Lipoedema – myths and facts Part 2

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Summary
Lipedema as a disease is associated with numerous myths. In this small overview of the myths surrounding lipedema, we throw a critical eye on popular statements regarding the disease; We have found that statements made in scientific publications decades ago have been repeated over and over again without criticism. These statements have become part of the general knowledge for lipedema patients and lipedema self-help groups. In the first part of our presentation we focussed on critically reviewing two popular myths about lipedema. We found that there were no scientific evidence for the following statements: "Lipedema is a progressive disease", and "Lipedema negatively affects mental health". In this our second contribution on the myths surrounding lipedema, we focussed on the edema aspect; i.e. on the so-called "edema in lipedema" and the subsequently recommended therapy – manual lymph drainage. Myth #3: Lipedema is primarily an "edema problem", and manual lymph drainage is thus an essential standard form of therapy, which must be conducted regularly! This statement also contradicts our daily experiences with this specific subset of patients to a high degree. Simultaneously we also established through extensive literature research, that there is no evidence for this concept. There is actually no indication that any form of relevant edema is present in lipedema patients, i.e. edema in the sense of fluid retention. There is also no scientific evidence that this barely measureable (in most cases entirely absent) edema is reponsible for the complaints of lipedema patients. There is thus no basis for the prescription of long-term and regular manual lymph drainage for treating this "edema". Lipedema is much more than just fat and painful legs! We must thus leave behind some of the old therapeutic methods, for which there is no scientific evidence and which furthermore directly contradict our clinical experiences. The comprehensive treatment of lipedema should thus consider all aspects of the disease, not only the immediately obvious such as observable changes and reported symptoms. Lipedema therapy must focus on the treatment of somatic complaints as well as on the psychosocial and sociological aspects of this complex disease, as outlined in our first contribution. The presentation of a comprehensive therapeutic concept for lipedema patients will be the subject of the last part of our short series on lipedema. We find new paths when we have the courage to walk down them – this is also true for the treatment of lipedema!

Schlüsselwörter
Lipödem, Ödem, Manuelle Lymphdrainage, Wissenschaftliche Evidenz

Zusammenfassung
In dieser kleinen Übersichtsreihe über die Mythen des Lipödems werfen wir einen kritischen Blick auf populäre Statements zum Lipödem; Statements, die vor Jahrzehnten schon Eingang in wissenschaftliche Publikationen gefunden haben und seither unkritisch und stetig wiederholt werden; Statements, die dadurch inzwischen zum selbstverständlichen Wissensallgemeingut von Lipödempatientinnen und vor allem auch von Lipödem-Selbsthilfegruppen geworden sind. Im ersten Teil unserer Darstellung haben wir uns kritisch mit zwei populären Mythen über das Lipödem auseinandergesetzt. Hierbei haben wir festgestellt, dass sowohl für das Statement „Das Lipödem ist eine progrediente Erkrankung" als auch für das Statement „Ein Lipödem macht psychisch krank" keine wissenschaftliche Evidenz vorliegt. In diesem zweiten Beitrag über die Mythen des Lipödems fokussieren wir uns auf den Ödemaspekt, auf das „Ödem im Lipödem" und die hieraus erfolgte therapeutische Konsequenz – die Manuelle Lymphdrainage. Daher: Mythos 3: Das Lipödem ist in erster Linie ein „Ödem-Problem"; daher ist die Manuelle Lymphdrainage essenzielle und regelmäßig durchzuführende Standardtherapie! Auch dieses Statement widerspricht in hohem Maße unserer seit Jahren bestehenden täglichen klinischen Erfahrung mit diesem speziellen Patientengut. Gleichzeitig haben wir im Rahmen unserer umfangreichen Literaturrecherche festgestellt, dass es keine Evidenz für diese Sichtweise gibt. Tatsächlich gibt es keinerlei Hinweis darauf, dass beim Lipödem ein relevantes Ödem – Ödem im Sinne von Flüssigkeit – vorliegt. Ebenso fehlt jegliche wissenschaftliche Evidenz dafür, dass dieses kaum (bzw. meist nicht) vorhandene Ödem für die Beschwerden der Lipödempatientinnen verantwortlich ist. Der regelmäßigen und dauerhaften Verordnung von Manuellen Lymphdrainagen mit dem Ziel der „Ödembeseitigung" fehlt daher jede Grundlage. Das Lipödem ist weit mehr als nur dickere, schmerzhafte Beine! Darum müssen wir manche der alten therapeutischen Pfade verlassen, Pfade, für die es keine

