

Lipedema: a clinical review

What is known about lipedema as of October 2026: definition, prevalence, mechanisms, diagnosis and treatment – with references to the international consensus, clinical guidelines, randomised trials and systematic reviews.

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AUTHOR

Anastasia Ignatieva

Chief surgeon, plastic and reconstructive surgeon



REVIEWER

Yevhenii Kolesnykov

Plastic and reconstructive surgeon · Doctor of Medical Sciences, Professor

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KEY POINTS

- **Lipedema** is a chronic disease of subcutaneous adipose tissue that occurs almost exclusively in women: symmetrical, disproportionate enlargement of the legs (often also the arms) with the feet and hands spared, pain on pressure and easy bruising. ICD-11 code: **EF02.2**.
- The true prevalence is unknown: estimates range from less than 1% to 12% of women, because studies use different criteria.
- The diagnosis is **clinical**. There are no validated laboratory tests, genetic tests or imaging methods to confirm it; BMI distinguishes lipedema poorly from obesity, so assessment of the waist-to-height ratio is advised.
- Compression, physical activity, nutrition and psychological support reduce symptoms, but lipedema-affected fat responds poorly to weight loss. There are no approved medicines.
- In the randomised LIPLEG trial (The Lancet, 2026), pain at 12 months was substantially reduced in 68% of patients after liposuction versus 8% on conservative therapy; adverse events were more frequent after surgery.
- Ukraine has no epidemiological studies of lipedema and no publications on it in PubMed.

Definition

Lipedema is a chronic disease of subcutaneous adipose tissue that almost always occurs in women. It is characterised by symmetrical enlargement of the legs, and often also of the arms, disproportionate to the rest of the body, with the feet and hands spared, pain and tenderness of the tissue on pressure, and a tendency to bruise ^[1]. The 2026 international consensus defines lipedema as a distinct chronic disease rather than a complication of obesity ^[1].

In the International Classification of Diseases, 11th Revision (ICD-11), lipedema has its own code, **EF02.2 “Lipoedema”**, in the chapter on diseases of the skin (disorders of the dermis and subcutaneous tissue). The WHO describes it as a diffuse, non-pitting fatty “swelling”, most often of the legs, thighs, pelvic region and upper arms, which may be mistaken for lymphoedema ^[2].

History and terminology

The disease was first described in 1940 by Edgar Allen and Edgar Hines of the Mayo Clinic as a “syndrome characterized by fat legs and orthostatic edema” ^[3]. In 1951, Wold, Hines and Allen published a description of 119 cases and formulated the clinical features that still underpin diagnosis: almost exclusively female sex, symmetrical enlargement of the legs, sparing of the feet, easy bruising and pain ^[4].

The literature uses the spellings *lipedema* (American) and *lipoedema* (British), and German-language sources use *Lipödem*; some authors propose the term *lipohyperplasia dolorosa* to emphasise that the principal feature is painful overgrowth of adipose tissue rather than oedema ^[25]. A separate code first appeared in ICD-11, which came into effect on 1 January 2022; ICD-10 had no separate code for lipedema ^[2].

The most comprehensive current clinical documents are the German S2k guideline of 2024 ^[5], the US standard of care of 2021 ^[6] and the 2026 international Delphi consensus of the Lipedema World Alliance, in which experts from 19 countries agreed on 59 statements on definition, diagnosis and treatment ^[1].

Prevalence

The true prevalence of lipedema is **unknown**. The 2026 consensus cites estimates ranging from less than 1% to 12% of women ^[1]. The spread reflects the fact that studies use different criteria and different methods of selection:

- a German review from 2020 estimated the prevalence at approximately **10%** of the female population ^[7];
- in the Lipödem-3 study of 62 working women, signs of lipedema of any degree (including mild lipohypertrophy without pain) were found in **39%**, and moderate or severe forms in **9.7%** ^[8];
- an online screening of 253 women in Brazil indicated a high probability of lipedema in **12.3%** ^[9];
- at the same time, a British dermatology centre recorded only 67 cases over 15 years, which points to underdiagnosis rather than rarity of the disease ^[10].

