An Overview of Stasis Dermatitis: Addressing Treatment Challenges in Family Practice

[1]Dr. Sharon Joseph., [2]Dr. John Abraham., [3]Dr. Clement Prakash., [4]Dr. Ganesh V., [5]Dr. Romate John., [6]Dr. Anieta Merin Jacob., [7]Dr. Ronnie George., [8]Dr. Ambika Akhoury

[1]Senior Resident, Department of Dermatology, St. John's National Academy of Health Sciences, Bengaluru

[2]Assistant Professor, Department of Family Medicine, St. John's National Academy of Health Sciences, Bengaluru

[3]Associate Professor and Unit Head, Department of Surgery, St. John's National Academy of Health Sciences, Bengaluru

[4]Assistant Professor, Department of Orthopedics, St. John's National Academy of Health Sciences, Bengaluru

[5]Professor, Department of Psychology, Central University of Karnataka, Gulberga

[6]Assistant Professor, Department of Oral Medicine and Radiology, Sri Venkateshwara Dental College and Hospital, Bengaluru

[7]Senior Resident, Department Of Orthopedics, AIIMS Jodhpur

[8]Intern, Atal Bihari Vajpayee Medical College and Research Institute, Bengaluru

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ABSTRACT

Stasis dermatitis (SD), also known as venous eczema or gravitational dermatitis, is a chronic inflammatory skin condition primarily affecting the lower extremities due to chronic venous insufficiency (CVI). This condition is characterized by edema, hyperpigmentation, and the risk of venous ulcers, leading to a significant burden on patients and healthcare systems. Despite its prevalence, SD is often misdiagnosed or overlooked in primary care settings, delaying treatment and increasing the risk of complications. This review aims to bridge the knowledge gap by providing a comprehensive evaluation of SD, including its epidemiology, pathophysiology, clinical manifestations, diagnostic challenges, and management strategies.

Keywords: Stasis Dermatitis, Venous Ulcer, Chronic Venous Insufficiency, Venous Clinical Severity Score, Clinical-Etiology-Anatomy-Pathophysiology, Metalloproteinases, Lipo-dermatosclerosis, Deep Vein thrombosis.

INTRODUCTION

Venous hypertension also referred to as sustained ambulatory venous pressure, is brought on by venous reflux at the lower extremities and results in chronic venous insufficiency.[1,2] The clinical spectrum of lower extremity chronic venous insufficiency frequently includes stasis dermatitis (venous eczema, stasis eczema or gravitational dermatitis). It can appear as an early indicator of chronic venous insufficiency, but it may persist or return at any stage and is frequently most noticeable in the presence of ulcers.[3]

Chronic venous insufficiency (CVI) patients frequently report pain, discomfort, itching, and occasionally severe heaviness in the affected leg's calf. One of the first features of chronic venous insufficiency is varicose

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veins.[4] On examination its noted as erythematous scaly patches and plaques over bilateral lower legs with overlying pigmentation.[5] Histopathological study of skin samples based on chronicity reveals hyperkeratosis, focal parakeratosis, acanthosis, and spongiosis. Dermal changes include edema, perivascular lymphocytic infiltration, hemosiderin-laden macrophages, and proliferation of dilated small blood vessels with fibrosis of vessel walls.[2,6] Typically, clinical signs and medical history are used to diagnose SD. The classification of the subjects was done based on the CEAP methodology and Venous Clinical Severity Score(VCSS) Chart 2 , which takes into account clinical, etiological, anatomical, and pathophysiological data.

Different ethnic groups and communities have varying rates of chronic venous insufficiency. About 15% of adults in Central Europe experience symptoms of chronic venous insufficiency, and 1% get venous ulcers. Age-related increases in prevalence are evident.[6] In the United States, between two and six million people suffer from advanced types of chronic venous insufficiency.[2,7] In a study of patients with varicose veins who were 15 or older, SD was discovered in 1.4% of 773 people.[8,9] A study on 4099 patients, 65 years or older by Yalcin et al, noted a prevalence of 6.2%.[10] The prevalence in 584 older patients with a mean age of 80 years was found to be 6.9% and 5.9% in 68 patients with a mean age of 74 years has been reported in other research.[11,12]