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wissenschaftliche Evidenz gibt, Pfade, die darüber hinaus auch unserer klinischen Erfahrung widersprechen. Eine umfassende Therapie des Lipödems sollte daher auch all jene Aspekte berücksichtigen, die nicht so offensichtlich sind wie das Augenscheinliche und das vordergründig Geäußerte. Lipödem-Therapie muss neben der Behandlung der somatischen Beschwerden auch auf die bereits in unserem ersten Beitrag beschriebenen psychosozialen und gesellschaftlichen Aspekte dieses komplexen Krankheitsbildes fokussieren. Die Vorstellung eines umfassenden Therapiekonzeptes für Lipödempatientinnen wird Inhalt im letzten Teil unserer kleinen Lipödemreihe sein. Neue Wege entstehen, in dem wir sie gehen – dies gilt auch für die Therapie des Lipödems!

Introduction

Lipoedema is far and away more than fat painful legs! Nevertheless – and this really has to be emphasised – not every fat leg is a case of lipoedema.

Each year at the Földi Clinic in Hinterzarten – the European Centre for Lymphology – we treat some 3000 patients with a diagnosis of lipoedema, offering both inpatient and ambulatory care. The vast majority of these patients come to our clinic with ideas and expectations that have been nourished not only by the media but also by their doctors: ideas about lipoedema that are a far cry from the scientific evidence and expectations that are often beyond the bounds of feasibility.

Myths about lipoedema are plentiful: statements have found their way into scientific thinking and consequently into patient know-how. We have already discussed two of these myths in the first part of our short series on lipoedema (1):

- Myth 1: Lipoedema is a chronic progressive disease
- Myth 2: Lipoedema negatively impacts mental health

In this contribution on the myths surrounding lipoedema we will address the oedema aspect (the supposed "oedema" in "lipoedema") and the subsequently recommended treatment with manual lymphatic drainage.

- Myth 3: The main problem of lipoedema is "oedema": regular manual lymphatic drainage is therefore essential standard treatment.

This statement seems to be lymphological dogma. Many national and international publications and lipoedema internet portals consider manual lymphatic drainage to be the essential standard therapy for lipoedema (2–5). Herpertz proposes "outpatient treatment for lipoedema, as a rule, once or twice a week with 45-minute sessions or 60-minute sessions in severe cases" (6). One of the websites frequently visited by women with lipoedema states that patients with lipoedema "usually require long-term treatment with manual lymphatic drainage" (7).

This consensus on the recommended therapy rests on the firm conviction that the main problem of lipoedema is "oedema" and that first-line treatment must target this aspect. In the Deutsche Ärzteblatt, Meier-Vollrath et al. wrote that "the aim of treatment is to combat the oedema" (8).

This dogma can be attributed to a false perception of lipoedema originating from the name given to the condition by E.V. Allen and E.H. Hines when they first described it in 1940, a name that is unfortunate from today's point of view. Examining the original articles from 1940 and 1951 more closely, we get the impression that they have to a large extent been misinterpreted. "Edema" [US spelling] tended to play a minor role in the two publications. The first paper gave an in-depth account of the pathophysiology of orthostatic oedema, concluding that "This is the situation in cases of lipedema" and again, "Near the end of a day of activity some edema may be demonstrable" (9). After the authors had studied 119 patients with lipoedema, oedema still seemed to play an insignificant role. In their second publication in 1951, they wrote, "Particularly at the end of the day, there may be some evidence of edema, although the evidence is not great enough to explain the patient's statement relative to the degree of swelling which has occurred as a result of orthostatic activity" (10).

