Dermatology or Wound Care?

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Every effort has been made to ensure the accuracy of the data presented here. Clinicians may care to check specific details such as drug doses and contraindications, etc., in standard sources prior to clinical application.

OBJECTIVES

- Identify the role of the wound clinician as it relates to dermatologic concerns in the wound patient
- Distinguish chronic versus acute dermatologic manifestations in the chronic wound patient
- Distinguish between wound related versus dermatologic manifestation of systemic disease
- Treat dermatologic issues based on evidence based medicine when required
- Recognize common and uncommon processes
The wound clinician in many instances serves as the interim dermatologic clinician, serving as a "bridge" between Primary Care and Dermatology. They may be the first fully examine the integumentary system, seen the more frequently compared to other clinicians, have more time spent at visit with patient, have a better rapport with patient and patient’s family, and are the closest discipline to Dermatology.

Lipodermatosclerosis
- A consequence of chronic venous hypertension
- Histological changes: change in venous wall, degradation of ECM, decreased type I collagen, decreased type III collagen
- A form of fibrosing panniculitis (inflammation of subcutaneous fat)
- Skin overlying panniculitis with heavily pigmented skin
- Fibrosis can be severe enough to cause a condition known as Champagne Bottle
- Increased risk of cellulitis (Strep/staph), venous ulceration

Atrophie Blanche
- Smooth ivory-white plaque surrounded by hyperpigmented border with telangectasia
- Strongly associated with livedoid vasculopathy (a thrombotic, non-inflammatory condition, with clots affecting medium sized arterioles)
- LV can occur by itself or in connection disorders such as SLE and antiphospholipid syndrome
- Atrophie Blanche may occur after an ulcer
Livedoid Vasculopathy

- Strong association with connective tissue and hypercoagulable disorders
  - Antiphospholipid syndrome
  - Systemic Lupus Erythematosus
  - Rheumatoid Arthritis
  - Scleroderma
  - Mixed Connective Tissue Disease
- High-risk patients: Protein C and S deficiency, Factor V Leiden mutation
- Anticardiolipin antibodies or Lupus anticoagulants, Dysproteinemias

Livedoid Vasculopathy

Differential Diagnosis

<table>
<thead>
<tr>
<th>Condition</th>
<th>Diagnosis</th>
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<tbody>
<tr>
<td>Chronic Venous Stasis</td>
<td>Pyoderma/Gangrenousmum</td>
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<tr>
<td>Peripheral Vascular Disease</td>
<td>Buerger’s disease (non-atherosclerotic inflammation)</td>
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<tr>
<td>Polyarteritis Nodosa (necrotizing vasculitis)</td>
<td>Mixed cryoglobulinemia</td>
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Biopsy should be fusiform (tapered ends) and include subcutaneous fat

Serology: CBC, CMP, UA, ESR, RA, cyclic citrullinated peptide antibodies
C3, C4, CH50, ANA, Cryoglobulins

Goal is to exclude connective tissue or risk of hypercoagulable disorder

- Referral: PCP, Heme-Onc, Rheumatology
**Livedoid Vasculopathy**

**Treatment**
- **Wound Care** - control for infection, maximizing the wound environment, decrease edema
- **Aspirin, anticoagulation, Trental** (if ulceration present or non contraindicated)

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**Case #1**

21 y/o female s/p split thickness skin graft of the right leg 17 days ago presents for follow up. She is currently in the outpatient PT program for energy based modalities 3 times per week. The skin graft has excellent take, however she asks you about a rash near her donor site. She reports the rash appeared 10 days ago. She reports that initially one large lesion appeared with subsequent smaller lesions spreading across the chest, back, and bilateral upper thighs 3 days later. She denies seeking treatment as she knew her appointment at your wound center was coming up.

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**History and Physical**

**ROS**
- Denies medication changes, no new lotions/saops or fabric softener/laundry detergent
- No fevers/chills, +Flu Vaccination 14 days ago

**T 98.8 BP 118/68 P 65 R 18 100% RA**

*Anterior Chest* with 4 cm ovoid pink macule with collaret scale. Multiple small pink macules with collaret noted on anterior chest, back and upper thighs bilaterally symmetric. Examination of the back reveals a Christmas tree pattern.

