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Reprint

Diabetic Peripheral Neuropathy

Adjuvant Homeopathic Treatment Enhances the Effects of Conventional Therapy

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Abstract

Context: This study compares the efficacy of a combination therapy consisting of alpha-lipoic (α-lipoic) acid plus Lymphomyosot (a homeopathic combination medication for treating edema in the extracellular matrix) to α-lipoic acid monotherapy in the treatment of diabetic peripheral neuropathy.

Patient population: 269 type II diabetics with peripheral neuropathy and residual sensation in toes/foot/ankle.

Methods: Multicenter, open-label prospective cohort study (add-on design).

Results: Statistically significant differences between treatments were seen in favor of the Lymphomyosot/α-lipoic acid combination therapy with regard to the following subjective criteria: light touch (monofilament), numbness, paresthesia, nocturnal spontaneous pain, and reduction in palpable edema in the foot/ankle. The combination therapy also produced more rapid improvement in symptoms, and physicians' overall ratings of its efficacy were higher. No adverse drug events were reported for either treatment group.

Conclusion: Adding Lymphomyosot to α-lipoic acid therapy for diabetic peripheral neuropathy results in statistically significant and clinically relevant improvements in sensation and palpable edema in comparison to α-lipoic acid monotherapy.

Keywords: Lymphomyosot, α-lipoic acid, diabetic neuropathy, homeopathy

Introduction

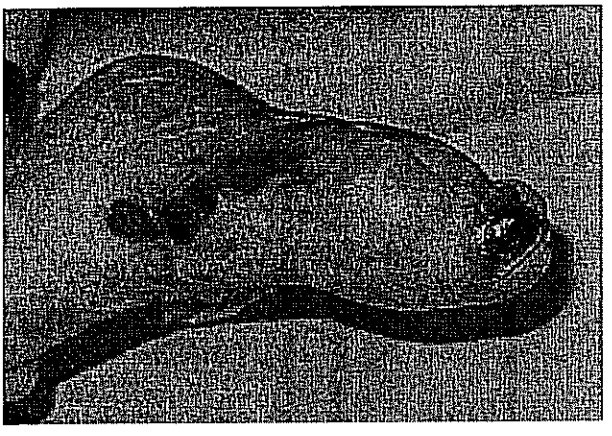
In Germany alone, diabetic peripheral neuropathy (DPN) results in approximately 25,000 foot amputations per year. As early as 1989, the World Health Organization's St. Vincent Declaration called for halving this amputation rate within ten years. This goal is still far from being accomplished.[1,2]

The WHO and the International Diabetes Federation predict a doubling in annually reported new diabetes cases by 2025.[1,3] The number of patients with diabetic foot lesions is also expected to continue to increase sharply, since at least one in three diabetics is affected by DPN.[4]

Neuropathic/neuroischemic causes account for 85-90% of cases of diabetic foot syndrome, and 45-60% of cases are assumed to be purely neuropathic in origin.[5,6] Typical symptoms of diabetic neuropathy include burning, stabbing pain ("burning feet"), paresthesia, hyperesthesia, and numbness. Other characteristics include nocturnal exacerbation of pain and improvement in symptoms while walking.[2,3,6]

Clinical monitoring of foot sensitivity to temperature, pain, movement and touch is very important. Deficiencies in or loss of these types of sensations indicate the development of complications such as neuropathic foot ulcers, edema, osteoarthropathy, Charcot's joint, and osteomyelitis.[4]

In the purely neuropathic diabetic foot, peripheral neuropathy is the sole cause of foot lesions. Rather than inadequate perfusion, the primary manifestation is severe damage to the sympathetic nerves of the vascular walls, which results in peripheral vasodilation and opening of the arteriovenous anastomosis with subsequent hyperperfusion and possible swelling of the foot.[7] Characteristic warning signs such as temperature sensitivity and pain during weight-bearing activities are not pronounced.[3] The foot appears rosy and warm with good perfusion.[8] Reduced sensitivity to pain prevents patients from noticing foot injuries and infections, which tend to heal poorly in diabetics, and inflammation may go unnoticed and untreated until it is severe.[9] Foot ulcers may not heal, and necrosis may eventually lead to loss of the limb.[2]



Absence of sensation of pain is the decisive factor in the pathogenesis of the most serious types of diabetic foot lesions, including gangrene, and amputation of toes.