In both publications, the mental health of the patients with lipoedema played a much greater role than "edema", which was mentioned only in passing at the time. In fact, only 24% of the 119 participants in this very first study on lipoedema actually had orthostatic oedema ("minimal to mild pitting edema"), while 29% suffered from an "associated neurosis" (11). We have already addressed the relevance of these psychological aspects in the opening article of this short series "Lipoedema – myths and facts" in the obesity special issue of this journal (12).

> By definition, oedema is an accumulation of fluid.

Clinical examination reveals the classic pitting appearance of the soft tissues. We often perform a high-resolution ultrasound scan on our patients with lipoedema; we examine the soft tissues using an 18.6 megahertz transducer and recently we have been using a MoistureMeter to measure the degree of moisture present. Neither of these methods demonstrated relevant fluid accumulations in the tissues of our patients. And last but not least, the findings on clinical inspection and ultrasound scan are identical in patients with lipoedema and patients with pure lipohypertrophy (painless disproportionate increase in adipose tissue).

► Figure 1 shows a patient with lipoedema, ► Figure 2 is the ultrasound scan of her thighs (the left and right thighs were identical). There are no abnormalities apart from widening of the subcutaneous tissue; in particular, there is no evidence of fluid.

► Figure 3 shows a patient with lipohypertrophy, ► Figure 4 is the ultrasound scan of her thighs (here, again, the left and right thighs were identical). The clinical picture and ultrasound images are virtually the same as those of the patient with lipoedema.

The patient in ► Figure 5 has three manifest clinical conditions: lymphoedema of the distal lower leg and forefoot; lipoedema that is restricted to the thigh and proximal leg; and morbid obesity with a body mass index (BMI) of 48 kg/m2.

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Fig. 1 Patient with lipoedema

▶ Figure 6 demonstrates the sustained pitting present in lymphoedema (lower circle) and the non-pitting nature of the soft tissues in the area of lipoedema (upper circle).

▶ Figures 7 and ▶ 8 show the ultrasound scans of this patient. Figure 7 (distal right lower leg) presents a typical ultrasound image of stage 2 lymphoedema with partial separation of the soft tissue structures, thickened dermis, and fluid in the tissues (small arrows). Figure 8 (proximal thigh) shows the typical ultrasound appearance of lipoedema with an unremarkable dermis, thickening of the subcutaneous tissues, and no evidence of fluid in the soft tissues.

At most, a small minority of our patients with "true" lipoedema ("true" meaning that we have excluded other oedema components, such as cardiac or lymphogenic oedema) present with relevant oedema on clinical examination – usually in the hot summer months.

Continuing with our historical review of lipoedema, we find that this condition almost vanished into oblivion after the two original publications by Allen and Hines (1940 and 1951). There were at most a few unsystematic individual case reports on lipoedema or conditions with painful adipose tissue in the 1960s and 70s (13,14). The topic was again called to attention in 1980, when Schmitz published an article entitled "Lipoedema – the fat leg in the healthy woman" in the journal Gynäkologie (15). However, the role of oedema – oedema in the sense of fluid accumulation – remained of no material significance! In 1982 Brunner wrote, "The underlying pathology consists of a disorder in the distribution pattern of the subcutaneous fat" and again "The fat layer has a soft consistency and does not allow pitting even over the tibia" (16). To Gregl (1987), lipoedema was a "mucoid pseudo-oedema". "In contrast to cardiac and dystrophic oedema, pressing over lipoedema does not cause pitting" (17). Examining his patients with lipoedema, Rudkin (1994) found only "1/4 + edema in the pretibial area" (1/4 + refers to an American oedema classification and means "hardly pitting") (18).