Additional etiological factors, most notably contact sensitization to chemicals in topical therapy, may act in concert over time. As one of the most frequent reasons for secondary dermatitis dissemination, stasis dermatitis (SD) is a complicated, multifaceted illness.[6]

Epidemiology

Chronic venous disease, including chronic venous insufficiency, is a common disease that increases with age and is more prevalent in women. Geographically, the highest reported prevalence of chronic venous insufficiency is across Western countries, ranging from < 1 to 40% in women and from < 1 to 17% in men[63]. Approximately 37–44% of individuals with leg ulcers had been diagnosed with SD[64]

Pathogenesis:

The clinical signs of chronic venous insufficiency are believed to be caused by microvascular abnormalities and chronic inflammation that result from changes in the macrocirculation. The pathophysiology is believed to consist of two components: the first is aberrant venous blood flow with reflux, and the second is a chronic immune process that happens at the microvascular level leading to the skin changes associated with chronic venous insufficiency.

Venous reflux is thought to be the primary cause of venous diseases. Retrograde flow, such as that which occurs in a vein in reaction to a calf squeeze, is known as reflux flow. It occurs when standing and with valvular incompetence. In the lower extremities, it can happen in the superficial, deep, and perforating veins. The causes of CVI are venous diseases (valvular defects, deep venous thrombosis), impaired calf muscle pump function, and congestive heart failure.[13,14]

Since its discovery in the 1980s, the buildup of leukocytes in the microcirculation of extremities with venous hypertension has come to be recognized as a hallmark of the early phases of chronic venous insufficiency, especially in SD.[2,15] Leukocytes adhere to the endothelium of vein walls and valve leaflets, resulting in the necrosis and apoptosis of endothelium, smooth muscle cells, fibroblasts and parenchymal cells of the venous wall. The venous walls and valve leaflets weaken and are destroyed.[16,17]Venous hypertension causes erythrocyte extravasation leading to hemosiderin deposition and macrophage accumulation at site leading to further skin changes.[18] By releasing oxygen free radicals and MMPs, activated endothelium and inflammatory cells can break down extracellular matrix constituents such collagen, elastin, laminin, and fibronectin.[19,20] The hyperpigmentation in SD may be caused by MMPs and other types of inflammation in addition to hemosiderin accumulation (Chart 1).[14] This hallmark hyperpigmentation of the condition manifests as brownish discoloration of the skin. The prolonged presence of fluid in the tissues leads to inflammation, skin thickening, and, eventually, ulceration if left untreated initially.

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Signs and Symptoms of SD

Various signs and symptoms associated with SD include pain, Itching, Edema, reduced ankle range of motion, reduced physical activity Sleep disturbances. Patient also presents reduced concentration due to lack of sleep. Scratching the already impaired skin can further increase the risk of secondary infection and wound aggravation. The negative psychological consequence of this disease on patients were presented with anxiety, depression, and even suicidal mental status .

General Practice perspective and challenges:

Verhoeven et al, in their practice population-based study, found that skin conditions make up 12.4% of the entirety of health concerns addressed by family physicians. There exists a certain subset of individuals with complex skin conditions that approach dermatologists, but the vast majority receive treatment primarily from their family physician.[21]

Stasis dermatitis makes up a significant portion of those that seek care from a family physician, particularly the elderly demographic. This is attributable to the heightened pressure on superficial veins in older individuals, stemming from both calf muscle weakening as well as vessel wall deterioration. [9],[22] Western countries report higher rates than Asian populations.[9,22-24] In the United States, stasis dermatitis (SD) has been approximated to affect a staggering 6% of individuals over the age of 50, amounting to about 15 to 20 million patients, twice as many as psoriasis.[22,25,26]

Early identification and prompt treatment of chronic venous insufficiency (CVI) can potentially avert the crippling complications associated with the disease. However, patients tend to seek medical attention often when the disease has already progressed, and those who do seek help may run into a myriad of diagnostic challenges. Various factors can contribute to a delay in diagnosis (refer to Table 1). The reason why diagnosis is deferred is often because for symptoms to truly affect the activities of daily living it takes an immense amount of time. Often when reported, stasis dermatitis (SD) is already classified as stage C4. This stage indicates that venous insufficiency has advanced drastically enough to induce alterations in the skin, accompanied by lipodermatosclerosis (LDS), edema, and pruritus.[27] At this juncture in the disease, the treatment aims to mitigate the cutaneous manifestations.[32-34]