Exit near the donor site, multiple small pink macules
Pityriasis Rosea

- Acute and self-limiting, possible viral etiology
- Inflammatory oval papulosquamous lesions of the trunk and proximal extremities
- Sometimes preceded by a prodrome
- Not shown to be associated with bacterial or fungal organisms

Most Common in children and young adults
- Women > Men
- Prodrome of headache, fever, malaise, pharyngitis may occur
- Typically asymptomatic, however pruritus common
- Initial “herald/mother patch” on the chest, neck, or back
- Days to 1-2 weeks later, multiple oval lesions similar to herald patch appear

The long axis of subsequent lesions tend to orient along the Langer’s lines of the skin
- Orientation causes “Christmas tree” pattern
- Eruption spreads centrifugally over a few days
- Erythema subsides, in addition to the eruption fading and desquamation ending
- Time for full resolution 8-12 weeks
- Sequela of post-inflammatory hyperpigmentation in darker skinned individuals

Not shown to be associated with bacterial or fungal organisms

Sequela of post-inflammatory hyperpigmentation in darker skinned individuals

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Diagnosis

- History and Physical
  - Absence of symptoms other than pruritis with presence of herald patch

- Differential Diagnosis
  - Secondary syphilis (palm/soles, no herald, + chancre)
  - Guttate psoriasis (coarser scale, no herald, past streptococcal infection)
  - Tinea corporis (Positive KOH)
  - Tinea versicolor (absent erythema, fine scale without collarette, +KOH)
  - Nummular eczema (annular lesions, serous exudate may be seen, extremely pruritic)

Diagnosis/Treatment

- Additional Differential Dx to consider
  - Lyme Disease, HIV seroconversion illness, Drug eruption (omeprazole, naproxen, captopril, terbinafine), Bone marrow transplantation

- Treatment
  - Symptomatic: Medium potency topical corticosteroids (eg., Triamcinolone 0.1%, Fluocinonide 0.05%)
  - Limit use to 2-3 weeks to avoid skin atrophy
  - Topical antipruritic lotions that contain pramoxine or menthol
  - Oral antihistamines

Summary

- Possible viral etiology
- Self-limited
- No laboratory, biopsy testing needed, clinically diagnosed
- Must consider differential diagnoses as it relates to your patient base
- Treatment aimed at symptomatic control
- Refer to Primary Care, Dermatology when indicated
Case #2

25 y/o AA male with pms significant for intermittent asthma and Hidradenitis Suppurativa, presents for periodic follow up s/p excision and split thickness skin grafting to bilateral axillae. He reports undergoing physical therapy for increased range of motion, and states he is progressing well with 1 session left. He denies any new outbreaks, however he requests that you examine a rash located on his tattoo. He is unsure of how long it has been there, but fears it will turn into new HS lesions. He reports using otc cortisone for the past 3 days without resolution of rash.

History and Physical

ROS: intermittent fevers, increased wheezing and use of rescue inhaler + dry mouth
Social Hx: no tobacco use, office worker
FHR 36, BP 128/80, P 68
Gen A&O x 3
CVS: S1/S2, RRR, no m/r/g
Pulm: + faint end expiratory wheezes
Integumentary: graft and donor sites healing well, left upper arm tattoo raised, firm and edematous, small papules noted
Ext: no clubbing, cyanosis

Follow Up

- Patient follows up 2 weeks later without
- resolution with mid potency corticosteroid
- equivocal diascopy due to skin pigment
- Biopsy reveals sarcoidal non-caseating granulomas
  - aggregates of epithelioid histiocytes, giant cells, and mature macrophages
Cutaneous Sarcoidosis

- Sarcoidosis = multisystem granulomatous disease (mixed inflammatory cells)
- Non-cavitating is a distinguishing feature
- Usually starts in the lungs and lymph nodes, however extrapulmonary sites such as heart, liver, eye, nervous system, musculoskeletal system, skin
- Unknown etiology
- Risks: African-American, Scandinavian, Puerto Rican, German descent
- Skin manifestations occur in 25% of patients
- May be the initial presentation of systemic sarcoidosis

Differential Diagnosis

Cutaneous Manifestations (common)
- Papular sarcoidosis
- Angiolupoid sarcoidosis
- Nodular sarcoidosis
- Plaque sarcoidosis
- Lupus pernio
- Hypopigmented sarcoidosis
- Atrophic and ulcerative sarcoidosis

Cutaneous Manifestations (Rare)
- Lupus variant
- Lupus erythematosus
- Psoriasiform sarcoidosis
- Erythrodermic sarcoidosis

Cutaneous Sarcoidosis

Skin findings can also provide clues to prognosis, clinical course, and guide therapeutic decision-making leading to prompt diagnosis and treatment.