Fig. 3 Patient with lipohypertrophy

> It is clear that "oedema" plays only a minor role in lipoedema, or none at all.

Even so, doctors treating lipoedema usually regard this barely demonstrable or non-existent oedema to be the cause of the symptoms. Cornely justified his "lympho-

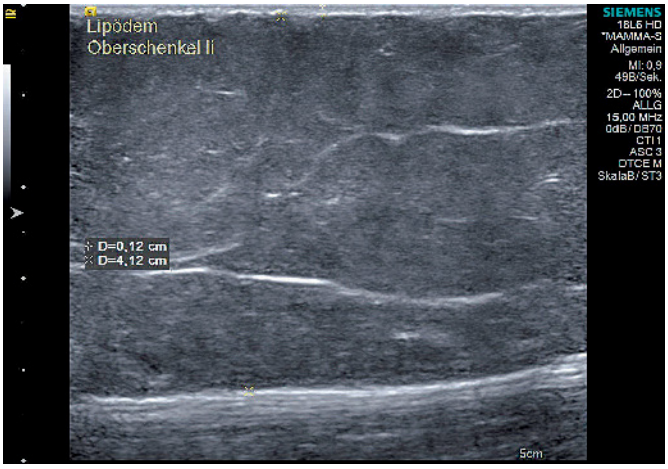

Fig. 2 Ultrasound scan of the thigh in the patient with lipoedema (left and right thighs were identical)

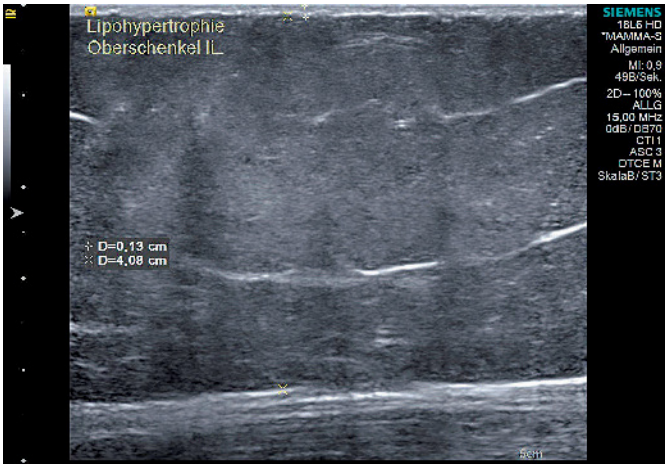

Fig. 4 Ultrasound scan of the thigh in the patient with lipohypertrophy (here, too, the left and right thighs were identical)

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Fig. 5 Patient with lymphoedema of the distal lower leg and forefoot and lipoedema restricted to the thigh and proximal lower leg

logical liposculpture" (the surgical aspiration of fat or liposuction) on the basis of "structural drainage insufficiency", a concept that he (and Marsch) developed (19). According to Cornely, "lipoedema leads to an overproduction of lymphatic fluid in the arms and legs, which is experienced as tenderness" and elsewhere there is "flooding of the adipose tissue with lymph, increasing the tension in the thickened extremities" (20).

> This concept is totally unsupported by any scientific evidence!

This "flooding of the adipose tissue" cannot be demonstrated either clinically or by ultrasonography.

Other imaging procedures such as computed tomography and lymphoscintigraphy have not revealed any fluid in the soft tissues or significant dysfunction of the lymphatic drainage in patients with lipoedema (21–23).

Nor is there any convincing histological evidence pointing to oedema in the adipose tissue. Kayserling examined the histology of the adipose tissue in patients with lipoedema and concluded that „The overall picture is one of a localised increase in adipose tissue consistent with lipomatosis" and further "The typical picture shows largely unremarkable adipose tissue". The only abnormalities seen were "isolated foci of fat necrosis" and increased numbers of macrophages in the interstitial tissue. "The macrophages typically express anti-CD 68" (24).