The primary approach to treating the cause of diabetic peripheral neuropathy is to eliminate hyperglycemia as an etiological factor by normalizing sugar metabolism to the greatest extent possible.[4] Alpha-lipoic acid is a frequently prescribed treatment for DPN. Administered either intravenously or orally, α-lipoic acid reduces neuropathic symptoms/deficits. Through reduction of free radicals, it relieves oxidative stress and increases glucose utilization, which in turn reduces levels of nonenzymatic glycosylation products. The resulting improvement in energy availability leads to improved nerve response and reduces vascular dysfunction.[10] Adjuvant therapies including physical therapy, balneotherapy, relaxation therapy, transcutaneous electrical nerve stimulation (TENS) and acupuncture may offer additional symptomatic relief from pain.[4] Platelet derived growth factor (PDGF) in the form of the recombinant drug beclapermin is a treatment that has recently become available for severe cases.[6,11]

In spite of all therapeutic efforts, however, the amputation rate among patients with diabetic foot syndrome is not declining worldwide.[1,2,7,12] As a result, the search is on for alternative and adjuvant measures. The antihomotoxic approach aims at optimizing lymphatic functioning in order to improve nutrition and waste removal on the cellular level.[8,13] Antihomotoxic medicine pays particular attention to the extracellular matrix as the transit area between capillaries and the cells (including neurons and their axons) that they supply with nutrients.[13] In diabetes, the detrimental effects of constant excess glucose cause the matrix to respond with inflammatory and edematous changes.[14] These changes are evident on ultrasound and angiographic images even in early-stage diabetic neuropathy, before vascular damage is detected.[8]

Lymphomyosot (N) (drops, tablets, or injection solution), manufactured by Biologische Heilmittel Heel GmbH of Baden-Baden, Germany, is a homeopathic combination medication containing components of plant, animal, and mineral origin processed in accordance with the regulations of the German homeopathic pharmacopoeia. The drug portraits of its constituents suggest applications in a number of pathologies (such as edema, susceptibility to infection, swollen glands, hypertrophy of the tonsils, tonsillitis, and perfusion disorders) in which edematous disorders of the extracellular matrix play a direct or indirect role.[15]

The therapeutic efficacy of Lymphomyosot has been confirmed by multiple clinical studies.[15-20] Furthermore, in an observational study of 90 type II diabetics, Dietz showed that Lymphomyosot improved clinical

symptoms of diabetic neuropathy by stimulating lymph flow.[8] After Lymphomyosot therapy, edema was reduced and sensation improved in a majority of patients. Based on these studies, the current multicenter, prospective, open-label cohort study was designed to systematically examine a larger patient population in order to determine whether a combination therapy of α-lipoic acid plus Lymphomyosot is superior to α-lipoic acid alone in its effect on symptom severity in diabetic neuropathy.

Results

Medical history

With regard to demographic parameters and vital signs, there were no obvious differences between the two groups at the beginning of the study. Furthermore, no statistically significant differences between groups were found with regard to average duration of the diabetic illness, type of diabetes therapy prescribed to date (oral antidiabetic medications, diet, insulin), or the presence of general risk factors such as hypertension and obesity. Significantly, diabetes management in both groups was rated only "satisfactory" by the physicians at the beginning of the study, and reduced compliance is also reflected in slightly elevated mean HbA_{1c} levels in both groups (Group A: 7.2%; Group B: 7.4%; see Table 1).

Analysis of data on the patients' history of DPN and skin lesions also revealed no significant differences between Group A and Group B with regard to these criteria. Detailed clinical assessments of peripheral neuropathy revealed that the predominant symptoms in both groups were: pain sensitivity (pinprick), sensitivity to light touch (monofilament), numbness, paresthesia, and (nocturnal) spontaneous pain. The symptom "palpable edema in the foot/ankle" occurred in a significantly higher percentage of patients in Group B (see Table 2).

In both groups, as expected, diabetes mellitus was accompanied by a number of concomitant illnesses, most commonly endocrine/metabolic/nutritional disorders, circulatory disorders, or diseases of the musculoskeletal system and connective tissue.

