IMA

EMERGING TRENDS IN MEDICINE

ENDOTHELIAL DAMAGE FROM COVID IN VENOUS DISEASE AND POTS

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INDEPENDENT™
MEDICAL ALLIANCE

EMERGING TRENDS IN MEDICINE

Endothelial Damage from COVID in Venous Disease and POTS

With

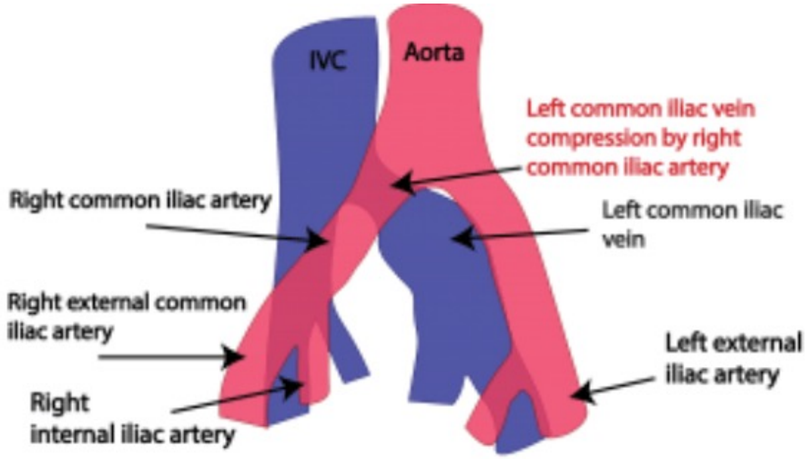
Dr. Jordan F Vaughn

- May-Thurner (Pelvic Venous Disease) Plus MCAS and OI (Orthostatic Intolerance) or POTS following Endothelial Injury (Spike Protein)

- **Common in:**
  - Hypermobile: EDS or EDS Like
  - Runners/Cyclist/Pelvic Trauma/Horseback Riders?
  - Multiparous Women/Pelvic Inflammation History (EndoMetriosis et al)
  - Men with Scrotal Fullness/Testicle History/NewED? including Varicocele, Torsion, Pudendal Nerve Pain

- **Often Results in Lower Extremity Symptoms Initially.**
  - Most Remember a TIME and DATE when things in their body were DIFFERENT
  - Many had Mild issues prior including Leg going to sleep when sitting for long times mild Orthostatic Intolerance

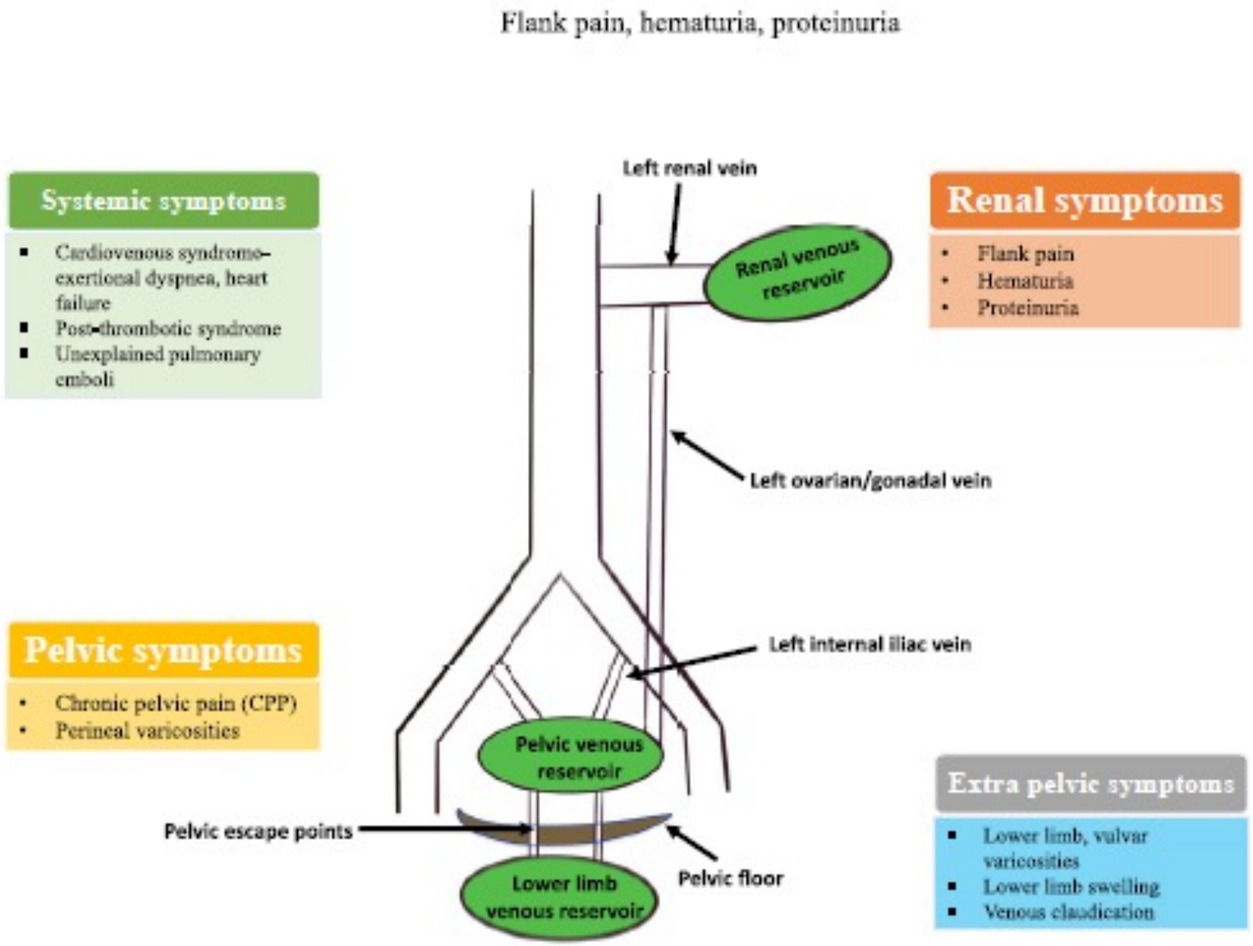
- Often but not Always: Symptoms Initially Lower Ext/Muscles Heavy, Fail to Recover with Exercise
- POTS/OI and MCAS symptoms increased.
- Triple Therapy (Antiplatelet and AntiCoags) Help but not Curative

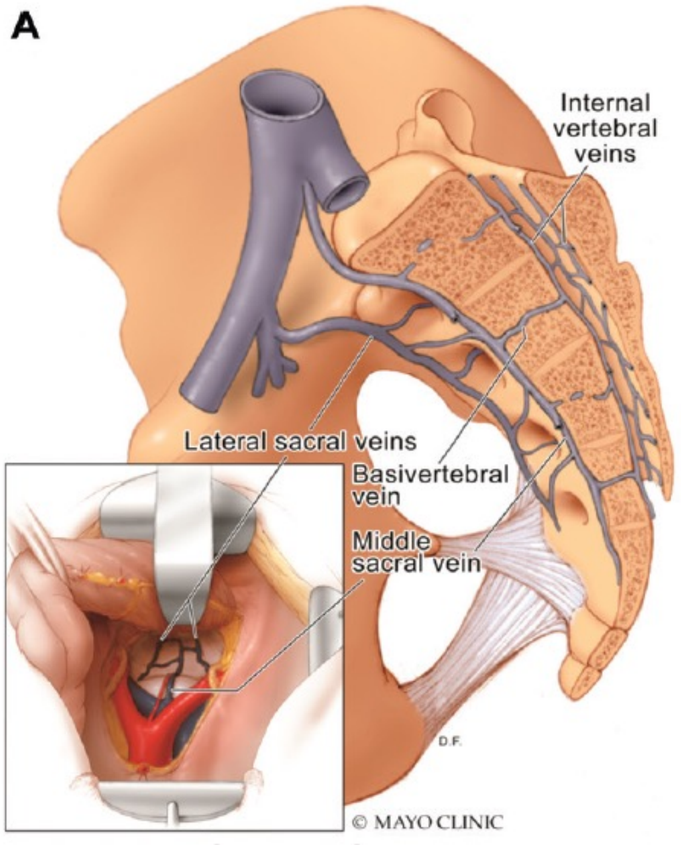


Spencer E, Elhage Hassan M, Saikia J et al. **Association and post-iliac vein stenting symptom improvement of postural orthostatic tachycardia syndrome and orthostatic intolerance with pelvic venous disorders: two retrospective studies**. eClinicalMedicine, 2026; 92