On average, about **18 years** pass from the onset of symptoms to diagnosis; in a Dutch survey, patients had consulted an average of 2.8 doctors before the diagnosis was made ^[11]. In men, lipedema is rare and occurs mainly in the setting of hormonal disorders ^[1].

Causes and mechanisms

The cause of lipedema has not been definitively established ^[1], ^[16]. The best-supported factors are hormonal and hereditary.

Hormonal changes. The disease almost always begins or worsens during periods of hormonal change. In a study of 707 American women, symptoms appeared during puberty in 48.0% and during pregnancy in 41.2% ^[12]. The most widely held hypothesis links lipedema to an imbalance of oestrogen receptors in adipose tissue and a local (paracrine) excess of oestrogen ^[15], ^[16].

Heredity. A family history of lipedema is present in 60–80% of patients ^[13]. Pedigree analysis is consistent with sex-limited autosomal dominant inheritance ^[10]. A genome-wide association study in the UK Biobank identified 18 genomic loci

associated with the lipedema phenotype, including loci near the genes *VEGFA* and *GRB14-COBLL1* ^[14]. No single “lipedema gene” has been found, so the disease is probably genetically heterogeneous ^[13].

Tissue changes. In thigh biopsies from women with lipedema, adipocytes are enlarged, and cell size increases with stage; the tissue contains more collagen (interstitial fibrosis) and more macrophages, predominantly with an M2-like phenotype ^[17]. Unlike classic obesity, in which a pro-inflammatory M1 response predominates, lipedema shows a different inflammatory profile, and metabolic function is often preserved; for this reason, some researchers regard lipedema as a distinct model of subcutaneous adipose tissue expansion ^[16]. MRI shows an increased sodium content in the skin and subcutaneous tissue compared with women without lipedema ^[18].

Lymphatic system. Lymphatic transport in the legs is often slowed in lipedema: in lymphoscintigraphy of 117 patients, clearance from the foot region was impaired in 79.5–87.2% ^[19]. However, lipedema is not lymphoedema: the oedema is usually non-pitting, with a negative Stemmer sign ^[1]. In later stages and in the presence of concomitant obesity, secondary lymphoedema (lipolymphoedema) may develop ^[5].

The consensus specifically emphasises that lipedema is **not a consequence of overeating or obesity**, and that lipedema-affected fat is largely resistant to dieting ^[1].

Clinical presentation

Typical features according to the 2026 consensus ^[1]:

- symmetrical, disproportionate accumulation of subcutaneous fat on the legs and/or arms with a relatively slim trunk;
- the feet and hands are not affected – above the ankles and wrists the tissue seems to “stop abruptly” (a “cuff”);
- pain and tenderness to touch and pressure;
- easy bruising;

- non-pitting oedema; usually a negative Stemmer sign;
- poor response of the affected areas to weight loss.

In the largest American survey (707 women with lipedema and 216 controls), the features most strongly associated with lipedema were swelling in hot weather (odds ratio 66.8), easy bruising (26.2), altered gait (15.5), flu-like symptoms (13.0), joint hypermobility (12.9), skin that is cool to the touch (12.2), varicose veins (11.3) and fatigue (9.6). The arms were affected in 70.3% of women, and 52.2% noticed no improvement with diet and exercise ^[12].

Comorbidities. In a Spanish study of 1001 women with lipedema, 63.4% had vitamin D deficiency, 47.2% venous insufficiency and 43.1% migraine. Compared with the general population, venous insufficiency was 5 times more common, urinary incontinence 4.9 times and migraine 3.9 times; hypertension and diabetes, by contrast, were less common ^[20]. Joint hypermobility was reported by 44% of the patients surveyed ^[21].

Psychological burden. Lipedema reduces quality of life: in a Dutch survey, the overall quality-of-life score was 59.3, compared with a national average of 74.9 for women ^[11]. Depression and appearance-related distress independently impair patients' functioning ^[22], and late diagnosis adds to the burden of the disease ^[1].

Stages and types

The stages describe the appearance of the skin and subcutaneous tissue ^[5], ^[6]:

Stage Features

I	Smooth skin, evenly thickened subcutaneous layer, small nodules
II	Uneven skin surface, larger nodules
III	Large lobules of tissue, overhanging folds, distorted contours, especially around the knees and on the inner thighs

Types by location are described separately: buttocks and hips; from the hips to the knees; from the hips to the ankles; arms; predominantly the lower legs. Types may

occur in combination [5].