Tab. 1: Demographic data, vital signs, blood test results, and characterization of patients' type II diabetes at the beginning of the study. (1Fisher's exact test, 2variance analysis, *multiple listings possible; SD = standard deviation).

Criteria	Group A: α-lipoic acid (N = 114)	Group B: α-lipoic acid + Lymphomyosot (N = 155)	Statistics
Gender (f/m; %)	58/42	53/47	P = 0.42[1]
Age (mean ± SD, in years)	65 ± 9.9	64 ± 12.0	P = 0.66[2]
Weight (mean ± SD, in kg)	81 ± 13.1	80 ± 14.8	P = 0.72[2]
Systolic blood pressure (mean ± SD, mmHg, seated)	145 ± 14.6	145 ± 16.9	P = 0.94[2]
Diastolic blood pressure (mean ± SD, mmHg, seated)	86 ± 9.1	86 ± 8.6	P = 0.28[2]
Pulse (mean ± SD, beats/min, seated)	76 ± 8.8	75 ± 10.4	P = 0.36[2]
Fasting blood sugar (mean ± SD, mmol/l)	148 ± 39.4	151 ± 44.0	P = 0.54[2]
HbA_{1c} (mean ± SD, %)	7.2 (± 1.1)	7.4 ± 1.2	P = 0.10[2]
Duration of diabetes (mean ± SD, in months)	101 ± 72.1	108 ± 75.4	P = 0.45
Diabetes management (mean ± SD) *Scale: very good (1), good (2), satisfactory (3)*	2.8	2.7	P = 0.97[1]
Type of diabetes therapy* (%)			
• oral antidiabetic medication	83.3	75.5	P = 0.12[2]
• diet	40.4	44.5	P = 0.50[2]
• insulin	20.2	29.0	P = 0.10[2]
• other	3.5	2.6	
Risk factors* (type and frequency, %)			
• total	97.4	94.2	P = 0.21[2]
• hypertension	71.9	65.8	
• obesity	53.5	51.6	
• fatty liver	26.3	21.9	
• hyperlipidemia	61.4	53.5	
• gout	18.4	13.5	
• nicotine/alcohol	23.7	20.6	
• medications	7.9	7.7	
• other	4.4	0.0	

Methods

Procedure

The participating physicians (47 family practitioners and/or internists, some specializing in diabetes care) were recruited by Heel GmbH of Baden-Baden. Medical histories and clinical data on a total of 269 patients ranging in age from 20 to 94 years were recorded using standardized questionnaires. Each physician reported on several patients treated exclusively with α-lipoic acid (Group A, n = 114) as well as several others treated with a combination of α-lipoic acid and Lymphomyosot (Group B, n = 155) with a maximum of six patients per physician. The type of treatment each patient received (i.e., with or without adjuvant Lymphomyosot therapy) was left to the physician's discretion. For both groups, duration of treatment was six months. Data were compiled on patients/treatments and target criteria (see categories below) during initial, interim (after three months) and exit examinations.

Patients

Inclusion criteria

- Diabetes mellitus type II (for medical history data, see below)
- Confirmed diagnosis of diabetic peripheral neuropathy (for medical history data, see below)

Exclusion criteria

- Total absence of sensation in toes/foot/ankle
- Known intolerance reactions to Lymphomyosot and/or α-lipoic acid

Demographics/vital signs

- Age (in years)
- Gender (f/m)
- Height (in cm)
- Weight (in kg)
- Blood pressure (seated; in mmHg)
- Pulse (seated; beats per minute)

Medical history: Type II diabetes

- Duration of diabetic illness (months)
- Overall diabetes management (very good; good; satisfactory; unsatisfactory)
- Type of diabetic therapy (oral antidiabetic medications; diet; insulin; other)
- Risk factors (no/yes; for list, see table 1)
- Fasting blood sugar (mmol/l)
- HbA_{1c} (%) (Scale: <6.5 very good; 6.5-7.0 good; 7.1-8.0 satisfactory; >8.0 unsatisfactory)
- Concomitant illnesses/therapies

