# Chronic Venous Insufficiency in a 55-Year-Old Female: Highlighting Overlooked Cases for Increased Awareness

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SOUHRN

**Úvod:** Chronická žilní insuficience (chronic venous insufficiency, CVI) je časté onemocnění s postižením toku krve žilami vedoucí k žilní hypertenzi. Tato dysfunkce je často výsledkem selhání funkce chlopní v důsledku varixů (křečových žil), poškození hlubokých žil nebo slabých žilních stěn v důsledku nedostatku kolagenu a elastinu. Chronická žilní insuficience se projevuje dilatací žil, otoky dolních končetin, bolestí a změnami kůže. Přes tyto významné důsledky zůstává CVI nedostatečně diagnostikována a léčena, proto často progreduje do postflebitického syndromu a tvorby žilních vředů. Cílem této studie bylo zdůraznit prevalenci CVI u této vysoce rizikové populace a popsat účinná preventivní opatření a léčebné strategie pro snížení počtů komplikací a zlepšení výsledného stavu pacientů.
**Popis případu:** V únoru 2024 se k lékaři dostavila 55letá žena s progredujícími otoky a ulcerací na levé noze; tento stav se zhoršoval již tři měsíce. Otoky zhoršující se při delším stání a zmírňující se při zvednutých dolních končetinách byly doprovázeny bolestí a pocitem těžkých nohou. Podobné symptomy měla nemocná již o šest měsíců dříve; ty se zmírnily krátkodobou léčbou. Fyzikální vyšetření prokázalo tuhý edém a odhalilo vřed o rozměrech 10 × 10 cm na levé dolní končetině. Hodnota indexu tělesné hmotnosti (BMI) pacientky byla 28,23, krevní tlak 130/80 mm Hg a kromě popsaných obtíží nebyla anamnéza ženy ničím významná. Ultrazvuk prokázal žilní nedostatečnost v nepřítomnosti obstrukce.
**Diskuse:** Chronická žilní insuficience vzniká na podkladě inkompetence žilních chlopní a vede k žilní hypertenzi a poškození tkání. Postižení progreduje z žilní stáze do změn kůže a tvorby vředů. Symptomy pacientky odpovídaly typickým projevům CVI a zhoršovaly je nadváha a skutečnost, že se jednalo o ženu. Účinná léčba je založena na konzervativních opatřeních, jako jsou používání kompresních punčoch, zvedání dolních končetin a změny životosprávy spolu s pokročilými léčebnými metodami, jako jsou skleroterapie, termální ablace a v případě potřeby chirurgická intervence. Popsaný případ podtrhuje význam časného stanovení diagnózy a komplexní léčby pro prevenci progrese a zlepšení výsledného stavu pacienta.
**Závěr:** Chronická žilní insuficience, která je často nerozpoznána, vyžaduje časnou intervenci, aby se předešlo závažným komplikacím. Popsaný případ podtrhuje nutnost zvýšené pozornosti a proaktivní léčby, zvláště u vysoce rizikových populací. Pro účinnou léčbu a zlepšení výsledného stavu pacientů jsou nezbytné komplexní léčebné strategie včetně úpravy životosprávy a použití pokročilých léčebných metod.



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ABSTRACT

**Introduction:** Chronic venous insufficiency (CVI) is a prevalent condition characterized by impaired venous flow leading to venous hypertension. This dysfunction often results from valve failure due to varicose veins, deep vein damage, or vein wall weakness influenced by altered collagen and elastin. CVI manifests as dilated veins, leg edema, pain, and skin changes. Despite its significant impact, CVI remains underdiagnosed and undertreated, often progressing to postphlebitic syndrome and venous ulcers. This study aims to highlight the prevalence of CVI in this high-risk population and to provide insights into effective preventive measures and management strategies to reduce complications and improve patient outcomes.

