Chicago Dermatological Society

April 2016
Monthly Educational Conference

Program Information
Continuing Medical Education Certification
and
Case Presentations

Wednesday, April 20, 2016
Gleacher Center
Sidney Barsky Lecture

Conference Host:
Division of Dermatology
Stroger Cook County Hospital
Chicago, Illinois

Program

Host: Stroger Hospital of Cook County
Wednesday, April 20, 2016
Gleacher Conference Center
450 N. Cityfront Plaza Dr., Chicago

All meeting activities take place on the 6th Floor of the Gleacher Center.

8:00 a.m.	**Registration & Continental Breakfast with Exhibitors** *6th Floor Lobby, Room 600 & North Foyer*
8:30 a.m. - 10:00 a.m.	**Clinical Rounds** Posters – *Room 600 (available throughout the morning)* Slide viewing – *Room 602 (available throughout the morning)* Possible patient viewing – *Room 604*
9:00 a.m. - 10:00 a.m.	**Resident/Basic Science Lecture** – *Room 621* *RESIDENT LECTURE:* "Pico: Is This Better than Nano?" *Christopher Zachary, MD*
10:00 a.m. - 10:30 a.m.	**Break and Visit with Exhibitors** – *Room 600/North Foyer*
10:30 a.m. - 12:00 p.m.	**Resident Case Presentations & Discussion** *Room 621*
12:00 p.m. - 12:15 p.m.	**MOC Self-Assessment Questions** *Room 621*
12:15 p.m. - 12:45 p.m.	**Box Lunches & visit with exhibitors** *Room 600/North Foyer*
12:45 p.m. - 1:00 p.m.	**CDS Business Meeting** *Room 621*
1:00 p.m. - 2:00 p.m.	**General Session** – *Room 621* *BARSKY LECTURE*: "Analyzing the Science of Devices in Aesthetic Medicine" *Christopher Zachary, MD*
2:00 p.m.	**Meeting adjourns**

Mark the Dates!
Next CDS monthly meetings –
- Wednesday, May 4, 2016 at Rush University, Chicago
- Wednesday, June 8 at Stephens Convention Center, Rosemont

Watch for details on the CDS website: www.ChicagoDerm.org
Save time and money – consider registering online!

Guest Speaker

CHRISTOPHER ZACHARY, MD

Professor and Chair
Department of Dermatology
University of California
Irvine, CA

Delivering the Sidney Barsky Lecture

Dr. Christopher Zachary is a dermatologist who specializes in skin cancer and reconstruction, cosmetic and laser surgery. A professor and chair of the Department of Dermatology at UC Irvine, Dr. Zachary is a frequent speaker at national and international symposia and is often contact by the media (e.g., NY Times, CNN, Today Show) for commentary about dermatologic issues and treatments. He is a past president of the Association of Academic Dermatologic Surgeons. In addition, he has written and edited numerous papers and books.

Dr. Zachary was born in Yorkshire, England. After completing medical school at the Royal Free Hospital, University of London, he trained in Internal Medicine and Dermatology. At the Institute of Dermatology, Guys and St. Thomas' Hospitals, he gained an interest in dermatological surgery. He completed his formal dermatological surgical education at the University of Michigan, Ann Arbor, MI.

Dr. Zachary returned to London to set up the first Mohs and laser skin surgery practice. In 1988, he became director of the University of Minnesota's Cutaneous Surgery and Laser Center. In 1997, he became a clinical professor and co-director of the Dermatologic Surgery and Laser Center at UC San Francisco Medical Center. In 2005, he accepted the position as professor and chair of the Department of Dermatology at the UC Irvine Health School of Medicine.

CONTINUING MEDICAL EDUCATION CREDITS

Chicago Dermatological Society

Presents

"Chicago Dermatological Society Monthly Meeting Series"

April 20, 2016 *Chicago, IL*

Please be sure to complete and return the attendance/CME claim form and the evaluation form before you leave the meeting. The information collected in the evaluation form represents an important part of the CME planning process. A certificate of credit will be mailed to you following the conference. Participants must attend entire session to receive full credit. SynAptiv will retain a record of attendance on file for six years.