Table 1 - Factors that contribute to a delay in diagnosis[41]

Initial SD-related symptoms are not very bothersome.
Patients do not report symptoms because they regard skin diseases as a cosmetic nuisance or part of the aging process.
Dermatological disorders are often underestimated and overlooked.
Physicians tend to focus on priority chronic conditions if present.
SD-related symptoms may overlap with those of other chronic conditions present.
SD symptoms are similar to those of several other cutaneous disorders, which often results in the misdiagnosis of SD.
There is a lack of awareness and knowledge regarding SD among patients as well as healthcare professionals.
A Primary Care Physician is not present during the diagnostic process.

Diagnostic Challenges

The diagnosis of Stasis dermatitis (SD) hinges on the visible characteristics of the affected skin and the patient's medical history.[1,22] Physical examination often reveals erythema and eczematous patches, primarily on the lower legs. These symptoms commonly manifest around the medial ankle, and in advanced stages, the

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inflammation may extend nearly to the knee.[1,2,30] Other associated symptoms include pruritus, restless legs, cramping, tingling, swelling, and various manifestations of chronic venous insufficency (CVI). Dermal hemosiderin deposition manifests itself as hyperpigmentation.[1,2,28] As SD progresses, pain, oozing, and ulcerations become apparent imposing an increased burden on the patient.[22] If the history and physical examination yield inconclusive results, further assessments utilizing methods such as Doppler/duplex ultrasound are warranted to identify venous insufficiency.[1,2] This technique proves accurate, cost-efficient, and non-invasive, however, its interpretation calls for an advanced level of expertise and skill.[1,2,31] Moreover, its application is limited in patients exhibiting excessive vascular calcification or obesity.[2,31] In certain clinical scenarios, the involvement of a specialist may be necessary to make a diagnosis. Skin biopsies could also be employed to exclude other skin ailments.[32] A skin biopsy of an acute stasis dermatitis (SD)-related lesion comprises a superficial, perivascular lymphocytic infiltrate, along with epidermal and subepidermal edema, dilated dermal capillaries, and neovascularization. On the other hand, examination of chronic lesions may reveal epidermal acanthosis with hyperkeratosis. In advanced stages of SD, dermal fibrosis often intensifies and there is marked fibroblast proliferation and collagen fiber buildup in the dermis, leading to epidermal loss which manifests as a venous ulcer.

Similarities with Other Cutaneous Disorders

Beyond the mentioned diagnostic challenges, the dermatological changes characteristic of SD may closely mimic other skin diseases (Table 2).[22] This similarity can contribute to inaccurate diagnoses, leading to delayed or inappropriate treatment and, subsequently, heightened patient anxiety and financial strain. SD and cellulitis present similarly and are hence, commonly confused; 10–30% of cellulitis patients were misdiagnosed, with SD being the most commonly accurate diagnosis.[25,26]

Table 2 - Cutaneous Disorders closely mimicked by SD[1,2, 22]

Cellulitis/Erysipelas
Contact Dermatitis
Atopic Dermatitis
Psoriasis
Pigmented Purpuric Dermatoses
Xerotic Eczema
Vasculitis
Cutaneous T-cell Lymphoma

Recommendation for specialist referral in case of Red Flags

1) **Rapid Worsening or Spread**: If the eczema is rapidly worsening or spreading beyond the typical area (usually the lower legs), it could indicate an infection or another underlying issue.

2) **Signs of Infection**: Symptoms such as increased redness, warmth, swelling, or the presence of pus suggest a secondary bacterial infection..

3) **Severe Pain**: While stasis eczema can be uncomfortable, severe pain, especially if it is out of proportion to the visible skin changes, could indicate cellulitis or other deeper infections.

4) **Ulceration**: The development of ulcers or open sores in the affected area can be a sign of chronic venous insufficiency worsening or may lead to complications if not properly managed.

5) **Sudden Swelling of the Leg**: If one leg suddenly becomes more swollen than the other, it could indicate a deep vein thrombosis (DVT), which is a medical emergency.

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6) **Unusual Skin Discoloration**: Changes in skin color, such as becoming very pale, purple, or black, could suggest necrosis or severe vascular compromise.

7) **Non-Responsive to Treatment**: If the eczema is not responding to standard treatments, this may suggest the need for further investigation or a review of the diagnosis.