Both fat necrosis and CD 68-positive macrophages support the hypothesis that inflammatory and hypoxic processes are responsible for the pain in lipoedema. Laboratory tests on patients with lipoedema also argue in favour of this interpretation. The glutathione status in the red blood cells (RBC) and malondialdehyde (MDA) in the plasma have been studied as biomarkers of oxidative stress. MDA levels in patients with lipoedema were clearly higher than in healthy patients and investigation of the RBC glutathione yielded similar results (25).

Strössenreuther, who examined several histological studies of lipoedematous tissue in the course of his dissertation, did not use any terms that would indicate the presence of "oedema" in the lipoedema or increased fluid in the adipose tissue (26).

But even renowned lipoedema experts see this barely demonstrable or absolutely non-existent oedema as the cause of the symptoms. For example, in 2007, Schmeller wrote, "The oedema causes pain from tension, pressure, and touch" (27) and, in a later publication, "The extent of this accumulation, and not the absolute fat volume, is the major reason for sensitivity of the tissue to pressure and touch" (28). Rappich expressed himself in virtually the same way when he concluded that the "increased capillary permeability which leads to orthostatic oedema" is "...responsible for the increased sensitivity of the tissues to pressure and touch" (29).

Quite apart from the fact that there is no clinical, imaging or histological evidence to support this view, anyone reading the above statements will immediately ask the following question: If oedema is the cause of pain in patients with lipoedema, why do patients with cardiogenic oedema or lymphoedema have no pain at all or only very mild discomfort?

In these last two types of oedema, both clinical and ultrasound examinations clearly demonstrate fluid in the tissues.

Looking at the attitudes to the problem in other European countries, a similarly critical approach to the "oedema" in lipoedema has been taken in both the United Kingdom and the Netherlands. The authors of the Dutch 2014 lipoedema guideline wrote, "Lipedema is an unfortunate term as it evokes the idea of swelling due to fluid accumulation. However, it refers to swelling – in a sense of an increase in volume – due to increased fat tissue" (30). In consequence, the authors of the current Dutch guideline have not listed "oedema" in the criteria that define lipoedema in the first place.

The fact that the "oedema" part of lipoedema has nothing to with true oedema in the sense of fluid accumulation is also reflected in the latest British lipoedema guidelines from 2017: "the word lipoedema means 'fat swelling'" (31).

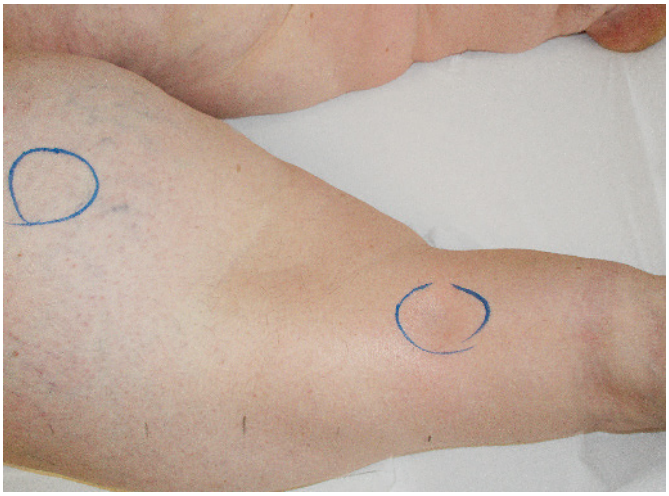

Fig. 6 Sustained pitting of lymphoedema (lower circle) and the non-pitting nature of the soft tissues in the area of lipoedema (upper circle)

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Why is the question of oedema so important? Why is clarification of this issue of immense practical significance?

As we said earlier, many national and international publications consider regular manual lymphatic drainage to be the standard treatment for lipoedema. This approach is based on the belief that oedema is responsible for the pain experienced in this condition. However, if no oedema (at least, no relevant oedema) can be demonstrated in patients with lipoedema, where is the rationale for prescribing manual lymphatic drainage?