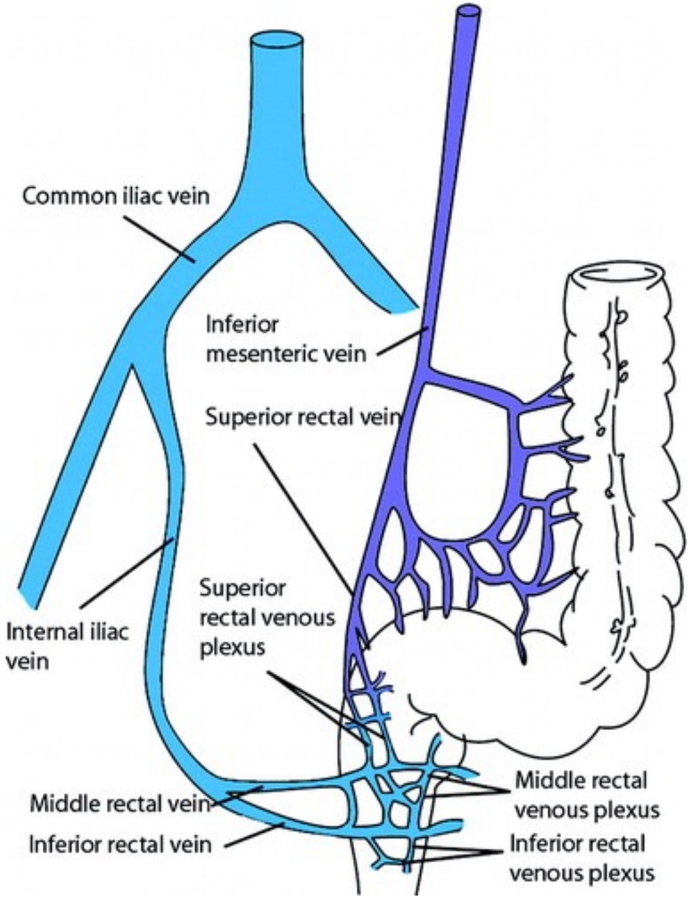
- Additional Issues:
  - Urinary Urgency or ‘Frequent Urinary Tract Infections’ or Interstitial Cystitis
  - Heavy Menstrual Cycles, Pelvic Pain, all Issues are worse before and during cycle
  - Hemorrhoids and Irritable Bowel Symptoms (Absorption and Transit Issues)
  - Lower Sacral Back Pain and Sacral Ileitis Symptoms
  - Hip Pain or Non healing Lower Extremity Injury or Procedure
    - (ex. Labral Tear of L Hip or Stress Fracture in Athlete)
  - Men: Boggy Prostate and Nocturia in Younger Men.

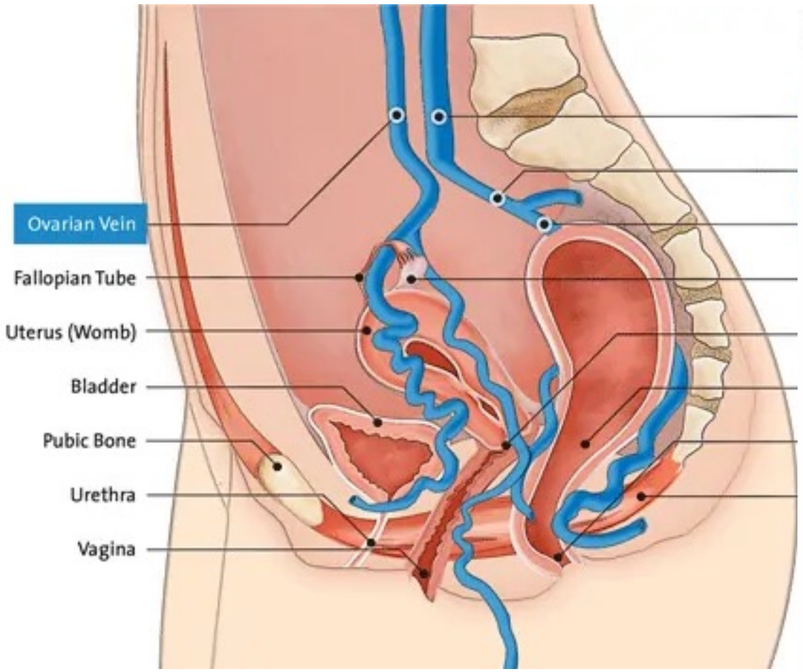
Spencer E, Elhage Hassan M, Saikia J et al. **Association and post-iliac vein stenting symptom improvement of postural orthostatic tachycardia syndrome and orthostatic intolerance with pelvic venous disorders: two retrospective studies**. eClinicalMedicine, 2026; 92

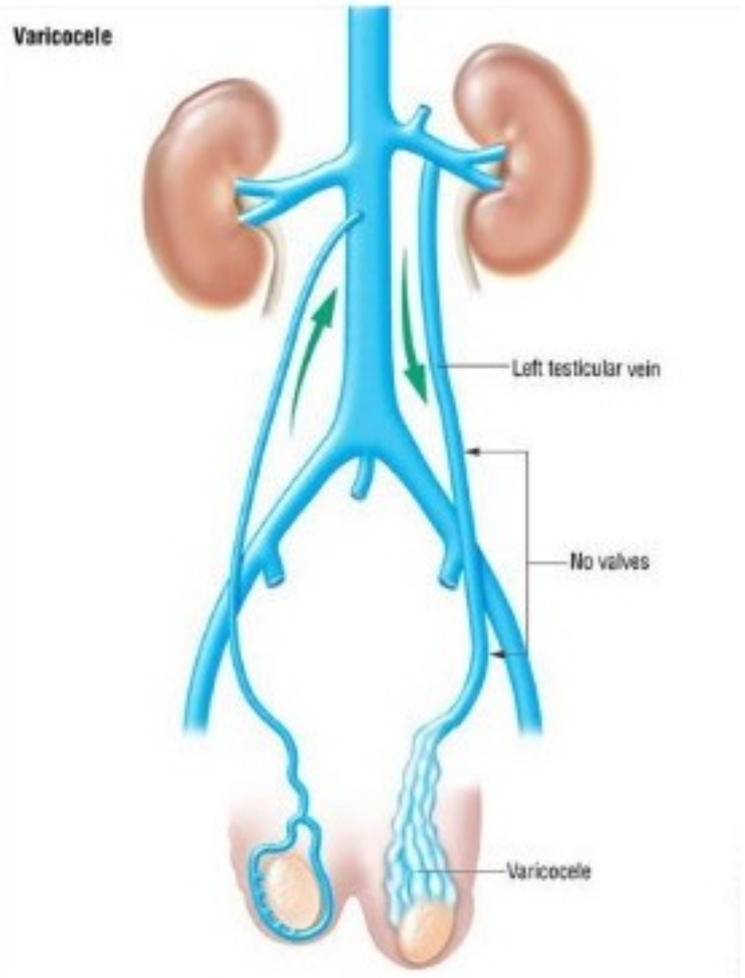




Sheikh AB, Fudim M, Garg I, Minhas AMK, Sobotka AA, Patel MR, Eng MH, Sobotka PA. The Clinical Problem of Pelvic Venous Disorders. Interv Cardiol Clin. 2022 Jul;11(3):307-324. doi: 10.1016/j.iccl.2022.03.003. PMID: 35710285.