JOINT SPONSOR STATEMENT

This educational activity is jointly provided by SynAptiv and the Chicago Dermatological Society.

GOAL / PURPOSE

To broaden the clinical knowledge of dermatologists in a number of areas relating to management of patients and new therapy options.

EDUCATIONAL OBJECTIVES

Upon completion of this series of activities, participants will be able to:

1. Discuss key factors in the diagnosis and treatment for viral diseases and bacterial infections.
2. Describe the manifestation of skin cancers and the efficacy of treatments available to the dermatologist.
3. List the therapeutic options available to the dermatologist for a variety of skin diseases, both medical and surgical, and discuss how new emerging treatments can be successfully incorporated into a dermatology practice.

Continued next page

CREDIT STATEMENTS

This activity has been planned and implemented in accordance with the accreditation requirements and policies of the Accreditation Council for Continuing Medical Education (ACCME) through the joint providership of SynAptiv and Chicago Dermatological Society. SynAptiv is accredited by the ACCME to provide continuing medical education for physicians.

SynAptiv designates this live activity for a maximum of 4.5 *AMA PRA Category 1 Credits*™. Physicians should claim only the credit commensurate with the extent of their participation in the activity.

DISCLAIMER STATEMENTS

The content, views and opinions presented in this educational activity are those of the authors and do not necessarily reflect those of SynAptiv and Chicago Dermatological Society. The authors have disclosed if there is any discussion of published and/or investigational uses of agents that are not indicated by the FDA in their presentations. Before prescribing any medicine, primary references and full prescribing information should be consulted. Any procedures, medications, or other courses of diagnosis or treatment discussed or suggested in this activity should not be used by clinicians without evaluation of their patient's conditions and possible contraindications on dangers in use, review of any applicable manufacturer's product information, and comparison with recommendations of other authorities. The information presented in this activity is not meant to serve as a guideline for patient management.

DISCLOSURE STATEMENTS

SynAptiv insures balance, independence, objectivity, and scientific rigor in all our educational activities. In accordance with this policy, SynAptiv identifies conflicts of interest with its instructors, planners, content managers, and other individuals who are in a position to control the content of an activity.

Our guest speaker, Christopher Zachary MD, has disclosed the following potential conflicts of interest: *Consulting fees* – Sciton, Solta, Zeltiq (scientific advisory board).

All other faculty, planners and/or content managers have no conflicts of interest to disclose nor do they have any vested interests or affiliations.

Fee Information - There is no fee for this educational activity.

Table of Contents

*Protocol to be posted same-day on the CDS website

Key locations: Oral mucosa, lower extremities **CASE 1**

Presented by Christina Kranc MD and Vidya Shivakumar MD

History of Present Illness
A 27-year-old woman presented to the emergency room with shortness of breath, and left knee swelling and pain secondary to a fall. She was admitted for symptomatic anemia and additional workup. Upon further questioning, she noted a two-month history of bleeding gums, epistaxis, rash on the lower extremities, and daily vaginal bleeding. She had no personal or family history of any bleeding disorders. She reported a ten-pound weight loss, which she attributed to a new diet consisting of only grains and broths for the past few months. She dislikes fruit and had recently stopped eating meat. She does not take a multivitamin.

Past Medical History
None

Medications
Herbal supplement

Allergies
NKDA

Social History
No alcohol, tobacco, or illicit drug use

Review of Systems
The patient endorsed generalized weakness, fatigue, and myalgias.

Physical Exam
Upper and lower gingivae: edematous and friable with focal erosions. Normal dentition.
Hard and soft palate: multiple petechiae
Lower legs: multiple non-blanching purpura and petechiae, predominantly perifollicular in distribution, with associated hyperkeratosis. Brittle corkscrew hairs, many completely broken on dermoscopy
Left knee: edematous, large, tender hematoma and surrounding ecchymoses, limited range of motion

Laboratory Data
The following labs were remarkable/abnormal:

Hemoglobin	4.9 g/dL	[11.7-14.9 g/dL]
WBC	3.1 k/μL	[4.4 - 10.6 k/μL]
Platelets	174 k/μL	[161-369 k/μL]
Protein	6.2 g/dL	[6.4-8.3 g/dL]
Albumin	3.6 g/dL	[3.8-5.2 g/dL]
Calcium	7.7 mg/dL	[8.5-10.5 mg/dL]
Iron	11.0 ug/dL	[45-182 ug/dL]
Folate	3.93 ng/mL	[5.90-24.80 ng/mL]
Vitamin B 12	114.00 pg/mL	[180.0-914.0 pg/mL]
Ferritin	46.00 ng/mL	[11.00-307.00 ng/mL]
Prothrombin Time	15.4 sec	[12.1-14.7 sec]
PTT	35.7 sec	[24.2-36.8 sec]

Antiparietal cell antibody screen	Negative
Antigliadin IgA/IgG	Negative
Antiendomysial (TTG) IgA/IgG	Negative

Synovial fluid analysis (left knee arthrocentesis)
Fluid analysis showed 197,000 RBCs, consistent with hemarthrosis. Results otherwise unremarkable.

EGD with multiple tissue biopsies
Minimal, chronic, nonspecific inflammation. Histological examination with immunohistochemistry and urease test from multiple sites was negative for Helicobacter pylori.

Diagnosis
Vitamin C deficiency (adult scurvy) in the setting of multi-nutrient deficiency due to restrictive diet

Treatment and Course
The laboratory findings revealed a normocytic normochromic anemia secondary to decreased iron, folate, and vitamin B12. The elevated prothrombin time was attributed to vitamin K deficiency. A Vitamin C level was sent to the laboratory, but the sample was exposed to sunlight and thus could not be processed. However, vitamin C deficiency was suspected based on clinical features and dietary history. The patient was treated with multiple dietary supplements, including ascorbic acid (500 mg twice daily), cyanocobalamin, calcium-vitamin D, vitamin K, folic acid, and ferrous sulfate. There was a rapid improvement in the patient's skin lesions, coagulation panel, and clinical symptoms following supplementation.

Discussion
Vitamin and mineral deficiencies are common in developing countries due to the limited availability of food containing essential vitamins. However, malnutrition in the U.S. is limited to at-risk patient groups, including those with inappropriate intake, restrictive diets, poor access to health services, and conditions causing malabsorption. Diagnosing a particular micronutrient deficiency can be difficult as most patients are deficient in more than one vitamin or mineral. Risk factors specific to vitamin C deficiency (scurvy) include allergies or intolerance to citrus foods and chronic alcoholism, as alcohol decreases vitamin C absorption. In a large U.S. survey including more than 30,000 patients, 14% males and 10% females were reported to be deficient in vitamin C.

Unlike other animals, humans cannot synthesize ascorbic acid, making it an essential nutrient derived from the diet. It acts as an antioxidant and a cofactor in many enzymatic processes, including collagen synthesis. Abnormal collagen synthesis disrupts blood vessel and hair integrity, and can lead to poor wound healing. Ascorbic acid also plays a role in the biosynthesis of carnitine and neurotransmitters, and modulates the absorption, transport, and storage of iron and folate. Anemia occurs in a majority of patients because of blood loss and concomitant folate and iron deficiency.

A few studies have successfully isolated vitamin C deficiency, rather than other aspects of malnutrition, as the cause of clinical findings seen in scurvy and provided a timeline in which symptoms present. One example is a self-experimentation in 1939 by John Crandon, a surgical resident who consumed a diet devoid of vitamin C for more than six months to prove that vitamin C deficiency alone impairs wound healing.

Early stages of scurvy are characterized by constitutional symptoms such as fatigue, weakness, joint swelling, and myalgias. More specific symptoms can be seen after six to eight weeks of

inadequate vitamin C intake. Follicular hyperkeratosis with subsequent perifollicular hemorrhage is pathognomonic for scurvy. Hemorrhagic skin lesions may become palpable, especially on the legs where hydrostatic pressure is highest. The combination of presumed "palpable purpura" and rheumatologic symptoms may resemble a systemic vasculitis. Other clinical features include broken or "corkscrew" hairs, leg edema and ocular hemorrhage. Oral findings include hyperplastic ulcerative gingivitis, periodontitis and mucosal hemorrhage. Advanced cases can present with bone pain, gastrointestinal symptoms, or heart failure (from myocardial hemorrhage).