Importantly, according to the 2026 consensus, the stages **do not reflect the severity** of symptoms and are of limited clinical value: pain may be severe in stage I and moderate in stage III [1].

Diagnosis

The diagnosis of lipedema is **clinical**: it is based on the history, inspection, palpation and the exclusion of other causes of limb enlargement [1], [5]. There are no validated imaging methods, blood tests or genetic tests to confirm lipedema [1].

Anthropometry. In lipedema, the body mass index (BMI) overestimates the degree of obesity, because a substantial proportion of body mass is located in the legs. The consensus recommends routine assessment of the waist-to-height ratio (WHtR) and the waist-to-hip ratio together with BMI [1]. In a German study, according to WHtR, 45.5% of patients had normal weight, 15.2% were underweight and only 17.3% had obesity; the authors concluded that BMI alone is unreliable in lipedema [25].

Ultrasound. Ultrasound makes it possible to measure the thickness of the skin and subcutaneous tissue and to exclude venous disease. In a Brazilian study of 89 women, measurement over the anterior surface of the lower leg (pretibial) best distinguished lipedema [23]. A systematic review of 32 studies (1154 patients) describes characteristic findings on ultrasound, lymphoscintigraphy, CT, MRI and DEXA, but concludes that the diagnostic accuracy of all methods remains limited [24].

Differential diagnosis. Lipedema is most often confused with obesity and lymphoedema; venous insufficiency, lipohypertrophy without pain, Dercum disease and other lipodystrophies, hypothyroidism, and oedema of cardiac or renal origin should also be excluded [5], [6].

Feature	Lipedema	Obesity	Lymphoedema
Sex	Almost exclusively women	Both	Both
Distribution	Symmetrical, legs and/or arms	Generalised, often abdominal	Often asymmetrical, one limb

Feature	Lipedema	Obesity	Lymphoedema
Feet and hands	Not affected	May be enlarged	Often swollen
Pitting on pressure	Usually absent	Absent	Present in early stages
Stemmer sign	Usually negative	Negative	Often positive
Pain, bruising	Typical	Not typical	Uncommon
Response to weight loss	Weak in affected areas	Reduction throughout the body	Independent of weight

Table sources: [1], [5], [6].

Course and prognosis

Lipedema is a chronic disease, but it does not progress in every patient [1]. In a prospective follow-up of 100 patients over a mean of 4.6 years, the condition remained stable in 62%, worsened in 28% and improved in 10%; progression was associated with increases in BMI and WHtR [26]. Weight control guided by the waist-to-height ratio is therefore one of the goals of treatment [1].

Treatment

Lipedema is managed by a multidisciplinary team; the aims of treatment are to reduce pain, oedema and restriction of movement, to improve quality of life and to prevent progression [1].

Conservative therapy

According to the consensus, the basis of conservative treatment is lifestyle and dietary modification, compression and physical activity; complete decongestive therapy (CDT) is not always required in its full form [1].

- **Compression and CDT.** In a randomised trial of 33 women with severe lipedema, exercise combined with CDT reduced limb volume and pain to a greater extent than exercise combined with intermittent pneumatic compression or exercise

alone [27]. In a small randomised trial, compression stockings combined with pneumatic compression reduced leg circumference and pain [28].

- **Physical activity.** A systematic review of 6 studies (115 women) showed that exercise training reduces pain and limb volume and improves quality of life and function, particularly in combination with compression [29]. The consensus advises aquatic exercise, walking and yoga [1].
- **Nutrition.** In a randomised trial of 70 women, a 1200 kcal low-carbohydrate diet for 8 weeks reduced pain by 1.1 points (95% CI 0.3–1.9), and this reduction was independent of weight loss [30]. After 7 months of a low-carbohydrate, high-fat diet, weight decreased by 12.9% and pain on the visual analogue scale (VAS) from 4.6 to 3.0 [31]. However, a systematic review of 9 studies (269 women) rated the risk of bias as moderate or high, and its findings do not allow any specific diet to be recommended [32].
- **Psychological support** is part of the recommended set of measures [1].

