estrogen excess, or liver disease.

Practice Gaps

- Lipedema is common among women seeking care for weight management or vascular medicine.
- Lipedema does not respond to medical or surgical weight-loss interventions. Cornerstones of therapy include compression and, in advanced cases, plastic surgery to remove affected adipose tissue.
- As a disorder of loose connective tissue, including adipose tissue, lipedema has important hormonal connections to its development and pathophysiology that endocrinologists should be aware of.

Discussion

Pathophysiology

The pathophysiology of lipedema is complex and generally involves abnormal expansion of subcutaneous adipose tissue, associated microvascular changes influenced by genetic predisposition, and environmental cues. Conceptually, this pathophysiology can be organized into vascular, endocrine, and adipose components, among others, for the purposes of this chapter.

Vascular Component

Despite the "edema" suffix, lipedema does not involve fluid accumulation in early stages. Unlike lymphedema, the hallmark of lipedema is disproportionate, symmetric accumulation of painful adipose tissue in the upper or lower extremities. Feet, hands, trunk, neck, and head are not affected.[4] The Stemmer sign, in which the examiner cannot pinch the skin of the toes, is negative in lipedema. By contrast, lymphedema stems from impaired lymphatic capillary drainage, often begins distally in the feet, and tends to be asymmetric. In this context, lipedema is often considered to have relative lymphatic sparing and minimal edema in early stages. The microvasculature in lipedema is compromised, leading to easy bruising and increased vascular permeability in fat, both of which indicate a microangiopathic state. Leaky capillaries may lead to bruising after minor trauma, as well as to pressure, heaviness, and pain on palpation. In later stages, secondary lymphedema can coexist, resulting in the condition termed lipo-lymphedema. The International Lipedema Consensus of 2020 proposed that chronic inflammation and tissue hypoxia in the enlarging fat are likely causes of the pain in lipedema, rather than fluid edema per se.[4] This insight shifts the treatment focus in lipedema beyond purely decompressive therapies toward anti-inflammatory and antiobesity strategies, among others. Vascular and lymphatic factors in lipedema appear secondary to adipose pathology but can progress as the disease advances, underscoring the importance of vascular and lymphatic care in later disease stages.

Endocrine Component

Lipedema development is closely associated with hormonal changes across the female life course.[6] Lipedema often appears or worsens during periods of major hormonal flux and weight change, classically

at puberty, sometimes during pregnancy, and frequently at menopause. A common hypothesis is that lipedema may be linked to estrogen surges during these transitions. Research suggests that lipedema adipose tissue may have an abnormal estrogen profile, including an imbalance in estrogen receptor alpha (ERα) and beta (ERβ) levels. Estrogen promotes the accumulation of adipose tissue in the gluteofemoral distribution, consistent with the lipedema fat distribution. One study reported reduced ERα and increased ERβ in adipose tissue from patients with lipedema, which could promote lipid accumulation in lower-body depots.[7] Progesterone and androgen levels have also been implicated in pregnancy and the menopausal transition, respectively, but direct evidence is lacking. Hormonal triggers are central to when lipedema manifests and flares, making it a quintessential example of an endocrine-influenced adipose disorder. Future research focused on the molecular endocrinology of lipedema could reveal targeted ways to interrupt this hormonal trigger cycle.