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**Case illustration:** A 55-year-old female presented in February 2024 with progressive swelling and ulceration of the left leg, worsening over three months. The swelling, exacerbated by prolonged standing and relieved by elevation, was accompanied by a heavy, painful sensation. An earlier episode of similar symptoms six months prior had improved with short-term treatment. Physical examination revealed non-pitting edema and a 10 × 10 cm ulcer on the left leg. The patient had a BMI of 28.23, blood pressure of 130/80 mmHg and no significant medical history aside from the current condition. Ultrasound showed venous insufficiency without obstruction.
**Discussion:** CVI is caused by venous valve incompetence leading to venous hypertension and tissue damage. This condition progresses from venous stasis to skin changes and ulcer formation. The patient's symptoms align with CVI's typical presentation, exacerbated by her overweight status and gender. Effective management includes conservative measures like compression stockings, leg elevation, and lifestyle changes, along with advanced treatments such as sclerotherapy, thermal ablation, and, when necessary, surgical intervention. The case underscores the importance of early diagnosis and comprehensive management to prevent progression and improve patient outcomes.
**Conclusion:** CVI, often under-recognized, requires timely intervention to prevent severe complications. This case highlights the need for heightened awareness and proactive treatment, especially in high-risk populations. Comprehensive management strategies, including lifestyle modifications and advanced therapies, are essential for effective treatment and improved patient outcomes.

## Introduction

Chronic veins insufficiency (CVI) is a condition where venous flow is disrupted which causes venous hypertension.[1] This is caused by failure of venous valve function due to varicose veins or damage to the deep veins secondary to venous thrombosis, trauma or venous obstruction.[2] Another study shows that apart from valve failure, weakness of vein's walls is more influential in causing CVI which is caused by changes in the composition of collagen and elastin.[3] The major clinical manifestations of chronic venous insufficiency (CVI) are dilated veins such as telangiectasias, reticular veins, varicose veins, leg edema, pain/achiness/heaviness, and skin alterations.[4] Despite its potentially fatal impact, CVI has been underdiagnosed and undertreated for a long time which is usually progressive and leads to postphlebitic syndrome and venous ulcers.[1,2] The prevalence of CVI is higher in women, 25–40% and in contrast 10–20% in men, with an annual incidence of 2–6% in women and 1.9% in men.[5] This is believed to occur due to various factors, including the influence of the hormone estrogen and pregnancy.[3,4] Other risk factors for this condition include older age over 30, heavy lifting, multiple pregnancies, oral contraceptive, obesity, sedentary lifestyle, prolonged standing occupation, history of leg trauma, family history, and prior history of deep venous thrombosis.[1,2,4] Furthermore, hereditary factors are also considered to play an important role in the development of CVI, such as the genetic disorders Klippel–Trenaunay and Parkes–Weber.[1]

Initial management of CVI is conservative treatment, such as compression stockings with a tension between 30–50 mmHg which can improve pain, edema and pigmentation.[1,2,4,6] Advanced CVI that damages the surface of the skin/ulcers can be treated with wound compression bandaging systems, topical moisturizers, and other wound care.[1,2,4,6] Other conservative treatments include leg elevation, weight management, and exercise.[1,4] Sclerotherapy using sclerosing agents include the hypertonic solution of sodium chloride (23.4%), polidocanol, sodium iodide, chromated glycerin, sodium tetradecyl sulfate, and sodium morrhuate is beneficial in treating telangiectasias, reticular veins, varicose veins (1–4 mm diameter), and veins with reflux.[1,6] It can be used as a primary treatment or in combination with other treatments.[1,6] For patients with GSV reflux, ablation utilizes thermal energy through radiofrequency or laser results in complete obliteration in 85% of patients after two years.[6] For iliac vein stenosis and occlusion, endovascular stenting can be used.[6] Sulodexide and pentoxifylline for adjuvant to compression therapy is an effective pharmacological therapy which targets multiple sites involved in the pathogenesis of CVD.[2,4,7] In individuals with persistent discomfort and disability after pharmacological or endovenous therapy, surgical surgery for CVI may be considered in addition to compression stockings.[1,2,6] The surgical method used depends on the pathophysiological process underlying the CVI, and may consist of stripping, simple ligation/division, and venous valve reconstruction.[1,2,6]