The diagnosis of scurvy is made based on clinical features, dietary history, and rapid improvement following supplementation. Laboratory investigations are not necessary for diagnosis, as plasma ascorbic acid levels reflect recent dietary intake rather than actual body storage. Supplementation of vitamin C with 300 to 1000 mg/day for one month is considered sufficient supplementation for treatment. Subjective improvement occurs within 24 hours, and complete recovery becomes apparent within three months.

Clinicians should recommend supplementation for those at risk of vitamin C deficiency and educate patients about vitamin C-rich foods. The recommended daily intake of vitamin C is 75 mg/day and 90 mg/day for women and men, respectively. More than 90% of vitamin C is derived from fruits and vegetables, with citrus fruits, tomatoes, peppers, broccoli, and potatoes being major contributors. The vitamin C content in foods can vary depending on growing conditions, season, transport, shelf time and storage. Ordinary cooking can reduce the vitamin C content by 20 to 40%.

Scurvy patients must be screened for concomitant nutritional deficiencies, particularly vitamin K. There are several vegetables (broccoli, brussels sprouts, cauliflower, and spinach) rich in both vitamin K and C, and patients with anorexia or limited vegetable intake are at risk for simultaneous deficiency. Furthermore, the clinical features of vitamin K deficiency often overlap with those seen in scurvy. Because vitamin K is required in the synthesis of clotting factors, hemorrhage is also a major symptom in vitamin K deficiency. Patients often have an elevated prothrombin time, and in severe cases, an elevated partial prothrombin. Corkscrew hairs and perifollicular keratosis with hemorrhage are signs specific to vitamin C deficiency and can assist in diagnosis. It is important to identify coexisting deficiencies, as complete symptom resolution requires supplementation of all necessary vitamins.

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Key locations: Abdomen, right foot **CASE 2**

Presented by Nicole Joy MD and David Reid MD

History of Present Illness
A 42-year-old man with myelodysplastic syndrome (MDS) with transformation to acute myeloid leukemia (AML) was admitted for induction chemotherapy with clofarabine prior to bone marrow transplant. Dermatology was consulted four days after completion of chemotherapy for evaluation of a dark, circular lesion on the patient's abdomen. The lesion had been present for about three days, was not growing or changing, and was minimally tender. There was no history of trauma at this site. He also reported three days of pain, redness, and swelling of his right foot. Of note, the patient had been admitted multiple times for neutropenic fever in the months preceding this admission, and was partially treated with voriconazole for presumed pulmonary Aspergillosis.

Past Medical History
MDS (2012), AML (2015) s/p induction chemotherapy with cytarabine and idarubicin with relapse, and subsequent treatment with decitabine, orchitis, proctitis, MSSA furunculosis, peripheral neuropathy, presumed pulmonary Aspergillosis (based on a weakly positive Aspergillus antigen and pulmonary nodules noted on CT scan)

Medications
Acyclovir, liposomal amphotericin B, daptomycin, fllgrastim, imipenem-cilastatin, oxymetaz, morphine

Allergies
NKDA

Social History
No history of alcohol, tobacco, or drug abuse
Employed as a landscaper

Review of Systems
Positive for intermittent fever, right foot pain, abdominal pain and distention, constipation, and mild headache. No other systemic symptoms.

Physical Exam
Left upper abdomen: oval, dusky to deeply violaceous purpuric patch with retiform branching at the borders, surrounded by a halo of pallor and bright pink erythema peripherally
Right foot, distal first and second digits, medial dorsal foot: tender, ill-defined, erythematous macules and patches

Laboratory Data
The following labs were remarkable/abnormal:

Hemoglobin	7.8 g/dL	[12.9 – 16.8 g/dL]
Hematocrit	23.0%	[38.1-49%]
WBC	0.1 k/µL	[4.4 – 10.6 k/µL]
Neutrophils	0.0 k/µL	[2.2 – 6.9 k/µL]
Platelets	23 k/µL	[161 - 369 k/µL]
AST	91 U/L	[0 - 40 U/L]
ALT	70 U/L	[5- 35 U/L]

Histopathology
Left upper abdomen, punch biopsy:
Epidermis with early ischemic changes. Underlying capillaries with sludging and intraluminal fibrin. In the deep reticular dermis, there is vessel occlusion by fungal hyphae. The presence of septation and degree of angulation cannot be assessed.
However, the hyphae appear greater than four microns, favoring *Mucor* species. PAS stain highlighting broad fungal hyphae with evidence of fungi passing through vessel wall.