Adipose Component

At its core, lipedema is a disorder of adipose tissue within loose connective tissue. Adipocytes play the starring role in pathophysiology, with both hypertrophic and hyperplastic changes documented.[7,8] Histological studies of lipedema fat have demonstrated adipocyte hypertrophy (enlarged fat cells) and adipocyte hyperplasia (increased number of fat cells) in the affected areas.[9] These observations imply that the subcutaneous tissue in lipedema can expand markedly in volume, partly by each fat cell storing more lipid and swelling, and partly by recruiting or differentiating new fat cells. This contrasts with typical obesity, where hypertrophy tends to predominate.[10] Lipedema fat often forms a nodular texture; microscopically, nodules of fibrotic connective tissue surround groups of adipocytes, especially in later stages. Notably, in early-stage lipedema, there may be relatively little fibrosis; the skin remains soft, and the fat, although nodular, is not hardened. Consequently, stage 1 lipedema skin feels smooth even though underlying fat nodules can be palpated like small peas. As the condition progresses to stages 2 and 3, fibrosis increases in the subcutis: collagen deposition and scarlike tissue form around larger clumps of fat, causing the overlying skin to indent and dimple (mimicking cellulite, but more extreme). By stage 3, sizable lobules of fat develop, suspended by fibrous septa, leading to deforming overhangs of tissue around the knees or arms. Despite fibrosis of the fat in lipedema, skin elasticity often remains relatively good (until very advanced disease), which is why compression and surgery can achieve good skin retraction. Importantly, pure lipedema (stages 1-3) does not cause the dermal fibrosis or peau d'orange texture seen in lymphedema. The skin of lipedema, aside from the nodules underneath, can be pinchable and is not woody or indurated. Only when lymphedema overlaps does the skin begin to thicken. Another component of the adipose tissue change is the inflammatory cell infiltrate. Research indicates that lipedema fat depots have a distinct immune cell composition compared to both normal and obesity-related fat. There is evidence of a higher proportion of M2 macrophages (alternatively activated, anti-inflammatory macrophages) in lipedema tissue, whereas obesity-related fat tends to have more proinflammatory M1 macrophages. This M2 predominance might sound counterintuitive, since lipedema is painful (one would expect inflammation to cause pain), but it likely reflects a chronic, unresolved state of tissue remodeling. Some theorize that the pain in lipedema is due to nerve irritation within the expanding fat. Indeed, hypoxia and mild inflammation can sensitize nerves. One hypothesis is that as fat hypertrophies, small nerve fibers in the tissue are stretched or compressed, leading to a neuropathic pain element. There is also evidence of abnormal adipose stem-cell function in lipedema. A study found that adipose-derived stem cells from patients with lipedema have impaired adipogenesis in vitro and altered adipokine expression. Intriguingly, these differences potentially could serve as biomarkers of lipedema or even as therapeutic targets. From a life-course perspective, once lipedema fat forms,

it tends to be resistant to loss. Calorie restriction or bariatric surgery may reduce overall weight, but often spares the lipedema fat. This suggests a fundamental dysregulation of adipose metabolism in lipedema tissue. Several groups have postulated defects in catecholamine or hormone receptors on lipedema fat cells, or localized adipose hypoxia, leading to insulin resistance in that tissue, but these hypotheses remain under investigation. Finally, unlike fibrotic lymphedema tissue, lipedema adipose tissue remains "soft" fat (especially in the early stages), composed of a loose connective tissue matrix that holds water and fat; thus, the term "loose connective tissue disease" is used for lipedema. The fat is not scarred in the way of postradiation lymphedema tissue; rather, it is an overgrowth of otherwise normal fat cells and supporting stroma. Understanding the adipose component is key to developing treatments; this is why liposuction can dramatically help by physically removing pathological fat cells. In summary, lipedema involves *both* adipocyte hypertrophy and hyperplasia, leading to a significant expansion of subcutaneous fat mass with distinctive nodular fibrosis and inflammatory changes. This adipose pathology, influenced by hormones and genetics, is the central driver of the disease, around which vascular and lymphatic issues revolve.

Treatment Options

There is currently no cure for lipedema, but a variety of treatments can manage symptoms, improve function, and slow disease progression. Optimal care is usually multidisciplinary, combining medical management, nutritional guidance, physical therapies, and sometimes surgery. The overall goals are to reduce pain and swelling, restore mobility, prevent or treat lymphedema, and address the psychosocial impact. As a provider, it is important to set expectations: while treatments can significantly improve quality of life, they typically do not eliminate all lipedema tissue (except through surgical removal), and ongoing maintenance is needed. The main treatment modalities in the standard of care are discussed in the following sections.

