

Stasis Dermatitis with Chronic Eczematous changes in a patient with Heart Failure, Chronic Kidney Disease, and Diabetes Mellitus: A Case Report

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Case Report

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Abstract

Background

Stasis dermatitis is a chronic inflammatory disorder of the lower limbs, commonly associated with edema and venous hypertension. Systemic conditions such as heart failure and chronic kidney disease (CKD) may contribute to or aggravate lower-limb edema, xerosis, pruritus, and impaired skin barrier function. Diabetes mellitus may further increase the risk of skin breakdown and secondary infection.

Case Presentation:

A 70-year-old man with stage 3 CKD, type 2 diabetes mellitus, and heart failure presented with a 6-month history of bilateral lower-limb blackish discoloration, pruritus, peeling, and edema. Clinical examination demonstrated bilateral lower-limb edema with marked hyperpigmentation, xerosis, scaling, excoriations, and superficial erosions/crusting. The clinical picture was suggestive of chronic eczematous dermatitis with a prominent stasis component. The patient was treated with topical fusidic acid for suspected localized secondary bacterial infection and mometasone furoate for the inflammatory eczematous component.

Conclusion

This case highlights the multifactorial nature of chronic lower-limb dermatitis in patients with heart failure, CKD, and diabetes mellitus. The combination of dependent edema, chronic hyperpigmentation, pruritus, and scaling should prompt consideration of stasis dermatitis, while CKD-associated xerosis and diabetes-related impairment of skin integrity may contribute to disease persistence and complications.

Introduction

Stasis dermatitis is a chronic inflammatory condition of the lower extremities that is usually associated with chronic venous hypertension and edema. It commonly presents with erythema, hyperpigmentation, pruritus, scaling, and eczematous changes and may progress to erosions, ulceration, and secondary infection.[1, 2]

Lower-limb edema may have multiple causes in older patients. In addition to chronic venous insufficiency, systemic conditions such as heart failure and renal disease can contribute to fluid accumulation in the dependent extremities. Persistent edema may worsen tissue inflammation and compromise the cutaneous barrier. CKD is also frequently associated with xerosis and chronic pruritus, while diabetes mellitus may predispose patients to skin infections and impaired wound healing.[3–6]

We report a case of chronic bilateral lower-limb dermatitis in an elderly man with heart failure, CKD stage 3, and type 2 diabetes mellitus, in whom the clinical findings were suggestive of stasis dermatitis with

superimposed chronic eczematous changes.

Case Presentation

A 70-year-old man with known **stage 3 chronic kidney disease, type 2 diabetes mellitus, and heart failure** presented with a 6-month history of bilateral lower-limb skin changes. The patient reported progressive blackish discoloration, itching, and peeling of the skin, associated with bilateral lower-limb edema.

On clinical examination, both lower limbs showed diffuse dark/blackish hyperpigmentation with prominent xerosis and scaling. Multiple areas of excoriation and superficial erosions with crusting were present. Bilateral lower-limb edema was also noted. The chronicity, bilateral distribution, dependent edema, pigmentation, scaling, and pruritus were clinically suggestive of a stasis-related eczematous dermatitis.

The patient's underlying heart failure was considered a relevant contributor to the lower-limb edema. CKD-associated xerosis and pruritus, together with diabetes-associated impairment of skin integrity, were considered additional factors contributing to the chronicity of the skin disease.

The patient received topical **fusidic acid** for clinically suspected localized secondary bacterial infection and **mometasone furoate** for the inflammatory eczematous component. Supportive skin care with attention to moisturization and restoration of the skin barrier was also considered important.

Differential Diagnosis

The main differential diagnoses considered were:

1. Stasis dermatitis secondary to chronic lower-limb edema/venous hypertension.
2. Xerotic/asteatotic eczema, particularly in the setting of CKD-associated xerosis.
3. CKD-associated pruritus with secondary excoriations.
4. Irritant or allergic contact dermatitis.
5. Diabetic cutaneous manifestations with impaired skin barrier and wound healing.
6. Secondary bacterial infection involving eczematous skin.