Before prescribing manual lymphatic drainage in cases of lipoedema, two questions need to be answered unequivocally:

1. Is there any scientific evidence that this barely demonstrable or non-existent oedema is the cause of the patient's symptoms?
2. Is there any scientific evidence that manual lymphatic drainage improves the symptoms of our patients with lipoedema by its drainage effects?

In the light of today's scientific knowledge, the answer to both of these questions is an emphatic "No!"

In our lymphology outpatient department, we are confronted daily by women with lipoedema urgently demanding manual lymphatic drainage. We are often referred patients with lipoedema who have been prescribed manual lymphatic drainage twice or even three times a week for many years. It has to be emphasised that this treatment makes no sense from either a medical or economic perspective. Many of these patients claim most emphatically that lymphatic drainage does them good, but "doing someone good" is not at all the same as being medically necessary.

Manual lymphatic drainage quite possibly has quite different effects that have little to do with decongestive therapy but which are experienced as pleasant by the patients.

A large majority of our patients with lipoedema have mental health issues that require treatment: these symptoms are completely unrelated to the lipoedema. Many of our lipoedema patients also suffer from depressive disorders, anxiety, and eating disorders. We confirmed this finding in a pilot study at our clinic, which we presented in the opening article of this short series on lipoedema (32).

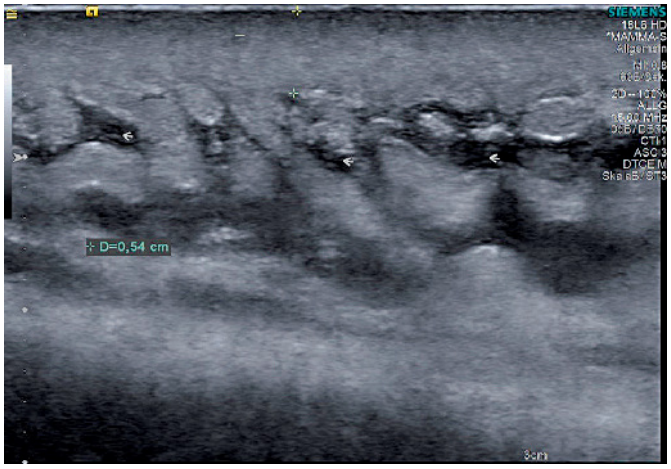

Fig. 7
Distal right leg of the patient shown in Figures 5 and 6: typical ultrasound image of stage 2 lymphoedema with partial separation of soft tissues, thickened subcutaneous tissue and fluid in the tissues (small arrows)

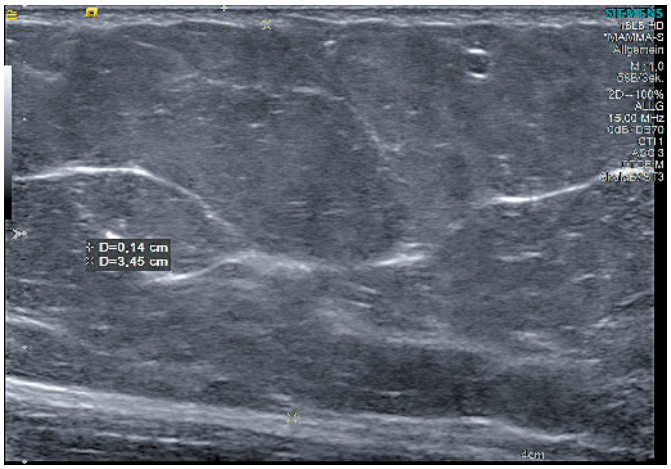

Fig. 8
Proximal thigh of the patient shown in Figures 5 and 6: typical ultrasound image of lipoedema with unremarkable dermis, thickened subcutaneous tissue, and no evidence of fluid in the soft tissues

Based on our results, we suggest that there are other aspects of manual lymphatic drainage that the patients find pleasant and positive: aspects of the massage itself, effects that have more to do with the reduction of stress and exhaustion than with decongestive therapy. Available data on the effects of massage therapy on psychological symptoms such as anxiety and depression are consistent with this view (33–35). In addition, we can assume that the patients' appreciation of manual lymphatic drainage lies in the touch and personal attention, in the contact between therapist and patient. And, last but not least, having treatment prescribed means that lipoedema has been recognised as a disease. Patients with lipoedema have often had a gruelling odyssey before their symptoms were finally taken seriously, with the subsequent diagnosis and treatment of lipoedema.