8) **New Onset in an Unusual Area**: If stasis eczema suddenly appears in an area that is not typically affected, it could indicate another condition, such as erythema nodosum or vasculitis.

9) **Systemic Symptoms**: Symptoms like fever, chills, or general malaise accompanying the stasis eczema might indicate a systemic infection or other serious conditions.

10) **Comorbid Conditions:** Poorly controlled diabetes mellitus, hypertension, coronary artery disease, congestive heart failure, chronic kidney disease, chronic liver disease, Chronic inflammatory bowel disease

Management of Medical and Surgical Considerations:

Elevating the affected legs and feet is rationally accepted. This helps reduce swelling by encouraging blood flow back towards the heart. Ideally, the feet should be elevated above the level of the heart several times a day or night. Current treatment goals for patients with stasis dermatitis (SD) aim to manage the clinical effects of underlying venous insufficiency, edema, and inflammation (itching and pain), as well as to enhance skin lesions and facilitate ulcer healing. [22] Primary treatment options for SD typically involve exercise, walking, and leg elevation, although these methods are typically effective only for mild cases of SD.[1,2] Pentoxifylline has been shown to be effective for the treatment of venous ulcers when added to compression therapy. Oral pentoxifylline is often used with or without compression therapy to reduce leg pain. These are inhibitors of platelet aggregation that reduce blood viscosity, thereby improving microcirculation. Micronized purified flavonoids fraction and Calcium dobesilate may play a supportive role in managing the condition by vascular protective effects. In conventionally, emollients or moisturizers can keep the skin hydrated and prevent cracking while corticosteroid creams reduce inflammation and itching.

The cornerstone of treatment for SD remains compression therapy through bandages or stockings, it is uses a high pressure of about 60 mmHg or a median pressure of about 30 mmHg which is effective. At present, the standard treatment approach involves applying highly potent glucocorticoids topically and, whenever feasible, permanent compression therapy. A pressure of approximately 60 mmHg or a median pressure of around 30 mmHg is typically recommended. [22, 27] Patients are treated with strategies such as avoiding positions that promote venous stasis, using elastic wraps, applying Unna's paste boots, wearing support hose, and utilizing graduated compression stockings.[28]

If all aforementioned interventions fall short, surgical methods are adopted. Typically, the lesion is targeted using a medial approach. With the patient in a slight Trendelenburg position, a longitudinal incision is made medially, starting from the subgeniculate area near the condyles and extending downward to encompass the ulcer if present, or through the healed ulcer bed if feasible. The incision penetrates through the skin, subcutaneous tissue, and the crural fascia. Dissection continues in the subcrural plane anteriorly to the edge of the tibia and posteriorly until the lesser saphenous vein is included. All perforating veins are identified, ligated, and divided. [29]

Surgery for varicose veins and its sequelae should be individualized according to the patient's preoperative evaluation. A combination of ligation, axial stripping, and stab phlebectomy may be applied as needed to the GSV, SSV, tributary veins, and perforating veins. Classic open surgical techniques have been replaced with minimally invasive procedures, such as ultrasound-guided foam sclerotherapy, endovenous thermal ablation, and ambulatory phlebectomy. Compared with conventional techniques, minimally invasive procedures are less painful, are associated with a lower number of complications, are more cost-effective, and have quicker recovery times. These minimally invasive approaches are characterized by reduced pain, fewer complications, greater cost-effectiveness, and faster recovery times compared to conventional techniques. [2,30-33] Patients are often depressed about SD and secondary venous ulcer recurrence after symptoms have faded, regardless of the type of treatment. While it has been shown that the occurrence of venous ulcers is higher in individuals who

get non-surgical treatment, studies have reported recurrence rates of venous ulcers after surgery ranging from 20% to 40%.[57-62]