Medical history: Peripheral neuropathy/skin lesions

- Duration of illness (months)
- Patient status (ongoing care/returning/first time treated)
- Extent of the neuropathy (toes only/foot/foot & ankle)
- Ulcers/gangrene (no/yes; size in cm: <0.5; 0.5-1.5; >1.5)
- Other skin lesions (no/yes; type: fissures; eczema)
- Pale/livid skin discoloration (no/yes; scale: slight; moderate; severe)

Treatment

The type of treatment for DPN was left to the discretion of each patient's physician and took individual circumstances into account. 43% of patients in Group A and 49% in Group B were prescribed 300 to 600 mg per day of oral α-lipoic acid while 43% of Group A and 37% of Group B were prescribed the same oral dosage preceded by two to four weeks of intravenous therapy (also 300 to 600 mg per day).

The majority of the 155 patients who received adjuvant Lymphomyosot (oral only) were prescribed the standard dosage of three tablets or 15 drops three times a day.

Average duration of therapy was 184 days for Group A and 182 days for Group B.

Target criteria

Neuropathic symptoms

Over the course of therapy, both treatment groups experienced obvious improvements in subjective symptoms. With regard to the target criteria sensitivity to touch (monofilament), numbness, paresthesia, and (nocturnal) spontaneous pain, however, findings revealed statistically significant advantages to administering α-lipoic acid in combination with Lymphomyosot. Especially telling are the results with regard to the criterion "palpable edema in the foot/ankle." At the beginning of the study, 49 patients (46%) in Group A and 88 patients (63%) in Group B had palpable edema in the feet/ankles ($P<0.01$). Edema was reduced (in comparison to initial examination findings) in only 18% of the patients in Group A who experienced that symptom. In contrast, 47% of the patients with edema in Group B (adjuvant Lymphomyosot) experienced improvement ($P<0.001$) (see Figure). Statistical analysis of the criterion "onset of improvement in symptoms" (a measure of general state of health) revealed significantly more rapid onset of efficacy in Group B (adjuvant Lymphomyosot) ($P_{total}<0.0058$). In the first two months, improvement in neuropathic symptoms occurred in only 24% of the patients in Group A in comparison to 37% in Group B.

Blood test results

In both groups, scores for "overall diabetes management" improved from "satisfactory" to "good" over the course of the study (from 2.8/2.7 at the beginning of the study to

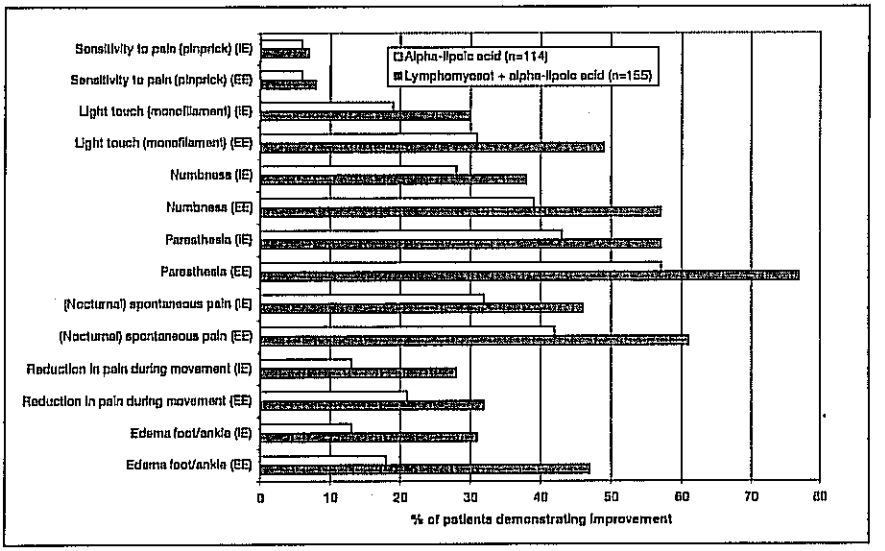


Fig.: Efficacy of treatment: Percentage of patients demonstrating improvement in target criteria as compared to entrance examination findings (IE = interim examination, EE = exit examination, * P<0.05)

2.3/2.2 at the end for Groups A and B respectively. At the same time, average blood sugar levels dropped (from 148/151 mmol/l at the beginning of the study to 133/131 mmol/l at the end), as did mean HbA_{1c} levels (from 7.2%/7.4% at the beginning of the study to 6.7%/6.8% at the end for Groups A and B respectively, $P_{group\ comparison}$ >0.05 in each case).