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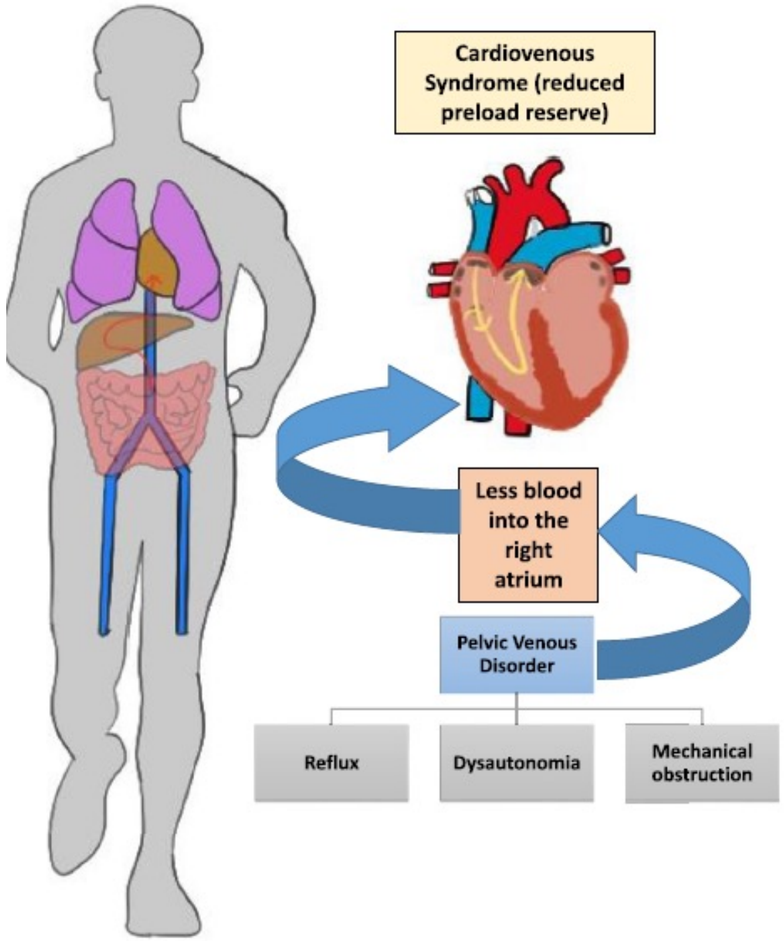

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Spike Protein or Immune response to viral infection or vaccination Mediated Injury to Vasa Vasorum and Vein Wall Muscle

1.Bind ACE2 Receptors

- ACE2 is expressed on **endothelial cells**, including those of the **vasa vasorum**.
- Binding disrupts **ACE2 signaling**, leading to:
  - **Endothelial dysfunction**
  - **Oxidative stress**
  - **Pro-thrombotic and pro-inflammatory states**

2.Trigger Endotheliitis

- COVID Virus Antigen can directly activate **inflammatory pathways** (e.g., **NF-κB**, **TLR4**) in endothelial cells.
- This leads to **immune cell infiltration**, **microthrombosis**, and **capillary leak**—
- **particularly damaging to the fragile vasa vasorum**.

3.Promote Microclot Formation

- Spike protein interacts with **fibrinogen**, altering clot architecture.
- Resulting **fibrin-rich microclots** are resistant to breakdown and may **occlude vasa vasorum**.

4.Induce Autoimmunity

- Molecular mimicry or immune priming can lead to **autoantibodies targeting endothelial cells**, platelets, or coagulation factors.
- This immune response may persist and cause **ongoing vascular injury**, especially in predisposed individuals (e.g., those with **MCAS, POTS, or connective tissue disease**).

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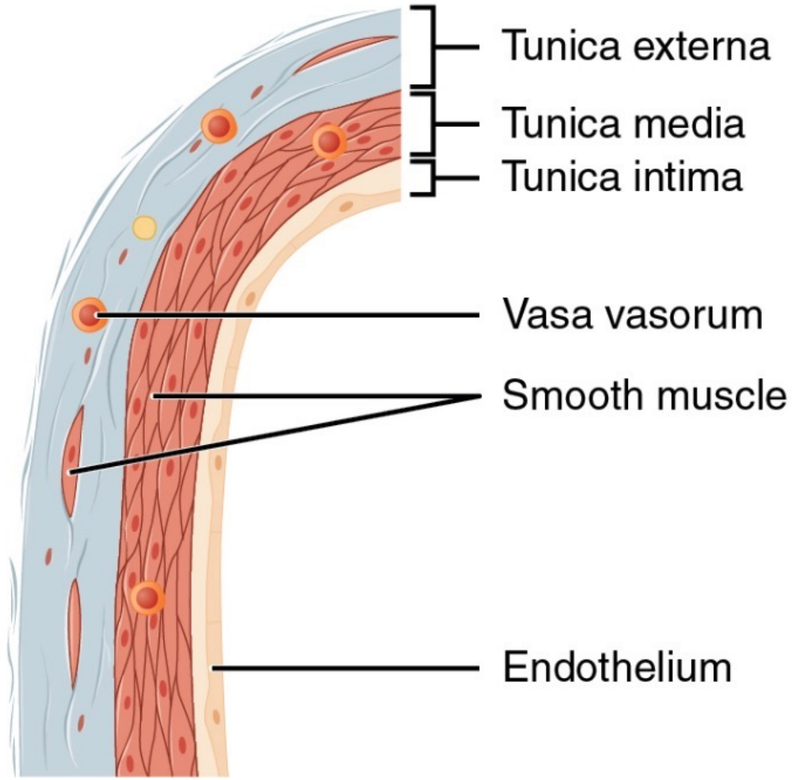
Impact on the Muscular Layer of Large Veins

When the **vasa vasorum are damaged**, the result is **ischemic injury to the tunica media**, including:

- **Smooth Muscle Cell Apoptosis**
  - From hypoxia or direct inflammatory signaling
- **Wall Weakening and Fibrosis**
  - Reduced contractility, increased stiffness
- **Vein Dilation and Valvular Dysfunction**
  - Contributing to **chronic venous insufficiency**
- **Loss of Vessel Tone**
  - Especially relevant in dysautonomia and orthostatic intolerance seen post-vaccine or post-infection



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Shimizu K, et al. — Vasa vasorum ischemia contributes to plaque instability and wall injury

Step 1: Venous Wall Damage

- **Hypoxia of tunica media** from vasa vasorum occlusion →
  - Smooth muscle apoptosis
  - Fibrosis
  - Loss of compliance
  - Venous dilation or collapse