### Sarcoidosis of Special Sites

<table>
<thead>
<tr>
<th>Site</th>
<th>Description</th>
</tr>
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<tbody>
<tr>
<td>Focal sarcoidosis</td>
<td>Sarcoidosis of the focal site</td>
</tr>
<tr>
<td>Acrofacial sarcoidosis</td>
<td>Sarcoidosis of the face and hands</td>
</tr>
<tr>
<td>Genital sarcoidosis</td>
<td>Sarcoidosis of the genital region</td>
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</tbody>
</table>

Treatment

**Treatment**

- First line: localized/mild. High potency steroid therapy for localized or mild sarcoidosis of only the skin is a high-potency topical steroid applied twice weekly, or intralesional steroid injections every 3 to 4 weeks.
- If no improvement, add systemic corticosteroids 20-40 mg q day for 1-3 months until improvement. Alternating taper by 5-10 mg every 2-4 weeks or prevent relapse with antimalarials (Dermatology).


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Summary

- A simple “dermatitis” can be much more.
- Tailor your differential to your patient population.
- Prompt referral for further work-up, definitive diagnosis, treatment.
- May need to start empiric therapy until seen by Dermatology.

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13/10/2015
Case #3

51 y/o female with pmm significant for HTN, IDDM-2, HLD and morbid obesity presents to the wound center for follow up of a chronic abdominal surgical wound. Since your last evaluation, 4 weeks ago, her abdominal wound is near closure, however she complains of bilateral lower extremity lesions present for the past 2 weeks. She reports developing extremely pruritic papules that later developed into erythematous bullae.

**History and Physical**

**ROS:** + pruritis, new ulcerations, tenderness in peri-lesion region, denies fever/chills, oral lesions, arthralgias, change in appetite, weight loss, hematuria. No history of ulcers in the past.

**MEDS:** HCTZ 25 mg po q a day, Lantus 45 units qhs, simvastatin 40 mg qhs, Garcinia Cambogia

**Allergies:** N/A

**PE:** T: 98.7, BP: 130/80, P: 70, R: 18 100% on RA

**Gen:** A&M x 3, NAD

**CVS:** S1/S2, RRR, No m/r/g. Doppler - Doppler triphasic DP/PT/AT/Peroneal

**Abdomen:** Midline peri-umbilical wound measuring 2.0x1.5cm, base with beefy red granulation, scant yellow exudate, no odor

**Extremities:** Bilateral lower extremities with palpable purpura, erythematous bullae with varying stages of denudation noted below the knees.
Leukocytoclastic Vasculitis

- Patient reports recently starting Garcinia Cambogia to augment weight loss efforts
- CBC, coagulation panel, LFTs, BMP all within normal limits, CRP increased, C3 and C4 slightly decreased, UA
- Biopsy with immunofluorescence; perivascular and interstitial inflammatory infiltrates composed of neutrophils, lymphocytes, eosinophils. No IgA deposition as in HSP

Leukocytoclastic Vasculitis

- Also referred to as Hypersensitivity Vasculitis
- Neutrophilic inflammation of small vessels
- The crops of circular purpura prefer dependent areas of the body - typically found on the legs, thighs, and buttocks in symmetric distribution
- The purpura is non-blanching, indicating extravasation of blood from the inflamed postcapillary venules
- Macule morphology is regular and circular 2/2 RBC exude radially in all directions from the inflamed and damaged venules

Leukocytoclastic Vasculitis

Differential Diagnosis

<table>
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<tr>
<th>Hemochromatosis Purpura</th>
<th>Age ≤ 20, IgA deposition</th>
<th>Superficial dermal vascular pattern</th>
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<tbody>
<tr>
<td>Meningococcemia</td>
<td>Neisseria meningitidis, n/V</td>
<td></td>
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<tr>
<td>Mixed Cryoglobulinemia</td>
<td>HSM, low complement, Chronic Liver Dz (Hep C)</td>
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- Etiology:
  - Medications (Loop/thiazide diuretics, Phenytoin, allopurinol, PCNs, Cephalosporins, sulfonamides)
  - Food additives such as Yellow dyes
  - Connective Tissue Disorders
Leukocytoclastic Vasculitis