DISCUSSION

Stasis dermatitis (SD) is a complex dermatological condition primarily caused by chronic venous insufficiency (CVI), which leads to venous hypertension, skin inflammation, and tissue damage. The condition presents significant challenges in diagnosis and management, often leading to complications such as venous ulcers, infections, and long-term skin changes if left untreated[1].A major issue in SD management is delayed diagnosis due to its subtle early symptoms, including mild erythema, pruritus, and scaling[2]. Many patients initially dismiss these symptoms, and physicians may misdiagnose SD as cellulitis, contact dermatitis, or other inflammatory dermatoses[3]. Studies indicate that up to 30% of suspected cellulitis cases are actually misdiagnosed SD, leading to unnecessary antibiotic prescriptions and increased healthcare costs[4]. Therefore, a thorough history, physical examination, and duplex ultrasound to assess venous insufficiency are crucial for accurate diagnosis[5].The pathophysiology of SD is driven by chronic venous hypertension, which results in endothelial damage, leukocyte activation, and inflammatory cytokine release[6]. This inflammation disrupts the skin barrier, leading to fibrosis, hemosiderin deposition, and, in severe cases, ulcer formation[7]. Histological findings reveal epidermal acanthosis, perivascular lymphocytic infiltration, and hemosiderin-laden macrophages, confirming chronic venous stasis as a key pathological process[8].Compression therapy remains the gold standard for SD management, as it enhances venous return, reduces edema, and alleviates symptoms[9]. However, poor patient adherence remains a significant issue, with discomfort and difficulty in application being common barriers[10]. Recent studies suggest that intermittent pneumatic compression and customized compression garments may improve patient compliance[11]. Additionally, patient education on the importance of long-term compression therapy is essential for treatment success[12].Topical corticosteroids are widely used to manage SD-related inflammation and pruritus[13]. However, prolonged use can cause skin atrophy, necessitating alternative therapies such as calcineurin inhibitors and barrier-repair moisturizers[14]. Emerging evidence supports the use of flavonoid-based treatments, such as micronized purified flavonoid fraction (MPFF), which have shown efficacy in reducing inflammation and improving venous circulation[15]. Systemic agents like pentoxifylline have also demonstrated benefits in microcirculatory function, potentially improving SD outcomes[16].For patients with severe CVI or non-healing venous ulcers, surgical interventions such as endovenous laser therapy (EVLT), radiofrequency ablation (RFA), and sclerotherapy may be necessary[17]. Studies have reported that minimally invasive procedures can significantly reduce venous reflux and improve skin changes associated with SD[18]. Despite their effectiveness, these procedures remain underutilized due to cost and accessibility limitations[19].Future research should focus on identifying biomarkers for early SD detection and developing novel therapies that address both the inflammatory and vascular components of the disease[20]. The integration of digital health tools, such as mobile applications and telemedicine, may enhance patient monitoring and improve long-term adherence to treatment plans[21].In conclusion, SD requires a multidisciplinary approach that includes early diagnosis, patient education, compression therapy, and, in severe cases, surgical intervention. Enhancing physician awareness, improving treatment adherence, and exploring novel therapeutic options are essential for better patient outcomes and reduced healthcare burdens[22].

CONCLUSIONS

Recognition of stasis dermatitis (SD) remains a challenge in healthcare settings, primarily due to a general lack of familiarity and behavioral outlook. [1, 40-44] Addressing this gap requires additional education and training of general and family practitioners. Consequently, during initial visits to family physicians, conditions like SD might not be acknowledged or maybe downplayed upon diagnosis. Timely identification and treatment of SD are crucial in averting complications, including venous leg ulcers (VLUs). Familiarity with SD becomes especially pertinent amid the expanding geriatric demographic.[1,2]Symptoms associated with stasis dermatitis (SD) pose a considerable burden and adversely affect the quality of life for both patients and their physicians. [1, 47-56] Furthermore, SD places a substantial financial strain on the healthcare system. [55,56] This is often exacerbated by misdiagnoses, ulcer development, infections, the necessity for wound care, and complications such as contact dermatitis resulting from excessive use of over-the-counter medications. [1,2, 55,56] Patients frequently exert significant personal efforts, including lifestyle modifications, to alleviate symptoms, even marginally.[1,2] Continuous patient education on the chronic nature of the disease, potential complications, and

the importance of adherence to the treatment plan is vital. Regular follow-up appointments are necessary to monitor progress and adjust treatment as needed.

Authors Contribution Statement:

All authors have made a substantial, direct, and intellectual contribution to the work and approved it for publication. Dr John Abraham contributed to the conceptualization and supervision. Dr Sharon Joseph and Dr Clement Prakash handled dermatological and surgical perspectives. Dr Ambika Akhoury, Dr Anieta Merin Jacob, Dr. Romate John, Dr Ganesh V and Dr Ronnie George played a key role in reviewing and editing the manuscript.