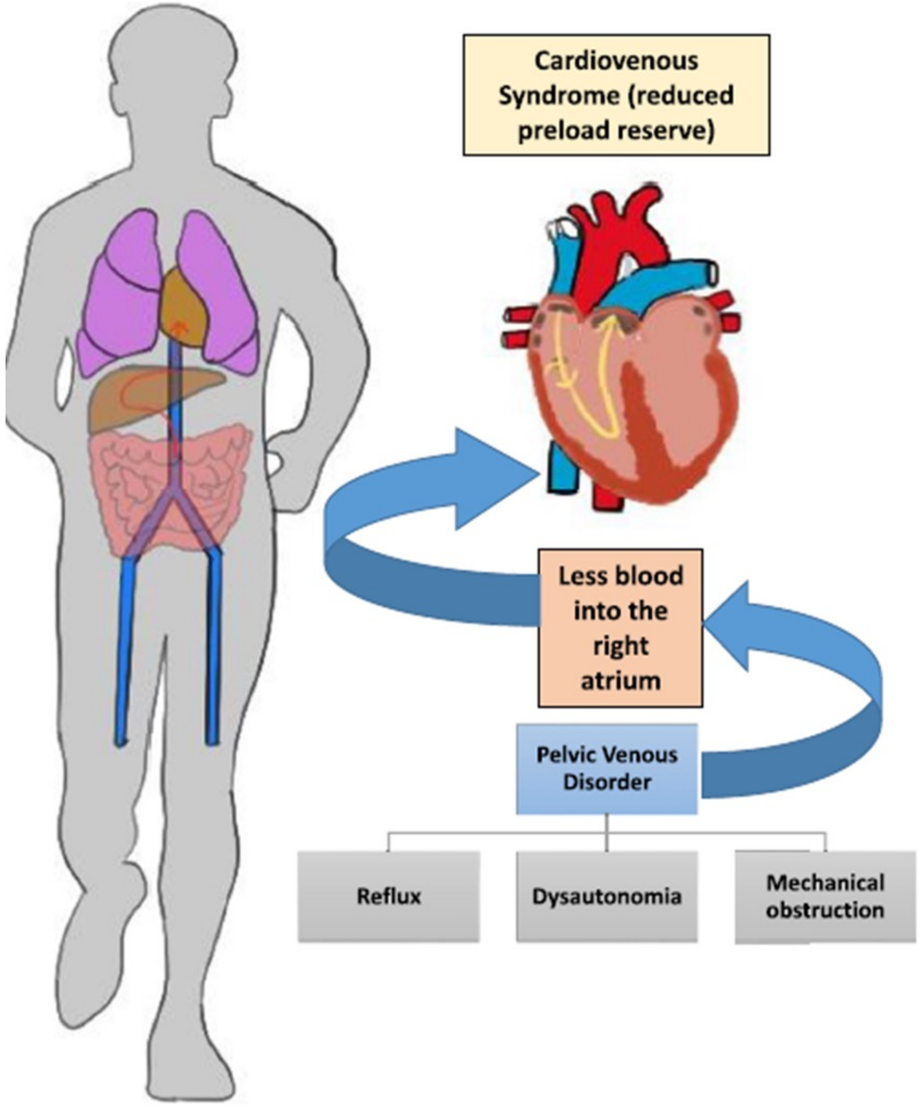
Step 2: Venous Compression Syndromes

- Injured or fibrosed veins are prone to **mechanical compression** at known anatomical choke points:
  - **Left common iliac vein** (May-Thurner syndrome)
  - **Jugular or brachiocephalic vein** (thoracic outlet compression)
  - **Left renal vein** (Nutcracker syndrome)
- Can lead to:
  - **Collateral vein formation**
  - **Stagnant flow**
  - **Further clotting or inflammation**

Davis, B.M.; Rantanen, P.; Seo, G.; Thadani, S.; Spencer, E.B.; Hepworth, E.; Cutchins, A. An Overview of Vascular Compression Syndromes and Associations with Autonomic Dysfunction: A Review. Biomedicines 2026, 14, 689. https://doi.org/10.3390/biomedicines14030689

Step 3: Right Heart Preload Failure

- Impaired **venous return** reduces **right ventricular preload**.
- Consequences:
  - **Low cardiac output**
  - **Reduced pulmonary perfusion**
  - **Exertional fatigue**
  - **Orthostatic intolerance**



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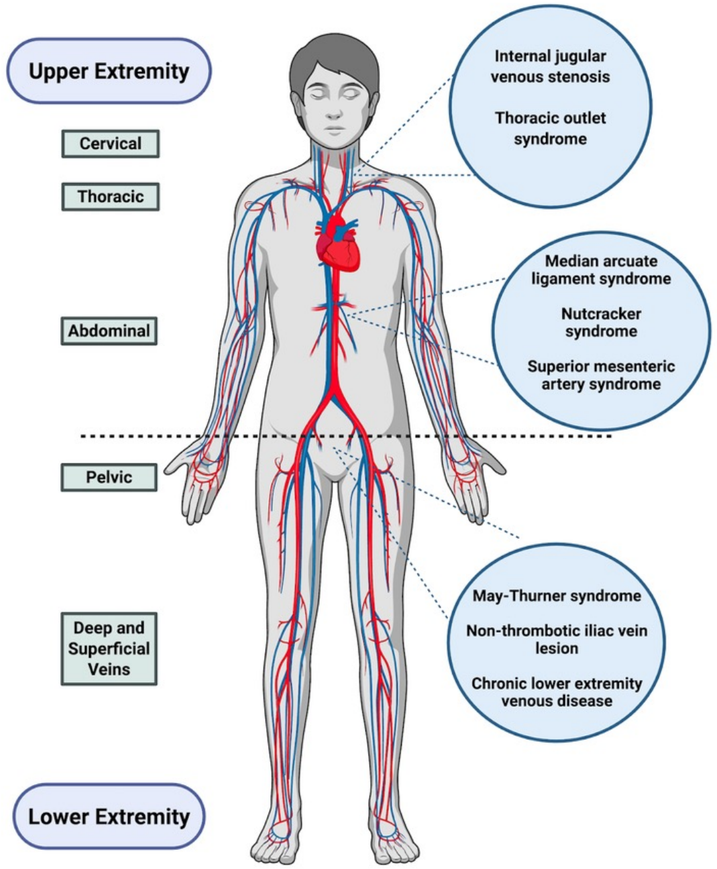
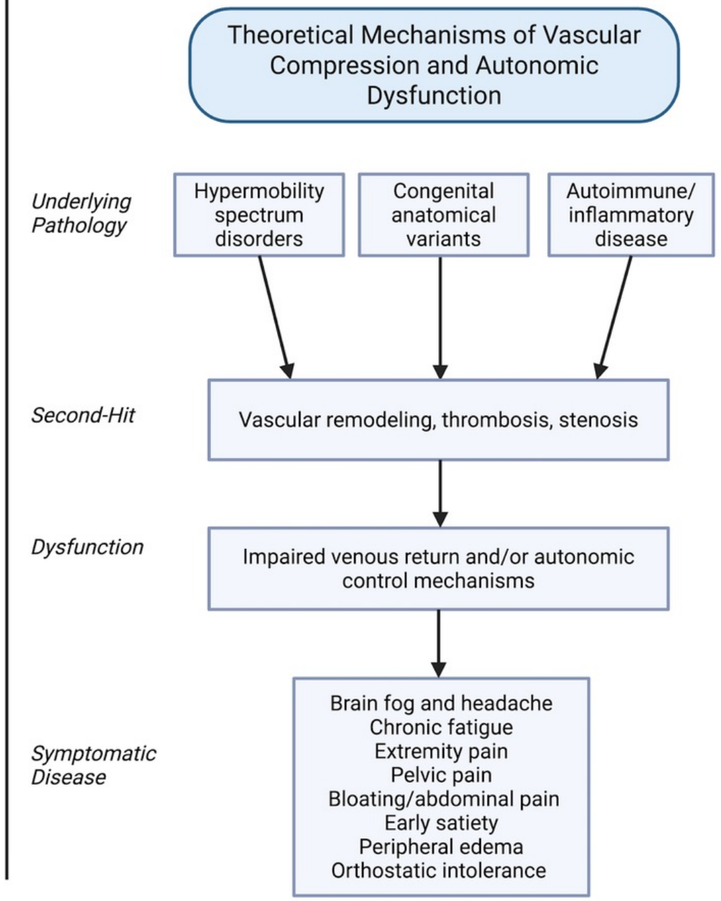
Step 4: Hypocapnic Cerebral Hypoperfusion

- **Low preload + low CO2** leads to:
  - **Cerebral hypoperfusion**—exacerbated by hyperventilation and **hypocapnia** (low CO_2)
- **Pathophysiology:**
  - **Hypocapnia** (low arterial CO_2) results from hyperventilation or abnormal respiratory patterns.
  - Decreased CO_2 (a powerful cerebral vasodilator) leads to **cerebral vasoconstriction**.
  - Vasoconstriction reduces cerebral blood flow (CBF), causing **cerebral hypoperfusion**.