Treatment
- Stop offending agent
- Oral corticosteroids 60-80 mg/day tapered over 3-6 weeks (mild)
- Topical corticosteroids and antibiotic ointment (mild)
- Refer to Dermatology if severe/persistent case- use of dapsone 100-150mg/day with immunosuppressive therapy if needed
- Local wound care


Summary
- History and Physical
- Stop offending agent
- Treat while awaiting Dermatology when indicated

Case #4
51 y/o AA female with pmh significant for HTN and venous stasis ulceration of the left medial ankle presents for initial evaluation. She reports the ulcer has been present for the past 6 weeks despite her use of left over calcium alginate in the home environment. She was referred to the wound center by her PCP for further management. She reports a previous history of a venous stasis ulceration in the same site 2 years ago, which healed successfully with the use of an unna boot. She also states that over the past 3 months she has had a very painful nodule on the side of her foot. She denies any preceding incident to the appearance of the nodule, however does report a crush injury to the area a couple of years ago requiring ORIF to the 5th metatarsal.
Case #4

Surgical History - ORIF 5th metatarsal of the right foot
FH - mother, SLE, father, HFN, DA-2
Social History - Never a smoker, no drugs. Lives at home with husband
Occupational history - Factory worker, on the production line with 10-hour shifts and the use of steel toe boots.
Medications: Trental, Norvasc

Physical Examination

Gen: A&O 3, NAD
Ext: Left medial ankle with 4.0x4.0x0.1 cm wound, bed with 75% yellow adherent slough, no odor. Periwound skin intact, no induration, erythema. Varicosities noted, non tender on palpation.
Doppler - Triphasic DP/PT/AT/Peroneal.

Calcinosi Cuts
Calcinosis Cutis

- 5 subtypes: Dystrophic, Metastatic, Iatrogenic, Idiopathic, Calciphylaxis

**Dystrophic Subtype**
- Trauma
- Pseudoxanthoma elasticum
- Subcutaneous fat necrosis of the newborn
- Dermatomyositis
- Pancreatic enzyme panniculitis
- Cysticercosis
- Ehlers-Danlos
- Porphyria Cutanea Tarda
- Cutaneous neoplasms
- Lupus panniculitis

**Calcinosis Cutis**
- Appear as multiple firm, whitish papules, nodules, or subcutaneous nodules or an isolated nodule
- Papules may extrude a white gritty substance
- May be painful or restrict joint mobility

**Treatment**
- Suppression of underlying autoimmune disease—refer to appropriate consultant
- May use agents such as diltiazem, minocycline, colchicine (Autoimmune)
- Surgical removal if symptomatic and discrete lesion—&D, excision, high speed burr

Case #5

65 y/o male immigrant from Saudi Arabia with pmh significant for IDDM-2, PVD, Hyperlipidemia, HTN, CED s/p DKDT 8 years ago on immunosuppression, presents for follow up of a left medial malleolar venous stasis ulcer. Your last encounter was 2 weeks ago, however he has a new issue that he wants to discuss. He reports a dark lesion on his right foot that he noticed 3 months ago. He worries that it will progress to a new ulcer. Upon inspection of the left foot, you notice a violaceous plaque, without scale, no perilesional erythema and no tenderness to palpation.

Physical Exam

[Image of a leg with a lesion]
Kaposi Sarcoma

- Classic — Kaposi sarcoma (KS) is an indolent cutaneous proliferative disease, mainly affecting the lower extremities of elderly men of Mediterranean and Jewish origin.
- Endemic — The endemic form of KS is found in all parts of equatorial Africa, affecting both children and adults, particularly in sub-Saharan Africa. It is not typically associated with immune deficiency. Endemic KS is frequently more aggressive than classic KS, and may be accompanied by dissemination to lymph nodes, bone, and skin.
- Organ transplant-associated — KS may occur after solid organ transplantation, with symptoms usually regressing after decreasing immunosuppression.
- Epidemic or AIDS-related — KS is the most common tumor arising in HIV-infected persons, and is an AIDS-defining illness. It is 20,000 times more common in persons with AIDS than in the general population and over 300 times more common in AIDS than in other immunosuppressed hosts, such as renal transplant recipients.
- Decreasing immunosuppression may help, so refer to Oncology, Dermatology, Transplant.

Ref: Penn I. Kaposi's sarcoma in transplant recipients. Transplantation 1997; 64:669

References
2. Penn I. Kaposi's sarcoma in transplant recipients. Transplantation 1997;64:669