Conservative methods reduce symptoms, but lipedema-affected fat is largely resistant to dieting [1].

Drug therapy

There are no approved medicines for the treatment of lipedema, and the consensus finds no evidence in favour of any drug therapy [1]. GLP-1 receptor agonists and dual GLP-1/GIP receptor agonists (semaglutide, tirzepatide) are being actively studied: in an online survey of 2719 women with lipedema, those taking such drugs reported less pain and swelling and a better general condition [34]. These are self-reports without a control group; a direct effect on lipedema itself has not been demonstrated, and these drugs are appropriate for their approved indications, namely obesity and diabetes [33], [35].

Bariatric surgery

Bariatric surgery also produces substantial weight loss in women with lipedema, but the core symptoms of lipedema – pain and disproportion of the lower body – do not

improve consistently after it ^[36]. The consensus notes that lipedema persists after bariatric surgery and that obesity and metabolic disorders should be treated before surgical treatment of lipedema ^[1].

Liposuction

The randomised LIPLEG trial (11 centres in Germany, published in *The Lancet* in 2026) compared liposuction with conservative therapy in 410 patients with stage I–III lipedema and leg pain of at least 4 out of 10. At 12 months, pain had decreased by at least 2 points in **68%** of patients after liposuction (190 of 278) versus **8%** in the conservative therapy group (10 of 132); odds ratio 26.2 (95% CI 13.1–52.5). Quality of life was also better after surgery. Adverse events occurred in 47% of patients after liposuction and in 21% of those on conservative therapy; serious adverse events occurred in 8% and 3%, respectively ^[37].

Other data:

- a meta-analysis of 10 studies (901 patients) showed a significant reduction in pain both at 6 months and later ^[38]; a meta-analysis of 20 studies showed improvement in quality of life, pain, tenderness, bruising, heaviness and walking, with rare severe complications ^[39];
- in an analysis of 519 liposuctions in 178 patients, the rate of postoperative adverse events was 2.89% ^[40];
- in long-term follow-up, the improvement persisted at 4, 8 and 12 years; complaints did not increase between years 8 and 12 ^[41], ^[42];
- liposuction under tumescent local anaesthesia did not impair lymphatic transport ^[19].

The consensus advises considering lymph-sparing liposuction, performed by surgeons with extensive experience in treating lipedema, after obesity and metabolic disorders have been treated ^[1]. In July 2025, Germany's Federal Joint Committee (G-BA) added liposuction for lipedema to the statutory health insurance benefit catalogue regardless of stage, provided that at least 6 months of continuous

conservative therapy have not relieved symptoms; where lipedema is accompanied by obesity of a certain degree, obesity is treated first [43].

Lipedema in Ukraine

There are no epidemiological studies of lipedema in Ukraine. As of 30 October 2026, no publications on lipedema with a Ukrainian affiliation or data from Ukraine were found in PubMed [44], and Ukraine is not represented among the experts from 19 countries in the 2026 international consensus [1]. If international estimates are applied to the female population of Ukraine, the number of women with lipedema could be in the hundreds of thousands, but this is only an extrapolation, not a measured value.

Open questions

- Why lipedema develops and which molecular mechanisms trigger the disease [1], [16].
- The true prevalence based on uniform diagnostic criteria [1].
- Objective biomarkers and imaging methods of proven accuracy [24].
- The optimal extent of conservative therapy and the role of specific diets [32].
- Long-term randomised data on liposuction: the main LIPLEG results cover 12 months after surgery [37].
- Whether drugs (in particular GLP-1/GIP receptor agonists) can affect lipedema itself, rather than only body weight [33], [35].

About this review

Methods. This review was prepared on the basis of a PubMed search (query “lipedema OR lipoedema” in the title or abstract) as of 30 October 2026. Priority was given to international consensus statements and clinical guidelines, randomised trials, systematic reviews and meta-analyses, and large cohort studies. Every figure has been checked against the abstract or the full text of the primary source.

Updates. We revise this review at least once every six months and after the publication of important studies. The revision history is given below.

Errors and suggestions can be sent to inbox.lipedema@gmail.com; corrections are recorded in the revision history.

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