- Symptoms:
  - **Brain fog**
  - **Lightheadedness**
  - **POTS-like features**
  - **Visual and cognitive disturbances**

Davis, B.M.; Rantanen, P.; Seo, G.; Thadani, S.; Spencer, E.B.; Hepworth, E.; Cutchins, A. An Overview of Vascular Compression Syndromes and Associations with Autonomic Dysfunction: A Review. Biomedicines 2026, 14, 689. https://doi.org/10.3390/biomedicines14030689





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Cerebral Blood Flow is Closely Tied to $EtCO_2$:

- Every **1 mmHg drop** in $EtCO_2$ is associated with ~**3% cerebral blood flow reduction**
- Novak's HYCH and POTS cohorts had **$EtCO_2$ drops >12 mmHg**, predicting **~36% cerebral perfusion loss**
- This degree of **hypocapnic vasoconstriction** explains the:
  - Orthostatic **brain fog**, **dizziness**, **lightheadedness**
  - **Cognitive decline** in chronic cases
  - Enhanced **central sensitization**

Novak P, et al. "Orthostatic intolerance with tachycardia and without represent a spectrum of the same disorder." Frontiers in Neurology. 2024.

A Clinical Way to Assess this with Orthostatic End Tidal CO2 Measurements

Variable	Controls	HYCH	POTS	p-value
Supine $EtCO_2$ (mmHg)	38.43 ± 1.74	36.18 ± 3.47	34.88 ± 3.92	<0.001
Orthostatic $EtCO_2$ (mmHg)	35.21 ± 1.60	28.60 ± 5.32	25.22 ± 5.28	<0.001
Max Δ $EtCO_2$ (orthostatic)	−3.98 ± 0.90	−11.98 ± 3.86	−12.95 ± 4.35	<0.001
Max % drop in $EtCO_2$	−10.33%	−33.25%	−37.31%	<0.001
Mean CBFv drop (orthostatic)	−8.57%	−29.21%	−31.53%	<0.001
Preload failure (from iCPET)	0%	100%	100%	—

Clinical Application:

Protocol:

1. Measure baseline $EtCO_2$ **supine after 5 min rest**
2. Measure $EtCO_2$ **after 5–10 min standing or tilt**
3. Monitor for:
   - **$\Delta EtCO_2$ ≥10 mmHg**
   - Absolute $EtCO_2$ **<30 mmHg**
   - Symptoms of **lightheadedness, dizziness, visual fog**

Significant drop in $EtCO_2$ during standing is a marker of **preload failure and cerebral vasoconstriction**, **even in the absence of tachycardia**

Source: Novak P, et al. "Orthostatic intolerance with tachycardia and without represent a spectrum of the same disorder." Frontiers in Neurology. 2024.

Journal Pre-proof

Concentration of Inflammatory Markers in Plasma of Varicose Ovarian Veins in Women With Pelvic Venous Disorders: A Pilot Study

Marcin Czeczelewski, Eryk Mikos, Sara Moqbil, Maciej Szmygin, Hanna Szmygin, Krzysztof Pyra

PII: S1078-5884(23)00456-2

DOI: https://doi.org/10.1016/j.ejvs

Reference: YEJVS 8824

To appear in: *European Journal of Vascul*

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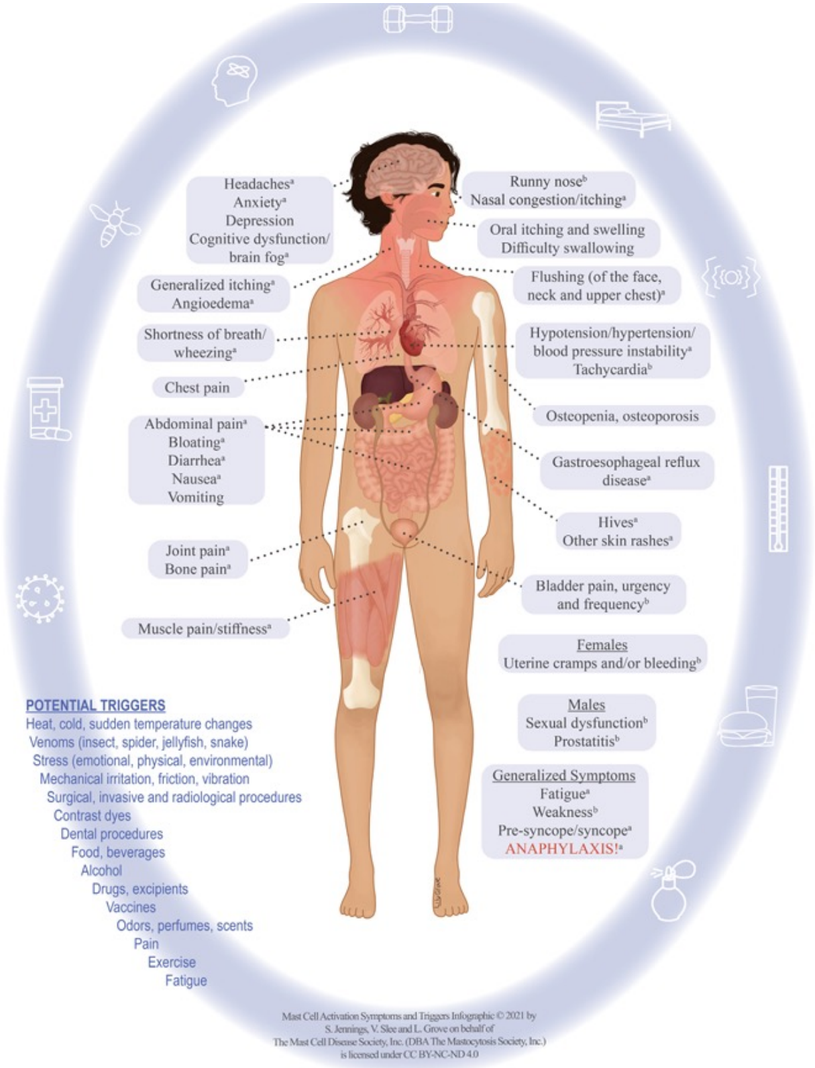
Accepted Date: 14 June 2023

Table 1. Comparison of blood counts and levels of inflammatory markers in samples from the antecubital vein and pelvic varicose vein of 25 patients of reproductive age referred for endovascular treatment of pelvic venous disorder.