The combination of bilateral edema, chronic hyperpigmentation, pruritus, scaling, and eczematous changes favored a significant stasis dermatitis component.[1, 2]

Discussion

The present case represents chronic bilateral lower-limb eczematous dermatitis occurring in a 70-year-old man with multiple systemic comorbidities, including stage 3 CKD, type 2 diabetes mellitus, and heart failure. The findings of bilateral lower-limb edema, marked hyperpigmentation, xerosis, scaling, pruritus, excoriations, and superficial erosions suggest a multifactorial process with a significant stasis

dermatitis component, superimposed on xerotic and inflammatory skin changes associated with CKD and diabetes.

Stasis dermatitis is a chronic inflammatory disorder of the lower extremities most commonly associated with chronic venous insufficiency and venous hypertension. It is particularly common in older adults and typically presents with lower-limb edema, pruritus, erythema or hyperpigmentation, scaling, and eczematous changes. Chronic venous hypertension produces inflammatory changes within the skin and may ultimately result in pigmentation, fibrosis, ulceration, and recurrent secondary infection.[1, 2] The bilateral distribution, edema, chronicity, pigmentation, and scaling observed in this patient are therefore clinically compatible with stasis dermatitis.

The history of heart failure is particularly relevant to the patient's lower-limb edema. Heart failure can result in systemic fluid retention and dependent peripheral edema. Although heart failure-related edema is not synonymous with chronic venous insufficiency, persistent edema may aggravate venous stasis and impair normal skin homeostasis. Thus, in this patient, the lower-limb edema may have a mixed systemic and venous contribution, with heart failure potentially exacerbating an underlying stasis process.

The marked hyperpigmentation further supports a chronic stasis-related process. In stasis dermatitis, venous hypertension and microvascular dysfunction promote extravasation of erythrocytes and deposition of hemosiderin within the skin, contributing to characteristic brown or dark discoloration. The inflammatory response associated with venous hypertension also contributes to pruritus, scaling, and eczematous inflammation.[1, 2]

CKD may have further contributed to the cutaneous manifestations. Xerosis and pruritus are common dermatological manifestations of CKD, and patients may develop dryness, scaling, pigmentary abnormalities, and persistent pruritus.[3, 4] Xerotic skin has impaired barrier function and is more susceptible to excoriation and secondary inflammation. In this case, CKD-associated xerosis and pruritus may therefore have amplified the inflammatory changes caused by edema and stasis.

Diabetes mellitus represents another important modifying factor. Diabetes is associated with xerosis, pruritus, recurrent cutaneous infections, microvascular abnormalities, and impaired wound healing.[5, 6] These abnormalities may increase susceptibility to superficial erosions and delay restoration of the skin barrier. Diabetes therefore may have contributed to persistence of the erosions and increased the risk of secondary infection, although diabetes alone does not adequately explain the characteristic bilateral edema and pigmentation.

The superficial erosions, crusting, and excoriations also raise the possibility of secondary bacterial infection. However, these findings should be interpreted cautiously because crusting, weeping, and excoriation can occur during an active eczematous flare without clinically significant bacterial infection. Current antimicrobial-prescribing guidance emphasizes that eczema may be colonized with bacteria without being clinically infected and that antibiotics should not routinely be used for uncomplicated

eczema flares.[7] In this patient, topical **fusidic acid** may be appropriate when there are clinical features of localized bacterial infection; however, prolonged or repeated use should be avoided because of the risk of antimicrobial resistance.[7, 8]

The use of **mometasone furoate** addresses the inflammatory component of the dermatitis. Control of inflammation and restoration of the epidermal barrier are important in stasis dermatitis and other eczematous disorders. Regular use of emollients is particularly important in patients with CKD-associated xerosis because improvement of skin hydration and barrier function may reduce scaling, fissuring, and pruritus.[1, 3]

Management of the underlying edema is equally important. In confirmed chronic venous insufficiency, compression therapy and leg elevation are important components of stasis dermatitis management because they reduce venous hypertension and edema.[1, 2] However, in a patient with **heart failure, CKD, and diabetes**, the cause and severity of edema should first be assessed and optimized. Evaluation for chronic venous insufficiency, including venous duplex ultrasonography when clinically indicated, may help establish the contribution of venous reflux.[2] Assessment of arterial circulation is also important before substantial compression therapy is initiated, particularly in an older patient with diabetes and possible peripheral vascular disease.