Medical Management

Medical management of lipedema focuses on treating symptoms and related conditions, since no pill or injection can yet specifically remove lipedema fat. Pain management is one aspect. Many patients benefit from analgesics, including nonsteroidal anti-inflammatory agents. If neuropathic pain is suspected, medications such as gabapentin or duloxetine may be tried under physician guidance. Treating coexisting issues is also crucial. For example, if venous insufficiency or varicose veins contribute to edema, medical or interventional treatments for those conditions (such as compression or vein ablation) should be provided. Likewise, managing hypothyroidism, vitamin D deficiency, or any metabolic disorders can help overall tissue health. A cornerstone of medical management is compression therapy, which is considered "medical" because it is prescribed and fitted by health care providers. Consistent use of graduated compression stockings or tights (usually class II, 20-30 mm Hg or higher) helps reduce daily swelling, supports the tissues, and can diminish pain and bruising by limiting microvascular leak. Finding the right compression garment often involves working with a specialist.[11] When lipedema overlaps with fluid edema, involvement of a certified lymphedema therapist is extremely beneficial. Certified lymphedema therapists are trained in complete decongestive therapy, which includes manual lymphatic drainage massage, multilayer compression bandaging, exercises, and skin care education.

Surgical Treatment

Another important treatment decision is whether to pursue surgical treatment, specifically, liposuction.[5] Liposuction for lipedema is generally categorized as a surgical intervention, but because it is one of the few ways to actually remove the pathological fat, it is part of the standard care discussion. Typically, conservative measures are tried for at least 6 to 12 months, and if the patient

still has significant pain or functional limitation, liposuction is considered. Liposuction (ideally using the tumescent technique with microcannulas by surgeons experienced in lipedema) can dramatically improve symptoms: it permanently removes lipedema fat cells, resulting in reduced limb size, improved contour, and often much less pain and bruising. However, liposuction usually requires multiple sessions, as each limb may require 2 to 3 operations staged months apart, and it carries risks typical of any surgery, including fluid shifts and potential lymphatic injury. In experienced hands, liposuction is safe and effective. For many patients with moderate-to-severe lipedema, liposuction is the closest thing to a "definitive" treatment. Some countries, such as Germany, have officially recognized liposuction as part of lipedema therapy and even cover it under insurance in certain cases. In the United States, insurance coverage varies and can be challenging.

Nutrition

There is no single scientifically proven "lipedema diet" at this time. However, several dietary approaches have been advocated by experts and patients, including anti-inflammatory diets, lower-carbohydrate or ketogenic diets, intermittent fasting, gluten-free or allergy-elimination diets, and general weight-loss diets. The common theme across these nutritional strategies is promoting a healthy, balanced diet that the patient can sustain over the long term. In conclusion, nutrition is a cornerstone of lipedema management, aimed at optimizing body composition and reducing inflammation. As Tomada noted,[6] proper nutrition and weight management can preserve mobility and function and potentially slow disease progression.

After decades of relative neglect, patient advocacy, scientific curiosity, and clinical urgency are driving progress in treating lipedema.[12] Ultimately, the vision for the future is one where lipedema is no longer an "orphan" condition, but a well-characterized medical entity with a range of effective treatments.

Clinical Case Vignettes

Case 1

A 52-year-old woman with class 2 obesity (BMI = 36 kg/m^2) presents with worsening bilateral leg swelling. She reports menarche at age 11 and a distant history of leg swelling during puberty. She had difficulty maintaining her weight throughout young adulthood, with "big legs." Over the past 3 years, the swelling has markedly increased, causing discomfort when putting on clothes and walking. She endorses occasional hot flashes and misses periods every 2 to 3 months.

On physical examination, the patient appears well and has a gynoid pattern of obesity. Palpation of her lower extremities reveals pearl-like nodules along her thighs and hypersensitivity to touch, with associated discomfort.

Laboratory test results are normal, including those from a basic metabolic panel, a complete blood cell count, B-type natriuretic peptide, and measurement of TSH and vitamin B_{12}.

What additional testing (if any) is needed to diagnose lipedema in this patient?

A. No additional testing needed
B. Vascular scintigraphy to exclude lymphedema
C. Coagulation tests
D. LH, FSH, and estradiol measurement

Answer: A) No additional testing needed

The diagnosis of lipedema relies on a thorough patient history, characteristic physical findings, and the exclusion of other conditions. At this time, there are no specific laboratory findings with diagnostic utility in lipedema, although exciting research is ongoing. Consequently, the lab-based findings listed in Answers B, C, and D can be suggestive but not diagnostic. Vascular scintigraphy is an invasive test that uses nuclear dyes to assess reverse flow in the lymphatic vasculature and can help exclude lymphedema. However, it is not diagnostic of lymphedema.