Variable	Antecubital vein $n = 25$	Pelvic varicose vein $n = 25$	p
Erythrocytes – 10^{12}/L	4.02 (3.17 – 4.91)	4.23 (3.30 – 4.86)	.002
Leukocytes – 10^{9}/L	4.62 (2.98 – 6.52)	5.21 (3.30 – 8.34)	.001
Platelets – 10^{9}/L	220.5 (163 – 279)	237 (196 – 302)	.005
Haemoglobin – g/dL	12.00 (9.70 – 15.20)	12.95 (10.80 – 15.40)	.001
D-dimer – pg/mL	9 884 (2 259 – 16 910)	13 330 (3 099 – 114 110)	.081
C-reactive protein – mg/L	0.15 (0.12 – 2.93)	0.24 (0.10 – 3.04)	.038
Fibrinogen – g/L	2.25 (1.7 – 4.50)	2.25 (1.60 – 3.60)	.410
Interleukin-6 – pg/mL	50.88 (36.58 – 87.16)	57.14 (40.03 – 79.38)	.038
von Willebrand – ng/mL	36.28 (21.15 – 168.6)	45.43 (20.68 – 89.00)	.442

Data are presented as median (range).

Mast Cell Activation Syndrome



igure 1. The most common presenting symptoms and potential triggers of mast cell activation.[1,2,4] Symptoms and triggers are unique to the individual. Specific criteria, as noted in he article text, must be met to fulfill a diagnosis of MCAS. Not all patients react to each of the listed triggers or experience each of the listed symptoms. Mast Cell Activation Symp-oms and Triggers Infographic 2021 printed with permission from The Mast Cell Disease Society, Inc (DBA The Mastocytosis Society, Inc). The superscript letter "a" indicates symp-oms reported by more than 45% of TMS MCAS survey respondents as affecting them either moderately or severely in the course of their illness with MCAS.[5] The superscript letter b" indicates symptoms not queried in the TMS MCAS survey. DBA, doing business as; MCAS, mast cell activation syndrome; TMS, The Mast Cell Disease Society, Inc.

Mast Cells in the Tunica Externa of the Large Vein Walls

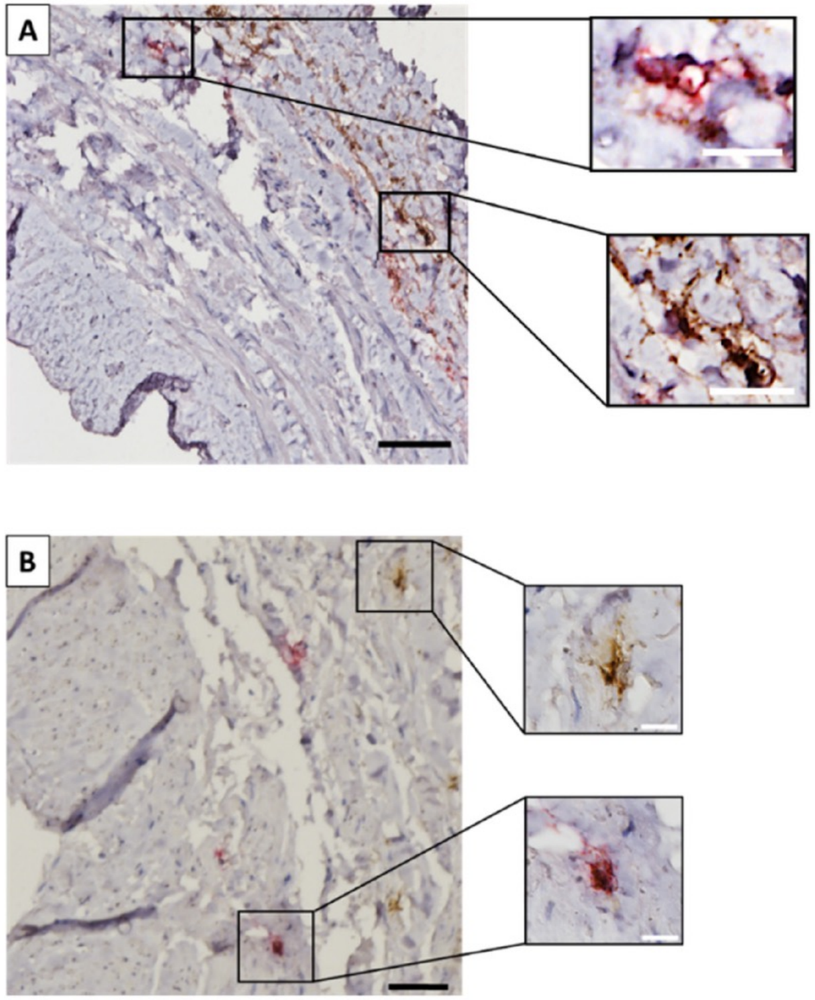
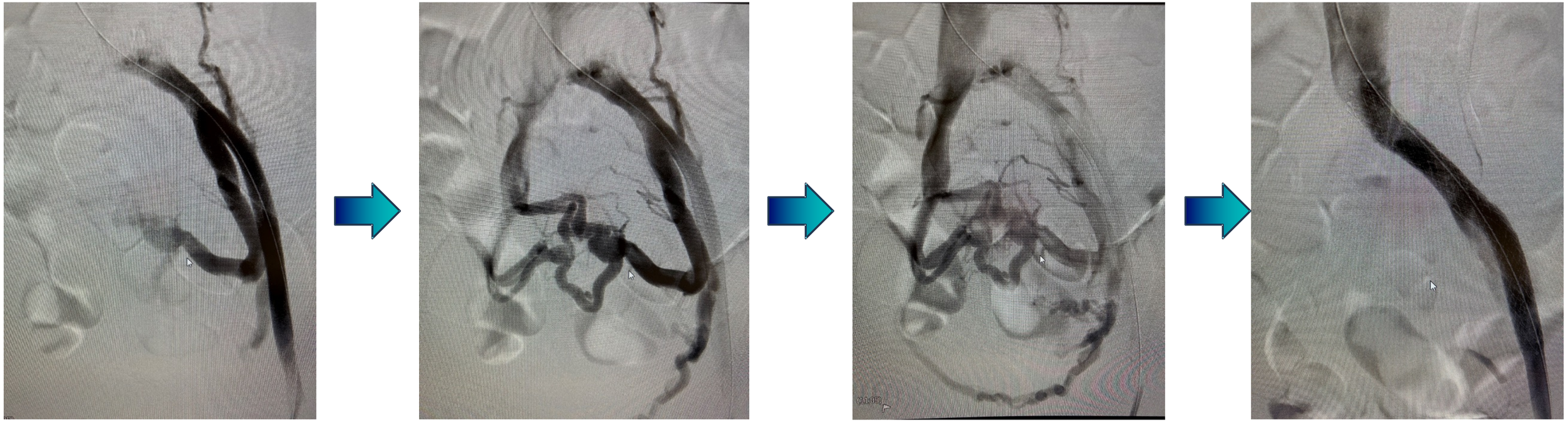


Figure 1. Localization and distribution of mast cells (MCs) in vein. (A,B): Illustration of MC localization in human saphenous vein: several 4 µm sections of healthy saphenous vein (hSV, (A)) or varicose saphenous vein (vSV, (B)) were stained with double horseradish peroxidase staining. Red staining: MCT. Brown staining: MCTC. Black bar: 100 µm, white bar: 10 µm. (C): Distribution of MCT and MCTC in both hSV (n = 3) and vSV (n = 5). (D): Overview of total MC counted per mm2 in hSV (n = 3) and vSV (n = 5). * p < 0.05, n.s.: non-significant p-value.