## Case description

In February 2024, a 55-year-old outpatient female with complaints of a wound and swelling on the left lower leg for three months was brought to the RS Universitas Airlangga. The swelling was localized to the left leg, with no involvement of the right leg. It began gradually, initially affecting the dorsum of the left foot and progressively extending upward below the knee over the last two months. The wound appeared suddenly and has progressively enlarged over the past month. The swelling worsens when the patient stands for extended periods or walks, and reduces when she sits and elevates her leg. Initially, the swelling would decrease to nearly match the size of her right leg, but over time it became persistent and difficult to reduce. The patient reported that her left leg feels heavier, with pain worsening as the swelling increased, making walking difficult. There was no associated redness, skin discoloration, or additional wounds on the affected limb.

Six months earlier, the patient had a similar episode of swelling and pain in the left leg, although the symptoms were milder. At that time, an ultrasound examination identified venous issues without obstruction. The patient was treated for five days, resulting in improve-

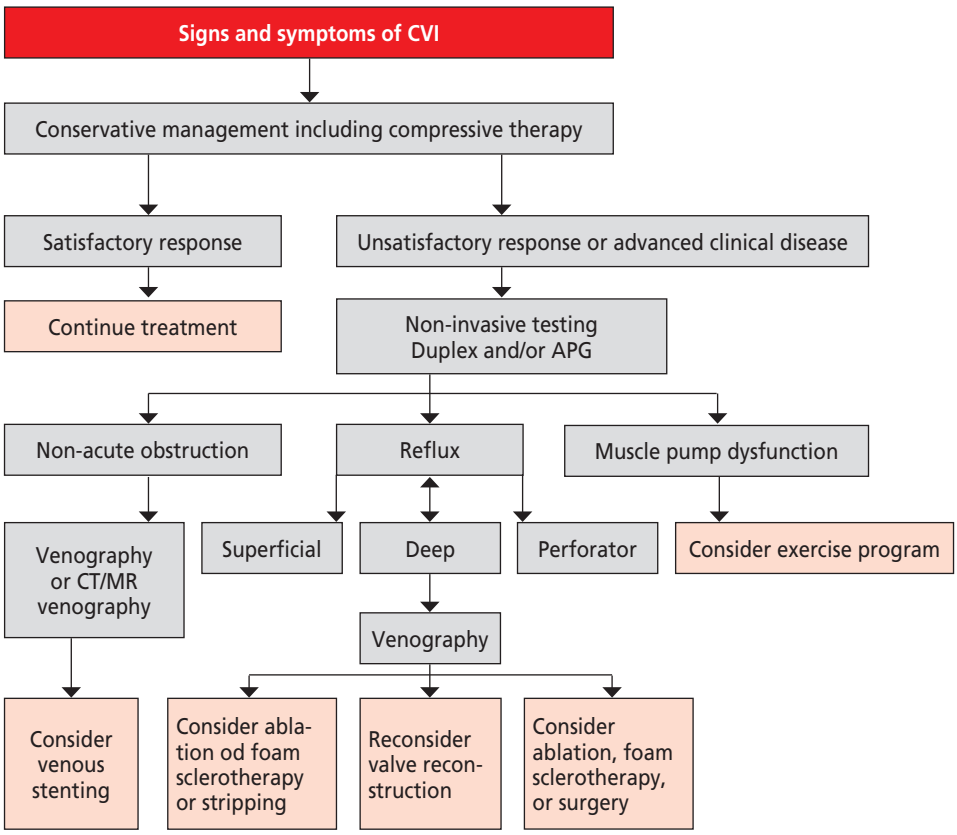


**Fig. 1 – Algorithm for diagnosing and treating CVI.[2]**

ment, after which she was discharged. However, she has not been on any routine medications for this condition. The patient's medical history is otherwise unremarkable, with no history of diabetes mellitus, hypertension, stroke, heart disease, or kidney disease.