Case 2

An 18-year-old woman describes a history of larger legs that accelerated during puberty. Despite multiple attempts at diet-induced weight loss and, more recently, medication-induced weight loss, she has been unable to reduce her leg size and has relied on wearing larger boots to conceal her appearance. She now seeks assistance for her enlarged legs, which are limiting her mobility.

On physical examination, the patient's vital signs are within normal limits. Her BMI is 32 kg/m^2, and she has a gynoid fat distribution with excess skin and dimpling. The Stemmer sign is positive. She has no pain to touch or nodularity in her legs.

Given her likely stage of lipedema, what treatment options should be *avoided* at this point in her disease course?

A. Dietary strategies
B. Complete decongestive therapy
C. Complete lymphatic therapy
D. Surgical removal

Answer: D) Surgical removal

This patient likely has class I or II lipedema based on the provided description and is unlikely to have had the disease for more than 10 years, given its onset at puberty. As such, there is ample opportunity to implement a layered approach to prevent progression of lipedema, including dietary strategies (Answer A) and mechanical therapies (eg, complete decongestive therapy [Answer B] and complete lymphatic therapy [Answer C]). Surgical removal (Answer D) of lipedema adipose tissue should be reserved for patients with significant pain or mobility limitations or advanced disease, but guidelines are evolving.

Case 3

A 37-year-old woman with hypertension and type 2 diabetes (hemoglobin A_{1c} = 6.3% [45 mmol/mol]), controlled for 3 years on metformin, presents to the endocrinology clinic with concern about leg swelling and oozing. She endorses increased leg mass since menarche and, over the past 4 years, has gained significant weight that has been unresponsive to diuretic treatment or GLP-1 receptor agonist therapy. She now reports pain and painful, pearl-like nodules in her legs, along with oozing from her heels.

On physical examination, the patient has a BMI of 41 kg/m^2 and stable vital signs. Her light-touch sensation remains intact on monofilament testing, and there are mild abrasions on the heel and plantar surfaces with clear fluid oozing. She has excess skin on her thighs and decreased mobility, with difficulty standing from her chair.

Given the patient's presentation with more advanced lipo-lymphedema, what initial therapeutic option should be considered?

A. Diuretic therapy
B. Complete decongestive therapy
C. Another trial of GLP-1 receptor agonist therapy
D. Surgical removal

Answer: B) Complete decongestive therapy

This patient with lipo-lymphedema would benefit from a layered approach, beginning with mechanical decompression therapies, such as complete decongestive therapy (Answer B), before proceeding to medical or surgical therapies. A vascular medicine evaluation will be important to assess the extent of lymphedema and its effect on lipedema progression. Following vascular evaluation, diuretic therapy (Answer A) may be considered. GLP-1 receptor agonist therapy (Answer C) is generally unable to remove lipedema fat once fibrosis has formed. Surgical removal (Answer D) may be considered after initial mechanical and medical therapies.

Key Learning Points

- Lipedema remains underdiagnosed in the endocrinology community as a disease of loose connective tissue, including adipose tissue,

often overlapping with obesity, characterized by abnormal lower-body fat distribution and pain, and primarily affecting women.

- Lipedema sits at the intersection of endocrinology, given the important hormonal cues to its development, and adipose biology, given the integral role of adipocytes in its development.
- Lipedema does not respond to conventional weight-loss treatments, including GLP-1 receptor agonists or dietary interventions, and treatment often requires a layered approach that includes mechanical, medical, and ultimately surgical interventions.
- Ongoing research aims to identify blood-based and imaging biomarkers that could inform future therapies.

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ENDO 2026
COLOR GALLERY

ADIPOSE TISSUE, APPETITE, OBESITY, AND LIPIDS

Lipedema at the Intersection of Endocrinology and Adipose Biology

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Adapted from the Lipedema Foundation, lipedema.org