Microbiology
Tissue culture (left upper abdomen): KOH with few non-septate hyphae; bacterial and fungal cultures negative

Multiple blood cultures: negative
Urine fungal and bacterial cultures: negative
Stool culture: negative
Sputum culture: one sample positive for *Candida albicans*; other samples negative
Multiple sputum AFB stains: negative
Urine Histoplasma antigen: negative
Latex Cryptococcal antigen: negative
Bronchoalveolar lavage bacterial, AFB, fungal, Legionella cultures: negative
Serum Galactomannan: negative

Radiology
CT abdomen and pelvis (one day prior to consultation): Compared to prior scans, there is a new, mass-like, ground-glass opacity in the right lower lobe. New, sub-centimeter, nodular density in the left lower lobe, and new, low density hepatic and splenic lesions.

CT head (one day prior to consultation): Unremarkable.

CT chest (one day after consultation): New basilar consolidations, right greater than left. New mid-lung ill-defined consolidations and ground-glass opacities. A few of the right lung consolidations have an unusual configuration, suggestive of infarcted lung. Newly developed hepatic lesion in immediate contact with the right hemidiaphragm and the adjacent lung consolidation. New superior spleen lesion that also contacts the left hemidiaphragm.

CT chest, abdomen, and pelvis (eight days after consultation): Interval worsening of extensive pulmonary opacities compatible with fungal pneumonia. New moderate right and small left pleural effusions. Slight worsening involvement of the liver and spleen, possible trans-diaphragmatic involvement of the hepatic dome as previously described.

Diagnosis
Angioinvasive fungal infection, likely zygomycosis

Treatment and Course
The patient was treated empirically with liposomal amphotericin B, daptomycin, and imipenem. Given severe hypokalemia, amphotericin B was discontinued, and caspofungin and posaconazole were added. Despite aggressive antimicrobial therapy, he remained febrile, and his respiratory status worsened. Repeat imaging showed progression of pulmonary, hepatic, and splenic disease. The patient was eventually transferred to the intensive care unit, where he required mechanical ventilation and pressor support. Repeat peripheral blood flow cytometry showed >30% blasts. The patient eventually died from progressive hematologic malignancy and septic shock.

Discussion

The term zygomycosis refers to infections caused by fungi in the class Zygomycetes, which includes the orders Mucorales and Entomophthorales. Fungi in the Mucorales order generally cause opportunistic infections, while the Entomophthorales tend to infect immunocompetent hosts in tropical areas. Because most reported human cases are caused by fungi in the order Mucorales, the terms mucormycosis and zygomycosis are commonly used interchangeably. These fungi are ubiquitous in nature and can be found in decaying organic matter such as wood, bread, fruits, and vegetables.

Though mucormycosis has been reported in immunocompetent hosts, it is most commonly an opportunistic infection. It is the third most common cause of invasive fungal infections after *Aspergillus* and *Candida,* but it is 10- to 50-fold less frequent than the other two infections. The exact incidence is not known, but one study reported that there are 1.7 cases per million per year in the United States.

Risk factors for infection include hematologic malignancy, stem cell transplant, prolonged and severe neutropenia, poorly controlled diabetes mellitus, iron overload, trauma, prolonged corticosteroid use, and intravenous drug use. Additionally, prior exposure to voriconazole (often used prophylactically) in patients with hematologic malignancy seems to be associated with an increased risk of mucormycosis, though this relationship has not been well established.

One review of 929 cases of mucormycosis reported that 22% of cases were hospitalized patients with hematologic malignancies or who had undergone stem cell transplant. Another smaller study reported that patients with hematologic malignancy made up 44% of cases. Of all hematologic malignancies, acute myelogenous leukemia (AML) portends the highest risk for infection, with incidences ranging between 1-8%.