On physical examination, the patient appeared moderately ill, with a Glasgow Coma Scale score of 15. Her vital signs were stable, with a blood pressure of 130/80 mmHg, pulse rate of 88 beats per minute, respiratory rate of 18 breaths per minute, and a body temperature of 36.8 °C. The patient had a BMI of 28.23 (overweight). The general examination revealed a normocephalic head, evenly distributed black hair, and symmetrical facial features. Eye examination showed no eyelid edema, normal conjunctiva and sclera, clear cornea, and equal, reactive pupils. The ears and nose were normal, with no hearing loss or septal deviation. The oral cavity and pharynx were unremarkable, and there was no thyroid enlargement or tracheal deviation. Jugular venous pressure was within normal limits. Thoracic examination revealed normal chest configuration, with vesicular breath sounds bilaterally and no adventitious sounds. The heart had a regular rhythm with normal S1 and S2 sounds, and no murmurs. The abdomen was flat, with normal bowel sounds, no masses, and no tenderness. The extremities were warm, with non-pitting edema more prominent in the left leg. A 10x10 cm ulcer with irregular edges was present on the left lower leg, with no associated warmth, erythema, or varicose veins. Capillary refill time was less than 2 seconds, and there were no signs of cyanosis or varicosities. This case highlights a recurrent venous disorder with persistent non-pitting edema and an ulcer, requiring further evaluation and management.

## Discussion

### *Pathophysiology*

Chronic venous insufficiency (CVI) is a complex and progressive condition characterized by the inability of the venous system, particularly in the lower extremities, to efficiently return blood to the heart, caused venous to reflux. This inefficiency is largely due to venous valve incompetence, which leads to venous hypertension, venous stasis, and, over time, a cascade of pathological changes.[8,9] Venous hypertension is a critical factor in the development of CVI, as it results in increased pressure within the superficial veins. Over time, this leads to damage to the venous endothelium, capillary leakage, and subsequent inflammation, ultimately culminating in tissue damage, skin changes, and ulcer formation, as seen in the patient discussed here.[8–10] The impaired valve function allows blood to pool in the lower extremities, which increases the pressure within the veins and leads to distention and leakage into the surrounding tissues. This is compounded by a failure in the calf muscle pump, which is essential for

venous return, especially during activities such as walking.[9,11]

The resultant inflammatory response involves leukocyte activation and adhesion, increased expression of inflammatory mediators (e.g., cytokines like TNF-α and IL-6), and matrix metalloproteinases (MMPs), which degrade the extracellular matrix and contribute to the breakdown of the skin and subcutaneous tissue. Persistent venous hypertension also leads to capillary permeability, causing protein-rich fluid to accumulate in the interstitial spaces (edema), which is a hallmark of CVI. Over time, this can lead to lipodermatosclerosis, atrophie blanche, and ultimately venous ulceration, as observed in the patient.[10,12]

### *Anamnesis*

The patient, a middle-aged female, presented with a history of unilateral lower limb swelling and ulceration that had progressively worsened over a three-month period. The symptoms were exacerbated by prolonged standing and relieved by leg elevation, a classic pattern in CVI. Notably, the patient had a history of similar, albeit milder, symptoms six months prior, suggesting a chronic and relapsing course typical of CVI. The ultrasound findings of venous insufficiency without evidence of occlusion align with the diagnosis of CVI, where valvular incompetence rather than thrombotic obstruction is the primary pathology.