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Aim

The primary aim of this review is to provide an exhaustive analysis of SD, highlighting the limitations of previous studies and offering updated insights into its diagnosis and management. By identifying gaps in existing literature, this review intends to propose an integrated approach for early diagnosis and effective treatment, particularly in family practice settings.

Objectives

1. To evaluate the epidemiology of SD, emphasizing its prevalence across different demographics and geographical locations.
2. To analyze the pathophysiology of SD, including the role of venous hypertension and inflammation in disease progression.
3. To discuss clinical manifestations, including signs and symptoms, and their impact on patients' quality of life.
4. To explore diagnostic challenges and propose strategies for early and accurate identification.
5. To review and compare current treatment modalities, including medical and surgical interventions.
6. To propose future directions for research and clinical practice, improving patient outcomes and reducing healthcare burdens.

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65. Figure 3: Nardin Awad,Stasis Dermatitis: Pathophysiology, Current Treatment Paradigms, and the Use of the Flavonoid Diosmin J Clin Aesthet Dermatol. 2024;17(1):15–23

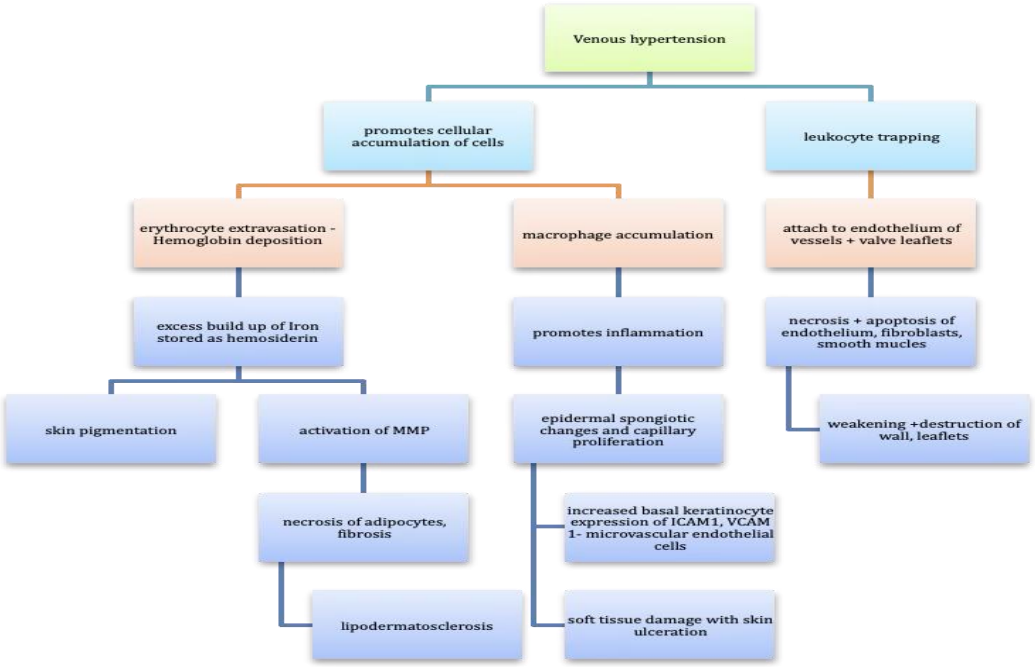

Chart 1: Pathogenesis of Stasis dermatitis

Attribute	Absent = 0	Mild = 1	Moderate = 2	Severe = 3
Pain	None	Occasional, not restricting daily activity	Daily, interfering but not preventing daily activity	Daily, limits most daily activity
Varicose veins	None	Few, isolated branch varices, or clusters, includes ankle flare	Confined to calf or thigh	Involves calf and thigh
Venous edema	None	Limited to foot and ankle	Extends above the ankle but below knee	Extends to knee and above
Skin pigmentation	None or focal	Limited to perimalleolar	Diffuse, over lower third of calf	Wider distribution above lower third of calf
Inflammation	None	Mild cellulitis, ulcer margin limited to perimalleolar	Diffuse over lower third of calf	Wider distribution above lower third of calf
Induration	None	Limited to perimalleolar	Diffuse over lower third of calf	Wider distribution above lower third of calf
Ulcer number	0	1	2	≥3
Ulcer duration	NA	<3 mon	>3 mon but <1 yr	Not healed >1 yr
Ulcer size	NA	Diameter <2 cm	Diameter 2–6 cm	Diameter >6 cm
Compressive therapy	Not used	Intermittent	Most days	Full compliance

An aggregate score for the limb is calculated by adding the individual component scores. The range of the total score is 0 to 30.
NA, not applicable.