Callesen, K.T.; Mogren, S.; Berlin, F.; Andersson, C.; Schmidt, S.; Klitfod, L.; Esteban, V.; Poulsen, L.K.; Jensen, B.M. Characterization of Mast Cells from Healthy and Varicose Human Saphenous Vein. *Biomedicines* **2022**, *10*, 1062. https://doi.org/10.3390/biomedicines10051062

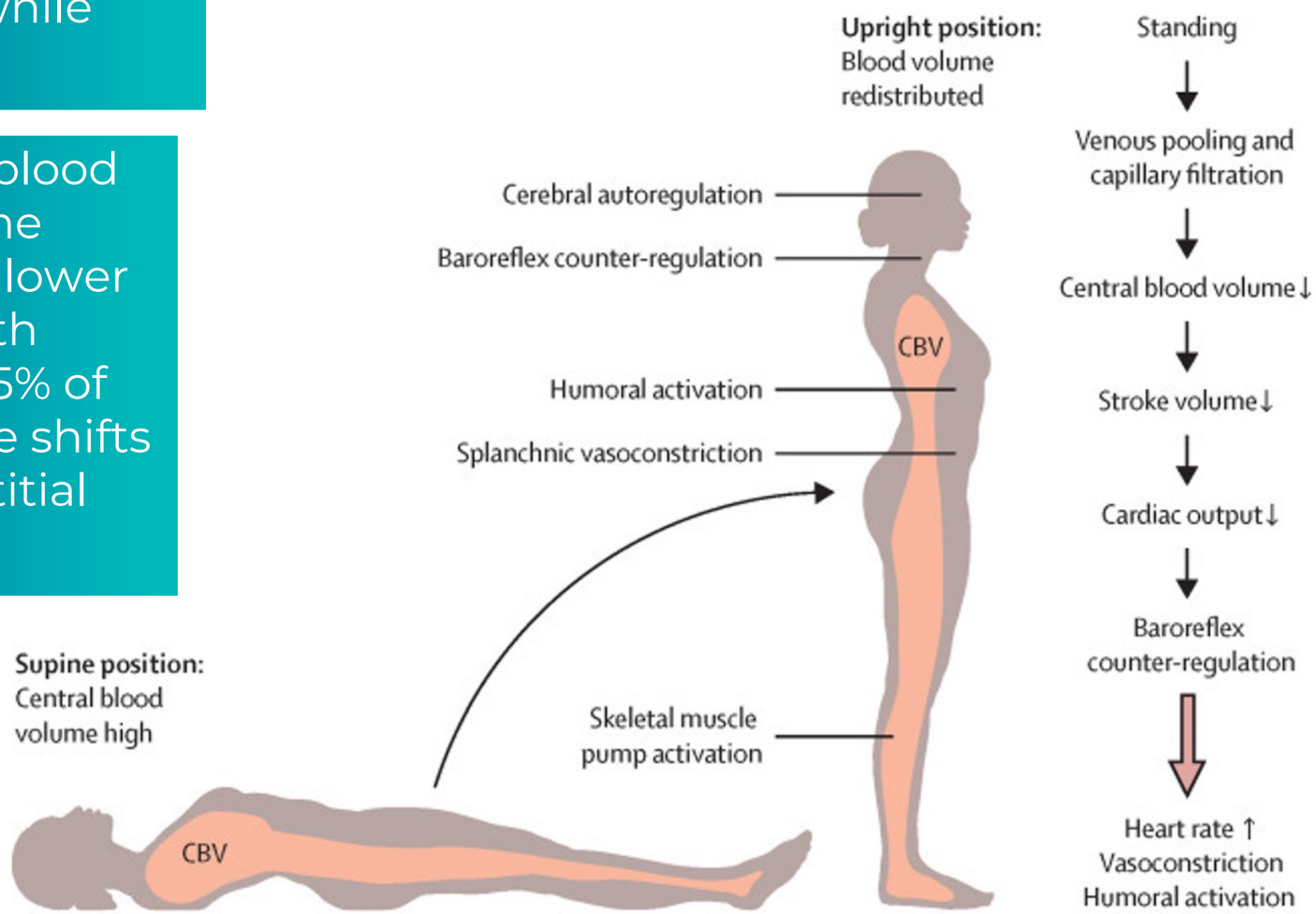
Venography of Iliac Vein and Intervention



OVERVIEW

30% of blood volume is in the thorax while supine

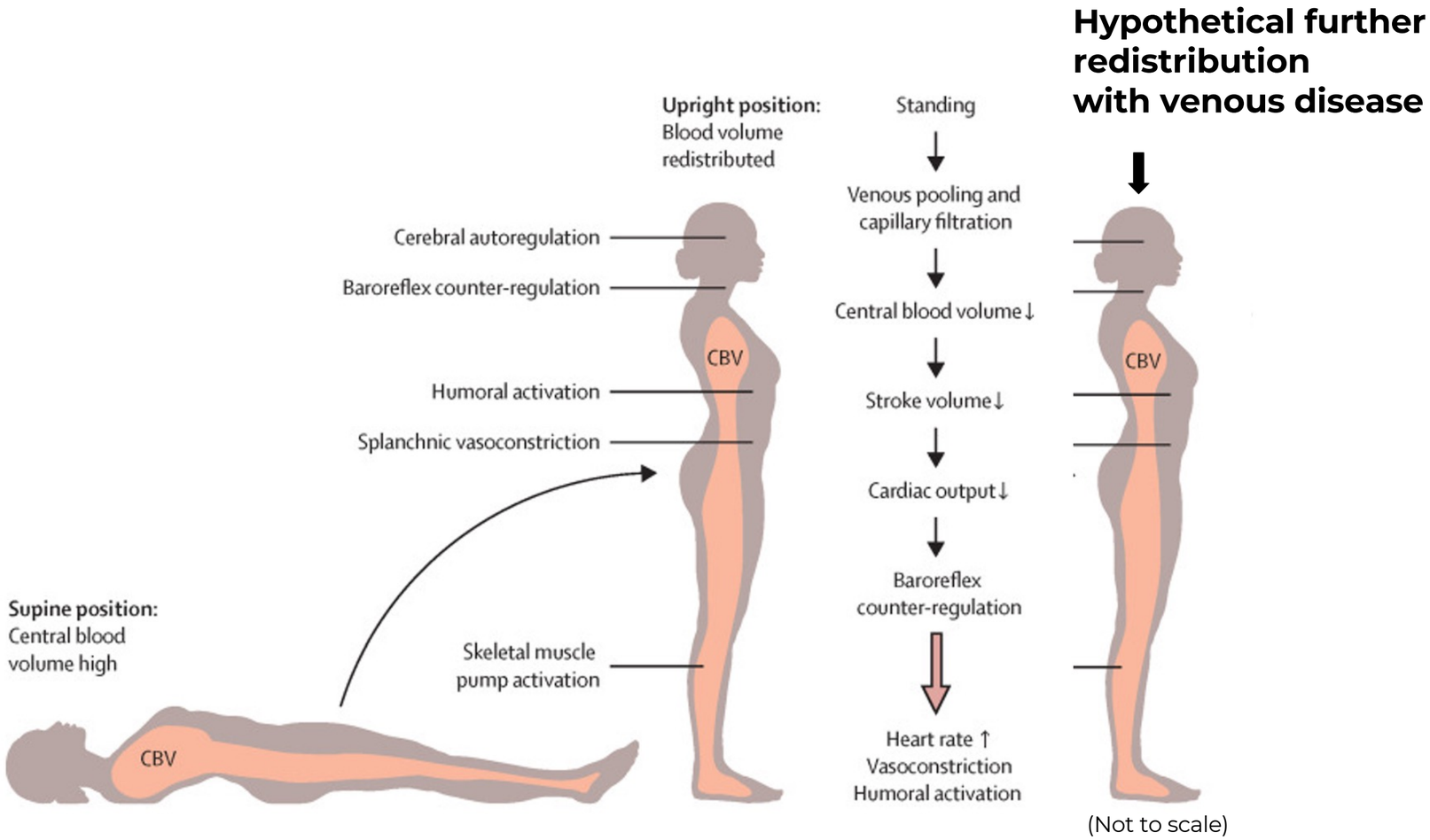
300-800ml of blood gravitates to the abdomen and lower extremities with standing (10-25% of plasma volume shifts into the interstitial tissues)