Evaluation of therapy
The superiority of the combined therapy is also reflected in ratings of the therapeutic results. In Group B, a significantly (P_{total} <0.0018) larger number of patients achieved "very good" or "good" results (70% of Group B vs. 46% of Group A). Similarly, the treatment failure rate in group A, at 16%, was four times as high as in Group B.

Tolerability
No adverse drug events occurred in either of the treatment groups during the course of the study. In both groups, the physicians rated patient tolerance of therapy as either "very good" or "good" in over 98% of cases.

Compliance/dropouts
The two treatment groups did not differ significantly with regard to patient compliance. In both groups, compliance was rated either "very good" or "good" in over 90% of cases.
A total of 10 patients (four from Group A and six from Group B) terminated treatment prematurely. Reasons given were: inadequate efficacy of the therapy (three cases) and personal reasons (seven cases).

Discussion

The causes of loss of sensation in diabetic foot syndrome include functional disorders of the autonomic nervous system. Poorly controlled diabetes, in particular, promotes denervation because the constant excess glucose flooding the extracellular matrix leads to nonenzymatic glycosylation processes that obstruct transit between blood/lymph vessels and peripheral nerves.[14, 21] Reduced matrix permeability leads to reduced lymph evacuation. In early-stage peripheral neuropathy, the resulting edema can be detected by means of sonography, NMR imaging, or body tissue measurements. Vascular damage can be detected with the help of color Doppler sonography and angiography.[8] The significance of edema in the development of diabetic foot syndrome is underlined by the observation that arterial and venous vascular damage is already evident in moderate edema; in severe chronic edema, increasingly serious vascular sclerosis with lumen constriction becomes evident.[8] Edema – specifically, extracellular edema – plays a crucial role in diabetes mellitus because it is associated not only with pronounced tissue degeneration (including diabetic foot syndrome) but also with peripheral neuropathy.
Consequently, treating the perineural matrix edema that accompanies peripheral neuropathy improves the efficacy of measures to prevent and treat diabetic foot syndrome. Several studies have already demonstrated that the homeopathic combination medication Lymphomyosot effectively "flushes out" edema.[15-20] The current study revealed decreases in palpable edema in only 18% (n = 49) of patients treated with α-lipoic acid alone but in 47% (n = 88) of those receiving adjuvant Lymphomyosot.
In the development of diabetic neuropathy, thickening of the basal membranes of the capillaries in myelin sheaths of peripheral nerves plays an important role because it hinders tissue fluid drainage and leads to overburdening of lymph vessels.[13] In addition, nonenzymatic glycosylation products in the matrix create an inflammatory situation, including formation of free radicals. The consequences are functional breakdowns of the affected nerves and/or slowed neural transmission.
Alpha-lipoic acid, which is used to

- Mycosis (no/yes; location: skin, interdigital, toenails)
- Anhidrosis (no/yes; scale: mild, moderate, severe)
- Pressure points/calluses (no/yes; scale: mild, moderate, severe)
- Reduced joint mobility (no/yes; scale: mild, moderate, severe)
- Paresthesia at rest (no/yes; scale: mild, moderate, severe)

Treatment prescribed for neuropathy

- Dosage and duration of therapy with Lymphomyosot (oral only) and/or α-lipoic acid (i.v., oral or parenteral)
- Other therapies (pharmaceutical/nonpharmaceutical)

Target criteria

- Severity of neuropathic disorders:
  1. sensitivity to pain (piprick) (no/yes/yes, scale: sharp, dull)
  2. light touch (monofilament) (no/yes/yes, scale: faint sensation, significant sensation, pronounced sensation)
  3. numbness (no/yes; scale: mild, moderate, severe)
  4. paresthesia (pricking) (no/yes; scale: mild, moderate, severe)
  5. (nocturnal) spontaneous pain (no/yes; scale: mild, moderate, severe)
  6. reduction in pain during movement (no/yes; scale: slight, significant)
- Palpable edema in the foot/ankle (no/yes)
- Onset of improvement in neuropathic symptoms (scale: < 1 month, 1-2 months, 2-3 months, 3-6 months, no improvement)
- Overall assessment of results of therapy (scale: very good, good, fair, no success, symptoms worsened)
- Overall assessment of patient compliance (scale: very good, good, fair, poor)
- Tolerability (overall assessment of therapy in terms of adverse effects) (scale: very good, good, fair, poor)
- Premature termination of therapy

