



Venolymphatic Edema And Its Treatment Options

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Raghu Kolluri, MD: Disclosures


- **Consultant/Advisor/ DSMB/ CEC -**
 - Abbott, Auxetics, Diachii Sankyo, Koya Medical, Medtronic, Penumbra, Philips, Surmodics, USA Therm, VB Devices
- **Board of Trustee**
 - The VIVA Foundation
 - Intersocietal Accreditation Council | Vascular Testing
- **President**
 - Syntropic Core Lab



Structural Vs Functional Edema

Other Causes of Lower Extremity Edema	Mechanism of action	Lymphed	Diagnos
Primary cause:			
Cardiac (right heart failure)	Increased venous pressure leading to increased capillary permeability and an increase in plasma volume	X	X
Renal failure	Increased venous pressure leading to increased capillary permeability and an increase in plasma volume	X	X
Heart failure with preserved ejection fraction	Increased venous pressure leading to increased capillary permeability and an increase in plasma volume	X	X
Heart failure with reduced ejection fraction	Increased venous pressure leading to increased capillary permeability and an increase in plasma volume	X	X
Renal	Decreased protein synthesis and decreased plasma oncotic pressure leading to increased systemic pressure	X	X
Thyroid and adrenal disorders	Decreased protein synthesis and decreased plasma oncotic pressure leading to increased systemic pressure	X	X
Chronic sinusitis	Increased venous pressure leading to increased capillary permeability and an increase in plasma volume	X	X
Alleged cause: angiodema and urticaria	Increased capillary permeability	X	X
Malabsorption and malnutrition	Decreased protein synthesis and decreased plasma oncotic pressure	X	X
Pregnancy related	Increased plasma volume	X	X
Postmenstrual edema	Increased plasma volume	X	X
Idiopathic	Unknown	X	X
Large calcium channel blockers, vasodilators, NSAIDs, antipsychotics, antidepressants, antidiabetics, hormone therapy, corticosteroids, acute emergency medicine, chemotherapy, radiotherapy	Vascular mechanisms including increased capillary permeability from vasodilation, increased plasma volume by acute-phase reactants, and increased capillary permeability	X	X
Lymphedema			
Primary	Genetic abnormalities of lymphatic flow: This chronic and obstructive buildup of protein-rich fluid in the interstitial and intracellular spaces is caused by the lymphatic system's ability to effectively transport the fluid	X	X
Secondary	Acquired lymphatic obstruction	X	X
Obstructive	Increased venous hypertension and capillary permeability	X	X
Non-obstructive	Increased venous hypertension and capillary permeability	X	X
Cellular	Increased capillary permeability	X	X
Complex regional syndrome	Increased capillary permeability is mediated by neurogenic/endothelial dysfunction	X	X
Tumor/malignancy related	Increased local venous hypertension	X	X
Neoplasms or lymphatic system malignancies	Increased venous hypertension and capillary permeability	X	X
Compartment syndrome	Local venous hypertension resulting in increased capillary permeability	X	X
Post-burn related foot	Endothelial cell dysfunction and increased capillary permeability	X	X
Post-burn calf muscle/transverse myelomelia	Distraction of blood and inflammation-related increased capillary permeability	X	X

Circ Cardiovasc Interv. 2024 Aug;17(8)



International Society of Lymphology (ISL) Lymphedema - staging

- Stage 0 - latent / sub-clinical /swelling is


Effects of CDT are to:

1. decrease swelling^{25,26}
2. increase lymph drainage from the congested areas^{27,28}
3. reduce skin fibrosis and improve the skin condition¹
4. enhance patient's functional status²⁹
5. relieve discomfort and improve quality of life^{8,25,26,30-34}
6. reduce the risk of cellulitis and Stewart-Treves-Syndrome, a rare form of angiosarcoma^{35-45,197}

Components of CDT

1. manual lymph drainage (MLD)
2. multi-layer, short-stretch compression bandaging
3. lymphatic exercise
4. skin care
5. education in lymphedema self-management, and elastic compression garments^{32,46}

<http://www.lymphnet.org/pdfDocs/nintreatment.pdf>



Phlebolymphelema




The clinical characteristics of lower extremity lymphedema in 440 patients


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Table IV. Final lymphedema diagnosis (principal and hybrid)

	Total (n = 440)	CVI (n = 198)	Cancer related (n = 198)	Phlebolymp (n = 198)	Lymph (n = 198)	Primary lymph (n = 198)	Hybrid
Sex	440	198	198	198	198	198	
Male	210	100	100	100	100	100	
Female	230	98	98	98	98	98	
Median (IQR)	62.0 (27.0 - 71.0)	62.0 (27.0 - 71.0)	62.0 (27.0 - 71.0)	62.0 (27.0 - 71.0)	62.0 (27.0 - 71.0)	62.0 (27.0 - 71.0)	
Median (IQR) ^a	440	198	198	198	198	198	
Male	210	100	100	100	100	100	
Female	230	98	98	98	98	98	
Median (IQR)	62.0 (27.0 - 71.0)	62.0 (27.0 - 71.0)	62.0 (27.0 - 71.0)	62.0 (27.0 - 71.0)	62.0 (27.0 - 71.0)	62.0 (27.0 - 71.0)	

J Vasc Surg: Venous and Lym Dis 2019; :1-9.