The patient's demographic profile (female gender, middle age) and her body mass index (BMI of 28.23, indicative of overweight status) are significant as they are known risk factors for CVI. Hormonal factors, particularly in women, contribute to venous wall relaxation and valve dysfunction, which may explain the higher prevalence of CVI in females.[13,14] Additionally, obesity is a well-documented risk factor due to increased intra-abdominal pressure and its contribution to venous hypertension.[14]

### *Symptoms and manifestations*

Chronic venous insufficiency is a condition in which the veins in the lower extremities have a decreased ability to effectively return blood to the heart, leading to a buildup of blood in the leg veins. This results in common symptoms such as leg discomfort, swelling, varicose veins, and skin changes or ulceration.[15] As CVI progresses, patients typically experience venous leg discomfort characterized by heaviness, throbbing, dull pain, and swelling in the legs, which worsens with prolonged standing and is often accompanied by a sensation of pressure; relief is generally achieved through measures that reduce venous pressure, such as elevating the legs, using compression stockings, or walking.[15,16] However, approximately 20% of patients with other clinical features of CVI do not experience leg discomfort, while around 10% of patients may have leg discomfort as their only clinical symptom.[15]

Patients with CVI may present without any complaints, but often show signs such as simple telangiectasia (<1 mm in diameter), reticular veins (1–3 mm in diameter), and varicose veins (>3 mm in diameter).[15] Varicose veins are dilated superficial veins that become increasingly tortuous and enlarged. They can develop episodes of superficial thrombophlebitis, which are identified by painful, hardened, and inflamed areas along the affected vein.[17] Edema initially manifests in the perimalleolar region and progresses upward along the leg due to dependent fluid accumulation. This swelling can evolve from pitting edema to more induration, resulting in increased hardness and stiffness of the tissues.[16,17]

Evaluation of the legs in patients with CVI may reveal skin changes such as hyperpigmentation, stasis dermatitis, erythema, eczema, and venous ulcers (VLU). Hyperpigmentation is thought to be caused by hemosiderin or melanin deposition due to venous hypertension. VLU represents a significant medical issue caused by CVI, driven by increased venous pressure, which may result from venous occlusion and/or varicose veins. The healing process for VLUs can span from a few months to several years, with approximately 25% remaining unhealed. These ulcers are the most prevalent chronic leg ulcers among the elderly population.[8] Advanced skin manifestations of CVI include atrophie blanche (ulcers with white scars), corona phlebectatica (widening of the superficial veins around the ankle), and ulceration around the medial and lateral malleolus.[15,17] Additionally, patients with CVI often exhibit lipodermatosclerosis, a fibrotic process affecting the dermis and subcutaneous fat, which is linked to an increased risk of cellulitis, leg ulceration, and delayed wound healing.[15,17]

Furthermore, CVI may lead to the development of lymphedema, with Stemmer's sign being a notable clinical feature of this condition. Approximately one-third of CVI cases result in secondary lymphedema, known as phlebolymphedema, which may improve or resolve with the correction of the underlying CVI.[16,17]

While the CEAP scoring system provides a valuable framework for classifying the severity of CVI, it does not offer a specific algorithm to definitively distinguish CVI from other conditions.[17] This lack of a precise diagnostic tool often leads to delayed diagnosis and suboptimal treatment. In the future, it is crucial to refine diagnostic algorithms to distinguish CVI from other conditions, especially those identifiable through straightforward physical examination methods. This focuses on distinctive, easily detectable signs could enhance for early diagnosis and treatment.

## Conclusions

Chronic venous insufficiency is a prevalent but often under-recognized vascular disorder with significant clinical implications, including the development of venous ulcers and chronic leg swelling. This case study highlights the importance of early recognition, accurate diagnosis, and comprehensive management of CVI to prevent progression and complications. The patient's presentation, characterized by recurrent leg swelling and ulceration, underscores the progressive nature of untreated CVI. Effective management strategies, including the use of compression therapy, lifestyle modifications, and when necessary, advanced interventions like sclerotherapy or surgical procedures, are crucial in improving patient outcomes. Given the higher prevalence of CVI in women and those with risk factors such as obesity, targeted awareness, and proactive treatment approaches are essential to

reduce the burden of this condition. This study emphasizes the need for increased awareness among clinicians and patients alike to ensure timely intervention and to bridge the gap between seen and unseen cases of CVI.

**Conflict of interest**
The authors declare no conflicts of interest.

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**Ethical statement**
The work was carried out in accordance with the Declaration of Helsinki.

**Informed consent**
The patient gave consent to publishing of the case report.

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