Chronic Tissue Injuries, Friction Skin Injuries, and Senile Gluteal Dermatosis: A Unifying Pathology – A Seven-Patient Case Series and Multi-Aspect Discussion

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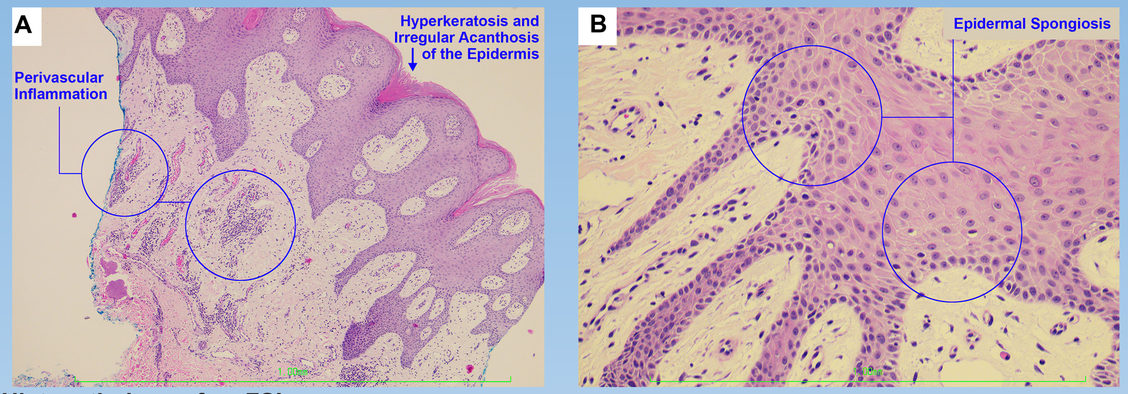
References

Background

Friction skin injuries are well-recognized in the wound care community; however, their pathogenesis remains largely unknown, and no comprehensive histopathologic evaluation of these lesions has been conducted. Over time, various terminologies, such as “friction skin injury” (FSI) and “chronic tissue injury” (CTI), have been proposed to describe these conditions. Dermatologists have studied skin friction dermatoses and have used the terms “frictional dermatosis” and “senile gluteal dermatosis” (SGD) in reference to frictional gluteal skin injuries. In this case series, we aim to determine whether the lesions described by dermatologists and wound care practitioners represent identical pathology and to identify diagnostic and treatment modalities.

Histopathology



Histopathology of an FSI:
a) 4x magnification; H&E stain: shows irregular acanthosis and hyperkeratosis,
b) 20x magnification; H&E stain: mild spongiosis

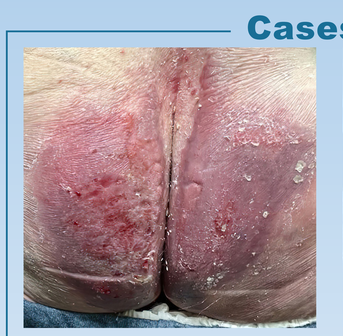
Clinical Characteristics of FSIs

1. Unilateral or bilateral hyperpigmented plaques in the triangle between the coccyx and ischial bones (“sitter’s sign”), or on the posterior legs.
2. Hyperkeratosis.
3. Lichenification.
4. Horizontal ridges.
5. Variable skin injuries from superficial erosions to full thickness ulcerations.
6. Typically painless, though a third of patients may complain of pruritus.

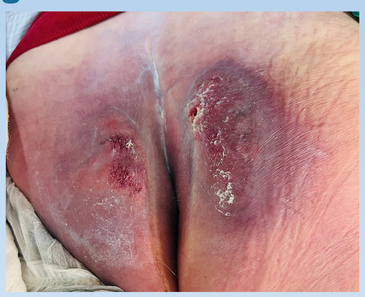
Histopathological Characteristics of FSIs

1. Psoriasiform epidermal hyperplasia.
2. Vascular dilatation in the papillary dermis.
3. Lymphohistiocytic infiltration.
4. Papillary dermal edema.
5. Small-vessel dilatation/proliferation extending down to the reticular dermis.