Psychological, psychosocial, and societal factors have a considerable impact on the symptoms of our patients with lipoedema. Even so – and make no mistake about it – lipoedema is primarily a somatic condition.

Lipoedema always develops from lipohypertrophy, but only in a very small number of those affected. It is not yet clear why some women (men are very rarely affected) develop pain in the adipose tissue, and the underlying pathology of this symptom is still subject to speculation.

Besides the oedema hypothesis presented earlier, widely differing ideas about the cause of pain abound in the scientific literature. Brenner summarised most of the suggestions in a review article (36).

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There is, however, a consensus that the pain is sited in the subcutaneous adipose tissue of the limbs.

As patients have usually experienced pain for more than six months at the time of diagnosis, this means that it can be termed chronic.

As a rule, the causes of chronic pain are degenerative processes in the musculoskeletal system (e.g. osteoarthritis), ischaemia (e.g. peripheral arterial disease), neuropathies (e.g. after a stroke, diabetic polyneuropathy), cancer, and inflammatory conditions (e.g. rheumatoid arthritis).

Looking at the literature on expanding subcutaneous fat, we find that two main processes are discussed: inflammation and hypoxia. For example, Pou et al. say that the subcutaneous adipose tissues "appear to be associated with chronic inflammation" (37) and Rutkowky et al. declare, "Adipose expansion results in tissue hypoxia" (38). Kayserling's findings, which we mentioned earlier, also seem to point in this direction. Crescenzi et al. very recently published their work comparing the adipose tissue of patients with lipoedema with a control group of women without lipoedema. They found an increase in the sodium content of the skin in the patients with lipoedema ("skin sodium is elevated in women with lipedema") and again, "Skin sodium accumulation is an emerging hallmark of inflammatory diseases" (39).

To date, inflammatory changes as a "chronic low-grade state of inflammation" (40) and hypoxia offer the most plausible models to explain the pain in these lipoedema patients. Therapeutic approaches to lipoedema should therefore always focus on these possible causes of pain.

Conclusions

There is no evidence that oedema plays a relevant role in the condition of lipoedema, still less that it is responsible for the pain experienced by the patients. Manual lymphatic drainage should therefore be prescribed only for a limited number of sessions when there is clinical evidence of oedema. This might be the case in the hot summer months when orthostatic oedema could be a problem. There is absolutely no basis for prescribing regular weekly sessions of manual lymphatic drainage in cases of lipoedema!

As stated at the beginning of this article, lipoedema is far and away more than fat painful legs!

We must therefore leave some of the old paths of thought which lack any scientific evidence and which contradict our clinical experience.

Comprehensive treatment of lipoedema should therefore take into account all those aspects which are not as immediately obvious as the observable changes and reported symptoms. In addition to alleviating the somatic symptoms, lipoedema therapy must also focus on the other aspects of this complex clinical picture, namely:

- a thorough work-up
- treatment of the pain
- the psychological vulnerability of women with lipoedema
- weight gain
- self-acceptance in an era obsessed with the body image of skinny models

In the final instalment of our short series on lipoedema, we will present a comprehensive therapeutic approach to this condition.

New paths are made by walking along them – and that is also true for the treatment of lipoedema.

Conflict of interest

The authors declare that they have no conflicts of interest.

Ethical guidelines

No studies on humans or animals were carried out in the preparation of this manuscript

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