Statistical analysis

Data compiled on patients/therapies were analyzed using exploratory statistical methods; absolute and percentage frequencies were calculated and graphed. Differences in treatment effects were calculated with 95% confidence intervals. The patients' demographic data and medical histories were compared (using ANOVA/Fisher's exact test to test for comparability of the treatment groups at commencement of therapy). Distribution of background characteristics in the two treatment groups was described using statistical parameters and absolute and relative frequencies. Then logistic regression (propensity score) method was used to test for possible interactions between background characteristics (structural variables) and treatments prescribed. Because patients with similar propensity scores exhibit similar distribution of background characteristics, comparisons within propensity score classes can reduce the influence of unequal distribution of these characteristics on treatment results.

Tab. 2: Characterization of diabetic neuropathy and/or skin lesions at the beginning of treatment (¹Fishers exact test, ²variance analysis, SD = standard deviation).

Criteria	Group A: α-lipoic acid (N = 114)	Group B: α-lipoic acid + Lymphomyosot (N = 155)	Statistics
Duration of symptoms (mean ± SD, in months)	28 ± 27.8	31 ± 28.2	P = 0.31 ¹
Patient status (%)			
• patients in ongoing care	27.2	33.5	
• returning patients	28.1	31.0	
• first-time patients	44.7	34.2	P = 0.08 ²
• not given	0.0	1.3	
Extent of neuropathy (%)			
• toes only	28.9	16.8	P = 0.02 ²
• foot	34.9	42.6	P = 0.16 ²
• foot/ankle	37.7	41.9	P = 0.49 ²
• other/not given	0.9	0.6	
Findings, neuropathy (% of patients exhibiting symptom)			
• ulcer/gangrene	5.3	6.5	P = 0.68 ²
• other skin lesions (fissures, eczema)	20.2	25.2	P = 0.34 ²
• pale/livid skin discoloration	37.7	42.6	P = 0.42 ²
• mycosis	32.5	34.8	P = 0.68 ²
• anhidrosis	29.0	25.2	P = 0.49 ²
• pressure points / calluses	47.4	45.2	P = 0.72 ²
• reduced joint mobility	50.9	51.0	P = 0.99 ²
• paresthesia at rest	86.0	87.7	P = 0.67 ²
• pain (pinprick) not felt or felt as dull pain	50.0	54.2	P = 0.73 ²
• light touch (monofilament) not felt or felt only slightly	75.4	79.4	P = 0.30 ²
• numbness	90.4	89.7	P = 0.83 ²
• paresthesia	93.0	96.8	P = 0.64 ²
• (nocturnal) spontaneous pain	72.8	72.9	P = 0.99 ²
• pain does not decrease with movement	36.8	40.7	P = 0.53 ²
• palpable edema in the foot/ankle	45.6	62.6	P = 0.01 ²
• A. tibialis posterior not palpable	7.9	11.6	P = 0.32 ²
• A. pedis dorsalis not palpable	9.6	10.8	P = 0.86 ²
• Achilles tendon reflex not present	16.8	17.4	P = 0.72 ²
• patellar reflex not present	15.8	17.4	P = 0.80 ²

deactivate free radicals in diabetic peripheral neuropathy, is a sulfur-containing fatty-acid. An easily oxidized compound, it assumes important biological functions in redox reactions, including catalytic functions in energy metabolism and antioxidant functions in hydrophilic and lipophilic cell compartments.