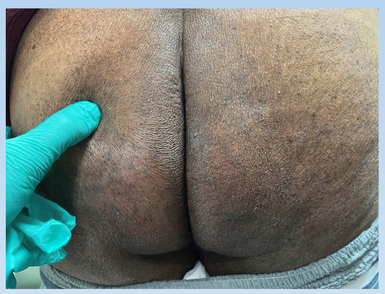
Cases



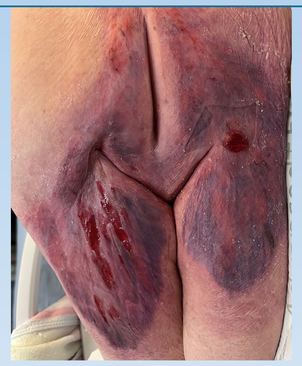
Case 1. 89 yo male spending over 8 hours/day plus all sleep hours in recliner. Bilateral gluteal hyperpigmentation with erosions.



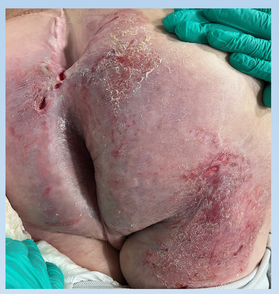
Case 2. 90 yo male spending over 8 hours/day in wheelchair. Bilateral gluteal hyperpigmentation with erosions.



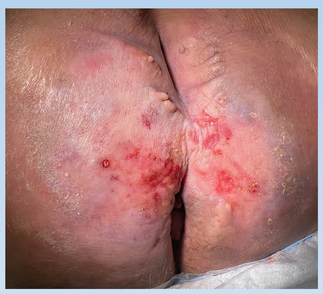
Case 3. 76 yo male spending over 12 hours/day in chair. Midcentral gluteal hypopigmentation, horizontal ridges, and hyperpigmentation of the gluteal folds.



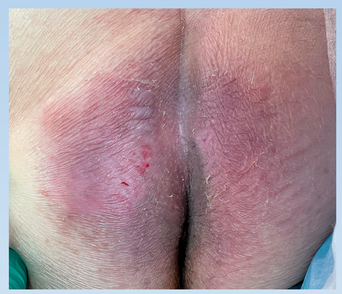
Case 4. 69 yo male with total bedridden status. Gluteal and posterior leg discoloration with partial and full thickness ulcerations.



5. 77 yo male spending over 8 hours/day in wheelchair. Gluteal folds and posterior thigh with purplish discoloration, erosive dermatitis with ridging and lichenification.



Case 6. 85 yo male spending over 8 hours/day in chair. Gluteal purplish discoloration and superficial erosions. ***NOTE: Punch biopsy site left buttock.***



Case 7. 78 yo male spending 4-8 hours/day in chair. Gluteal purplish discoloration and superficial crusted erosions.

Findings

The histopathology results for the 7 patients were as follows: psoriasiform epidermal hyperplasia (7/7: complete in cases 5, 6, 7 and incomplete in cases 1, 2, 3, 4); vascular dilatation in the papillary dermis (4/7: cases 1,2, 3,6); lymphohistiocytic infiltration (2/7: cases 2,7 and lymphoplasmacytic in cases 3,4,5); papillary dermal edema (4/7: cases 1,3,4,7); small-vessel dilatation/proliferation extending down to the reticular dermis (5/7: cases 1,2,3,5,6). In addition, chronic spongiotic dermatitis was diagnosed in 6/7 cases, and one case (3) was thought to be due to resolving inflammation of unknown origin.

Treatment

The saving grace of many skin friction lesions is that many patients are long-term nonprogressors. MASD, including incontinence-associated dermatitis, is often a compounding factor in immobile elderly patients. Since moisture increases friction, moisture control is of paramount importance. Emollients are often recommended as they keep skin hydrated and prevent drying. Topical steroids, keratolytic agents (2% salicylic acid ointment or 10% urea cream), or topical retinoid acid have been used with variable success.We use barrier ointments (e.g., Vaseline-based) in patients with incontinence to enhance skin barrier function and decrease friction at the same time. When inflammatory changes are present, manifesting as erythema often with superimposed erosions, a topical steroid can be helpful. Turning and repositioning may not only treat FSIs but also prevent pressure injuries in this population. Support surfaces facilitating microclimate (e.g., air cushions) are very helpful. In bedridden patients, it is important to keep the head of the bed elevated at 30 degrees and to keep the foot part of the bed elevated as well to decrease sliding down the bed.

Conclusions

Our histopathology findings support the hypothesis that FSIs, CTIs, and SGD represent a single clinical-pathological entity. Biopsies consistently showed features of chronic spongiotic dermatitis with vascular proliferation and perivascular inflammation. The diagnosis is primarily clinical as the histopathologic features are nonspecific. Management should emphasize friction and moisture reduction, patient repositioning, and pressure injury prevention strategies.