The differential diagnosis includes xerotic/asteatotic eczema, CKD-associated pruritus with secondary excoriations, allergic or irritant contact dermatitis, diabetic skin disease, and secondary bacterial infection. However, the combination of **bilateral dependent edema, chronic dark hyperpigmentation, pruritus, scaling, and eczematous changes**, together with advanced age and heart failure, makes a stasis-related component particularly plausible.[1, 2]

An important limitation of this case is the absence of objective vascular investigations such as venous duplex ultrasonography and ankle-brachial pressure assessment, as well as the absence of microbiological culture or histopathological examination. Therefore, stasis dermatitis should be considered a clinical working diagnosis/major contributing component rather than a definitively confirmed diagnosis of chronic venous insufficiency.

Overall, this case highlights the interaction between systemic fluid retention associated with heart failure, possible chronic venous hypertension, CKD-associated xerosis and pruritus, and diabetes-related impairment of skin integrity. Addressing the underlying edema and circulatory abnormalities, together with appropriate anti-inflammatory and skin-barrier therapy, is important for achieving sustained improvement.

Treatment

The patient was managed with topical **mometasone furoate** for the inflammatory eczematous component and **fusidic acid** for clinically suspected localized secondary bacterial infection. Regular

emollient therapy and appropriate skin-care measures were advised to improve xerosis and restore the cutaneous barrier.

Given the presence of heart failure and bilateral edema, management of the underlying systemic fluid status was considered an important component of care. Evaluation for chronic venous insufficiency and assessment of arterial circulation should be considered where clinically appropriate before compression therapy.

Outcome

The patient's clinical response should be documented according to follow-up findings, including reduction in pruritus, scaling, erythema/inflammation, erosions, and edema. Long-term management should focus on control of the underlying edema, optimization of heart failure and other systemic comorbidities, regular emollient use, and avoidance of unnecessary prolonged topical antibiotic therapy.

Conclusion

This case demonstrates stasis dermatitis with chronic eczematous changes in a patient with heart failure, CKD stage 3, and type 2 diabetes mellitus. The combination of bilateral lower-limb edema, chronic hyperpigmentation, pruritus, scaling, and xerosis suggests a multifactorial dermatological process in which systemic edema and probable venous stasis play important roles. Recognition and management of the underlying edema, together with appropriate anti-inflammatory treatment and skin-barrier care, are essential to reduce recurrence and complications.

Learning Points

- Bilateral lower-limb edema with chronic hyperpigmentation, pruritus, and scaling should raise suspicion for stasis dermatitis.
- Heart failure-related fluid retention may aggravate lower-limb edema and contribute to worsening stasis-related skin inflammation.
- CKD may contribute to xerosis and chronic pruritus, further compromising the skin barrier.
- Diabetes may increase susceptibility to skin breakdown, infection, and delayed healing.
- Erosions and crusting do not necessarily indicate bacterial infection; antibiotics should be reserved for clinically suspected infection.
- Fusidic acid should generally be used only when clinically indicated and not prolonged unnecessarily because of antimicrobial resistance.
- Before initiating significant compression therapy, particularly in patients with diabetes and systemic disease, vascular status and the clinical stability of heart failure should be considered.

Declarations

Written informed consent for publication of clinical details was obtained from the patient.

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Figures



Figure 1

Figure 1- Stasis Dermatitis a type of Eczema due to Venous insufficiency cleaning with Hydrogen Peroxide and Betadine.



Figure 2

Figure 1- Stasis Dermatitis a type of Eczema due to Venous insufficiency after cleaning with Hydrogen Peroxide and Betadine.



Figure 3

Figure 1- Stasis Dermatitis a type of Eczema due to Venous insufficiency application of **fusidic acid and mometasone furoate**



Figure 4

Figure 1- Stasis Dermatitis a type of Eczema due to Venous insufficiency antibiotic and anti inflammatory cream applied.



Figure 5

Figure 1- Stasis Dermatitis a type of Eczema due to Venous insufficiency Dressing done.