The efficacy of α-lipoic acid in treating DPN has been debated for years.[22] The ALADIN study demonstrated that clinical symptoms of peripheral neuropathy can be significantly reduced by a three-week course of intravenous α-lipoic acid (600 mg/day) in comparison to placebo.[23] The ALADIN II study found that 24 months of α-lipoic acid therapy, when administered as a combination of five intravenous doses followed by oral administration of 600 mg per day, produced significant improvements in sensory neural transmission speed in comparison to placebo.[24] The ALADIN III study, however, found that three weeks of intravenous α-lipoic acid therapy followed by six months of oral therapy (both with 600 mg of α-lipoic acid per day) was not significantly more effective than placebo in reducing neuropathic symptoms.[10]

The purpose of the present study was to investigate whether a combination therapy of Lymphomyosot plus α-lipoic acid is superior to α-lipoic acid alone in treating diabetic peripheral neuropathy. In this exploratory approach, no special requirements were imposed regarding the dosage of α-lipoic acid or its method of administration (i.v., injection, oral). The decision to limit the treatment period to six months takes the etiology of peripheral neuropathy into account and aligns the study with comparable clinical investigations.[10]

The results of this study confirm

that improving the cell matrix milieu through adjuvant use of Lymphomyosot improves the prospects of success in treating diabetic peripheral neuropathy. In other words, a combination therapy that includes this homeopathic medication has advantages over treatment with α-lipoic acid alone.

During the average six-month treatment period, the target criteria sensitivity to touch (monofilament), numbness, paresthesia, (nocturnal) spontaneous pain, and edema in the foot/ankle showed significantly greater improvement under the combination protocol than with α-lipoic acid alone. In addition, improvements occurred significantly early (after one to two months of treatment). This "enhancing" property of Lymphomyosot is also reflected in assessments of therapeutic results. In the group receiving combination therapy with Lymphomyosot, results of therapy were rated "very good" or "good" in 70% of cases in comparison to only 47% of cases treated with α-lipoic acid alone.

It is not known how much influence the severity of the patients' neuropathy (toes only, foot, foot/ankle) had on overall results, especially since Group A included a significantly higher percentage of patients with symptoms limited to the toes. In such patients in particular, the possibility of clinical confirmation of documented subjective symptoms is limited and symptomatic improvements are difficult to differentiate. (For this reason, statistical analysis that separates out these patients is currently in progress.)

The effect of the confounder "diabetes management" – in other words, of lower blood sugar levels – on improvements in symptoms is also not known. For purposes of answering the question posed by this study, however, this issue is irrelevant because if the effect of improved diabetes management is assumed to be not one-sided (i.e., to have had the same size effect in both groups), it has no influence on any assessment of treatment effects or of how they differed between the groups (α-lipoic acid alone vs. α-lipoic acid plus Lymphomyosot).

Independent of these questions, the results of the present study give diabetics reason to hope that their unpleasant and painful symptoms can be reduced and their quality of life enhanced. Adding the homeopathic medication Lymphomyosot to their therapeutic regimen can contribute significantly to the prophylaxis of lesions of various types. Persistent sensory deficits in the feet and/or ankles due to diabetic peripheral neuropathy often allow seemingly minor injuries to go undetected by the patient. In combination with the delayed wound healing common in diabetics, such injuries can quickly develop into chronic infections that can lead to ulceration, gangrene, and even the loss of the limb.[2,9]

In this context, Lymphomyosot's excellent tolerability should be emphasized. The fact that this homeopathic medication has no counterindications, side effects, or known drug interactions makes it suitable for long-term use. In contrast, the allopathic medications used for symptomatic relief of diabetic neuropathy (such as tricyclic antidepressants and anticonvulsants) have been linked to serious side effects that limit their long-term use. (These side effects include anticholinergic effects such as cardiac dysrhythmias, changes in blood pressure, tremors, dry mouth, excessive perspiration, and urination disorders. Monoamine oxidase inhibitors are also associated with sleep disorders, nausea, headache, and blood pressure changes; serotonin uptake inhibitors have been linked to headache, nausea, and vertigo.)

Conclusions

This study's findings indicate that the addition of the homeopathic combination medication Lymphomyosot enhances the effect of α-lipoic acid therapy. The mechanism on which this effect is based is still unknown, but it is conceivable that Lymphomyosot's plant ingredients (including pine, yellow gentian, and horsetail) support the action of α-lipoic acid by trapping free radicals.[25] It is also possible that Lymphomyosot, an alkaline product, improves α-lipoic acid utilization by counteracting tissue acidosis. Further studies are needed to answer these questions.

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