Primary + CVI: 16/440 (4%)
 Primary + CVI + surgery: 2/440 (0.5%)
 Primary + surgery: 5/440 (1%)
 CVI Chronic venous insufficiency



Central venous hypertension


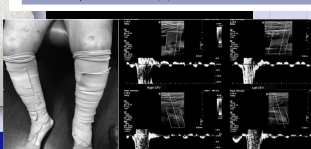


Table III. Venous reflux study results

Venous reflux study	(N = 264)
Superficial reflux	218 (82.6)
Deep vein reflux	99 (37.5)
Combined deep and superficial reflux	89 (33.7)
Chronic deep venous thrombosis	20 (7.6)
Pulsatile venous flow	3 (1.1)

Values are reported as number (%).



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Prevalence and predictors of elevated central venous pressure and obstructive sleep apnea in patients with lower extremity chronic venous disease

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Venous Reflux Study		n=264	
CV confirmed, n (%)	223 (84.2)		
CEAP score, right extremity, median (range) ^a	3.0 (0.0 - 6.0)		
CEAP score, left extremity, median (range) ^a	3.0 (0.0 - 6.0)		
CEAP score, right extremity, n (%)		Right ^b	Left ^b
C0	9 (3.5)	10 (4.0)	
C1	19 (7.5)	15 (5.9)	
C2	34 (13.4)	37 (14.6)	
C3	69 (27.7)	63 (25.7)	
C4	82 (32.2)	82 (32.4)	
C5	19 (7.5)	17 (6.7)	
C6	22 (8.7)	23 (9.3)	
VCSS score, median (range)	7.0 (0.0 - 30.0)	Right ^b	Left ^b
		7.0 (0.0 - 30.0)	

Table 1: Demographic and Clinical Characteristics		Overall
Age, mean (SD)		59 (10.1)
Female, n (%)		133 (50.0)
Obstructive sleep apnea, n (%)		117 (44.3)
Current smoker, n (%)		11 (4.2)
Never		11 (4.2)
STOP AHA/ASA score, n (%) ^c		11 (4.2)
Low risk		11 (4.2)
Moderate risk		0 (0.0)
High risk		0 (0.0)
Obstructive sleep apnea, n (%)		117 (44.3)
Diagnosed with CPAP therapy ^d		60 (51.3)
Hypertension, n (%)		17 (6.7)
Diabetes mellitus, n (%)		40 (15.1)
Coronary artery disease, n (%)		49 (18.5)
Peripheral artery disease, n (%)		10 (3.8)
Chronic kidney disease, n (%)		10 (3.8)
Stroke, n (%)		10 (3.8)
Presence of postoperative cellulitis, n (%)		10 (3.8)
Final history of congestive heart failure, n (%)		10 (3.8)
Final history of atrial fibrillation, n (%)		10 (3.8)
Final history of liver disease, n (%)		10 (3.8)

J Vasc Surg Venous Lymphat Disord. 2020 Sep;8(3):775-782

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Compression (not GCS)



Figure 1. Types of compression therapy:

1. Elastic bandage, 8 mm x 4 mm
2. Tubigrip, 8 mm x 4 mm
3. Solaris wrap, 8 mm x 4 mm
4. Biocare wrap, 8 mm x 4 mm
5. Velcro wrap, 8 mm x 4 mm
6. Tubigrip wrap, 8 mm x 4 mm
7. Tubigrip wrap, 8 mm x 4 mm




Curr Treat Options Cardio Med (2011) 13:169-178

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Medium stretch + Tubigrips = Multilayered wraps



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Velcro Compression Therapies



Circaid



Solaris



Biocare

Curr Treat Options Cardiovasc Med. 2011 Apr;13(2):169-78

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Lymphedema pumps







MAKE SURE THERE IS NOT EVIDENCE OF RHF!!

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Venous therapies



RLE – 4 Ablations | LLE – 3 Ablations



Summary

- **Lymphedema CDT + NO venous therapies** – If the patient needs compression therapy long term and there are no stasis changes + patient is compliant – Symptoms are stable
- **Lymphedema CDT + Non-Thermal venous therapies** – If the patient has
 - Progressive symptoms despite compliance with Lymphedema CDT/ pumps/ compression etc
 - Severe LDS
 - C6 disease
 - Venous hemorrhage