Chart 2: Revised Venous Clinical Severity Score (VCSS)/ Clinical Scenarios.

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a, c- venous eczema with overlying erosions and oozing noted over peri malleolar region (mild grade in VCSS)

b- healed atrophic scar noted above right medial malleoulus with surrounding pigmentation over ankle joint. (mild grade in VCSS)

d- ill defined pigmentation with few healed ulcers leaving behind hyperpigmentation, xerosis, mild edema noted over bilateral lower legs(mild grade in VCSS).

e- Single non healing ulcer noted over anterior aspect of right lower leg with surrounding pigmentary changes and edema. (moderate grade to severe in VCSS)

f- erythematous scaly eczematous plaques, with overlying erosions overlying the ankle and lower shin. (mild grade in VCSS)

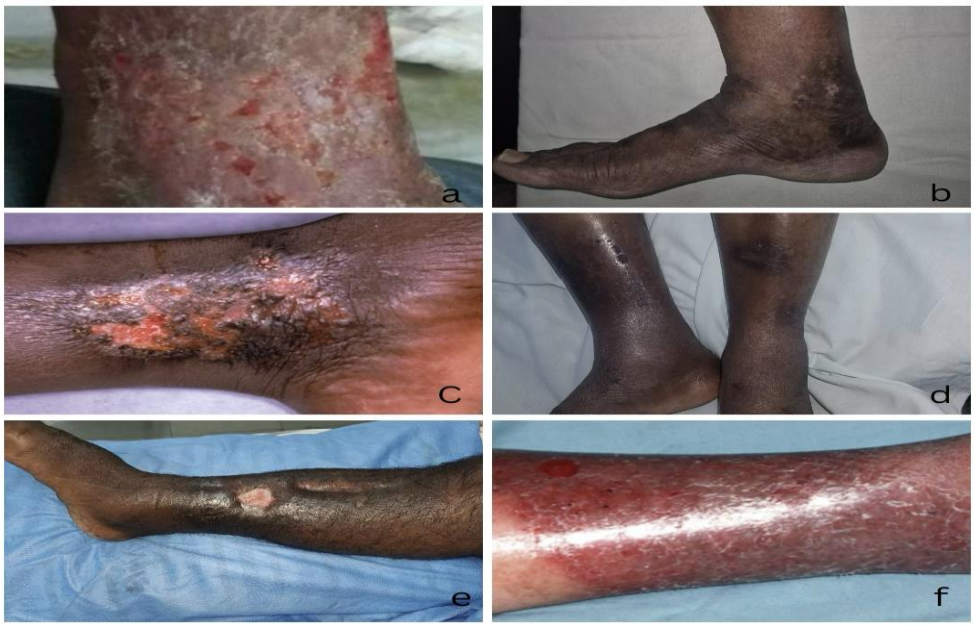

Chart 3 : Table of Treatment Options in Stasis Dermatitis

CEAP Classification	**Management Type**	**Details**
CEAP C1-2 – Symptomatic Varicosities	**Patient Management**	- Lifestyle changes - Compression therapy
	Pharmacologic Management	- Oral flavonoids (MPFF)
	Procedural Management	- Sclerotherapy
CEAP C3 – Edema	**Patient Management**	- As in C1-2 + leg elevation - +/- increased compression grade
	Pharmacologic Management	- Oral flavonoids (MPFF)
CEAP C4 – Cutaneous Change	**Conservative Management**	- As in C3 + emollients
	Pharmacologic Management	- Oral flavonoids (MPFF) - For active inflammation: > 1st Mid-to high-potency TCS > 2nd Other anti-inflammatory
	Procedural Management	- Endovenous ablation
CEAP C5/6 – Cutaneous Change + Ulcer	**Patient Management**	- As in C4 + wound care (if active ulcer)
	Pharmacologic Management	- Oral flavonoids (MPFF) - Topical anti-inflammatories as in C4
	Procedural Management	- Endovenous ablation