In normal patients this results in10-15 bpm increase in HR and increase in diastolic BP of 5-10mmHg

W Weiling MD et al. Diagnosis and treatment of orthostatic hypotension. The Lancet Neurology. Volume 21, Issue 8, August 2022, Pages 735-746

OVERVIEW



Adapted from: W Weiling MD et al. Diagnosis and treatment of orthostatic hypotension. The Lancet Neurology. Volume 21, Issue 8, August 2022, Pages 735-746

Another Phenotype: ILIAC VEIN COMPRESSION

Non-Invasive Interventions to help

Medications:

- Anticoagulation/Antiplatelet
  - Low Dose Aspirin and DOAC (Eliquis)
  - Pentoxifylline (Increases Oxygen Delivery/Antithrombotic/Antiplatelet)
- Mast Cell Stabilization:
  - H1 and H2 Blockade (Xyzal/Allegra plus Famotidine)
  - Mast Cell Stabilizers
    - Compounded Oral Ketotifen 1mg Twice a day
    - Cromolyn Sodium (Best for MCAS with GI issues)
    - Quercetin(High Dose 1gram to 2 Grams/Turmeric/Curcumin/Black Pepper)
- Endothelial Repair:
  - Diosmin 500mg-750mg Daily; Pycnogenol 50mg Twice a day
  - SGLT-2i (Jardiance etc)
- Increase Intravascular Volume:
  - Salt/Hydration
  - Fludrocortisone

Another Phenotype: ILIAC VEIN COMPRESSION

Non-Invasive Interventions to help

Physical Interventions: **Venous System is LOW Pressure and w/o a PUMP**

- Increase Venous Return:
  - Low Extremity Movement = Venous Pumping
    - Walking
    - Recumbent Biking or Rowing: (Horizontal Means Lower Pressures to Work Against)
      - IVC Pressure Lying Down: 8-10mmhg versus Standing Up: 20-22mmhg
    - Typically Feel almost Normal in Water (Bath, Pool, Ocean)
      - Buoyance of Water Decrease Gravitational Gradient
      - Weight of Water Acts Like Whole Body Venous Compression Device as Weight of Water Augments Venou Return.
  - Compression Stockings: (Ok but Not Dynamic: Veins Muscle work in concert with Muscle Pumping)

Non-Invasive Interventions to help

Physical Interventions: **Venous System is LOW Pressure and w/o a PUMP**

- Intermittent Compression Devices:
  - For 20-30 Minutes Prior to Bedtime and 20-30 Min upon Waking or for Recovery
  - Remember legs (aka venous Pump) moves Minimally During (Normal) Sleep
  - We Sleep Horizontal because of the Venous System not the Arterial System

- Dynamic Muscle Stimulation Devices:
  - For use when sitting for Long Periods, Travel, or Recovery from Exertion/Exercise
  - Firefly Muscle Recovery

Another Phenotype: ILIAC VEIN COMPRESSION

Non-Invasive Interventions to help

Physical Interventions: **Venous System is LOW Pressure and w/o a PUMP**

- Current Trial Using High Frequency Ultrasound Technology Directly on Iliac Vein
  - Weekly to Biweekly Treatments in those with Mild to Moderate Compression
  - So Far Outcomes have been Promising.

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Association and post-iliac vein stenting symptom improvement of postural orthostatic tachycardia syndrome and orthostatic intolerance with pelvic venous disorders: two retrospective studies

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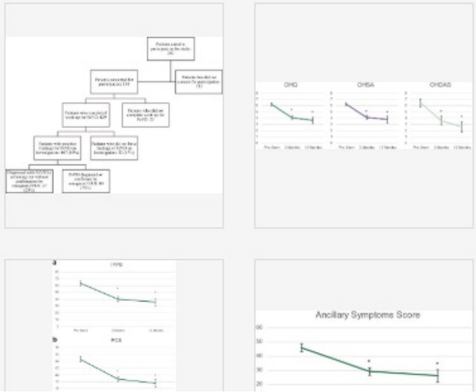
Summary

Background

Patients with Postural Orthostatic Tachycardia Syndrome (POTS) and Orthostatic Intolerance (OI) commonly present with symptoms suggestive of Pelvic Venous Disorders (PeVD). The presence of PeVD may contribute to orthostatic symptoms in these patients due to venous obstruction with stasis and pooling. Iliac vein compression, a PeVD, has historically been treated with iliac venous stenting. The authors hypothesize that patients with POTS frequently have findings of PeVD, that venous outflow obstruction from iliac vein compression exacerbates POTS/OI symptomatology, and that treating PeVD improves POTS/OI symptoms.

Figures (7) Figure Viewer



Pelvic Venous Compression in Patients with Postural Orthostatic Tachycardia Syndrome

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Introduction

Patients with Postural Orthostatic Tachycardia Syndrome (POTS) commonly present with orthostatic intolerance along with pelvic pain, lower back pain, leg pain/edema, urinary symptoms, dysmenorrhea/menorrhagia, and gastrointestinal symptoms. This constellation of symptoms suggests evidence of pelvic venous disease that may contribute to orthostatic symptoms in these patients, due to venous obstruction with stasis and pooling. We hypothesized that patients with POTS frequently had findings of pelvic venous disease.

Methods

Patients formally diagnosed with POTS were assessed for pelvic venous compression via pelvic venous ultrasound, MR angiography or venography, CT angiography or venography, and/or venogram with intravascular ultrasound.

Results

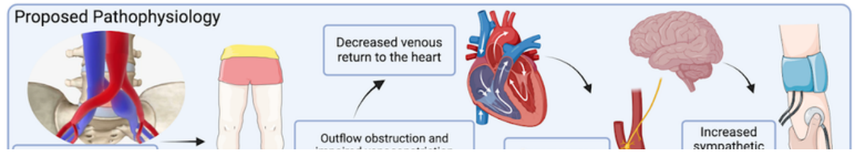
94 patients with a diagnosis of POTS presenting to a single healthcare center were screened. 21 patients were excluded from analysis due to incomplete work up with pending imaging results. The remaining 73 patients (93% female) were assessed for pelvic venous disease. Symptoms of pelvic venous disease, documented in the patient's chart, were common in this population (Table). 56 patients had confirmed pelvic venous compression (iliac vein, renal vein or both) on imaging with at least one modality.

Summary and Conclusions

77% of our POTS patients were diagnosed with pelvic venous compression, suggesting an association between the two diagnoses. Future studies should investigate the progression of POTS outcomes and pelvic imaging findings after intervention, such as stenting, to alleviate the pelvic venous compression.

Discussion

Figure: Proposed relationship between Pelvic Venous Disease and Postural Orthostatic Tachycardia Syndrome



Results

Table: Frequency of Symptoms and Diagnosis of Pelvic Venous Compression in Patients with Postural Orthostatic Tachycardia Syndrome

	Number of patients (N=73)
Pelvic pain or heaviness	45 (62%)
Lower back pain	21 (29%)
Lower extremity pain	36 (49%)
Lower extremity edema	43 (59%)
Lower extremity discoloration	14 (19%)
Varicose veins	19 (26%)
Urinary symptoms (frequency/urgency/dysuria)	42 (58%)
Dysmenorrhea/menorrhagia (N=68)	35 (51%)
Upper gastrointestinal symptoms (heartburn/nausea)	49 (67%)
Lower gastrointestinal symptoms (constipation/diarrhea)	44 (60%)
Pelvic venous compression confirmed on imaging	56 (77%)

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Symptomatic Improvement in Orthostatic Intolerance and Postural Orthostatic Tachycardia Syndrome and Pelvic Pain after Iliac Vein Stenting

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