Clinically, mucormycosis presents in six major forms: rhinocerebral, pulmonary, cutaneous, gastrointestinal, disseminated, and other/uncommon presentations. The most commonly involved sites are the sinuses, lungs, and the skin. Cutaneous mucormycosis can be divided into primary and secondary infection. Primary cutaneous mucormycosis results from direct inoculation into the skin, where secondary infection is due to dissemination into the skin from another location, usually rhinocerebral. Primary cutaneous infection, as seen in our patient, represents 7-15% of all cases of mucormycosis. Common causes of trauma include needle sticks, adhesives, and excoriations. Because of the angioinvasive nature of the fungi, necrosis is commonly seen. Infection can be rapidly progressive and can invade underlying tissues such as muscle, bone, etc. Dissemination from primary cutaneous infection into the bloodstream and other organs can occur. However, spread to the skin from distant sites is very rare.

Diagnosis of mucormycosis can be challenging. Definitive diagnosis usually requires positive cultures and visualization of fungal elements on tissue biopsy. However, tissue cultures are often falsely negative—as often as 50% of the time. The sensitivity of culture has been cited to be as low as 70%. It is thought that the fragile hyphae are easily destroyed during processing. Therefore, if mucormycosis is suspected, tissue should be minced into small pieces instead of homogenized to optimize growth. When cultures are positive, morphological analysis to determine species can be challenging, as many fungi in the Mucorales order share many features. Additionally, blood cultures in infected patients are usually negative. Molecular identification methods, such as PCR, can be very helpful in diagnosis, though these are not always available.

Treatment includes anti-fungal therapy (usually amphotericin B), surgical debridement, and reversing underlying medical comorbidities. Unfortunately, mucormycosis is associated with a

high mortality - up to 96% in patients with disseminated disease. In patients with malignancies, the overall mortality is nearly 70%. The patient presented in this case died despite aggressive therapy. Though we suspect that his abdominal skin lesion was primary cutaneous infection, it is unclear if his pulmonary, hepatic, and splenic lesions (noted on CT) represent dissemination by the same organism or represent a second infection, such as *Aspergillus*.

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CASE 3

Presented by Jackelyn Tanios MD, Warren Piette MD, and David Reid MD

44-year-old woman with chronic kidney disease status post renal transplant in 1996, on chronic immunosuppression, was admitted for a two-week history of fevers, chills, productive cough, and right hand swelling. Dermatology was consulted for a painful, red lesion on her right fifth digit.

UNKNOWN

Key locations: Lower extremities **CASE 4**

Presented by Steven Nwe DO, Warren Piette MD, and Kubinne Kim MD

History of Present Illness
A 42-year-old man presented to the emergency room with a five-day history of progressive left elbow pain and swelling. The patient noted a nodule of similar duration on his left lower extremity. Over the next five days, he developed additional painful nodules on his lower extremities. The patient denied preceding trauma, injection, or history of similar lesions in the affected areas.

The patient had a history of chronic gouty arthritis with one tophus on his left hand, which was never painful. In the emergency room, the patient was found to have orthostatic hypotension with severe left elbow pain. A joint lavage revealed over 500,000 WBCs in the joint aspirate, therefore, the patient was admitted for presumed septic arthritis of the left elbow. Dermatology was consulted for evaluation of his new lower extremity nodules.

Past Medical History
Gouty arthritis, alcohol abuse, hypertension

Medications/Allergies
Stopped allopurinol five months ago

Social History
Drinks one pint of vodka 4-5 times a per day
Smokes 1/2 pack of cigarettes per day for 15 years
No illicit drug use
Unemployed
No household pets

Review of Systems
The patient denied fever, chills, night sweats or weight loss.

Physical Exam
Left elbow: linear incisional scar from orthopedic wash out with sutures in place
Left hand, 3rd digit: PIP joint with soft non-tender nodule consistent with gouty tophus
Right anterior thigh and left anterior leg: tender, firm, skin-colored, subcutaneous nodules with peripheral erythema

Laboratory Data
The following labs were remarkable/abnormal:

WBC	14.9 k/uL	[4.4-10.6 k/uL]
Hgb	12.6 g/dL	[12.9 -16.8 g/dL]
Hct	38.3%	[38.1-49.0%]
MCV	103.2 fL	[81.9 - 97.8 fL]
BMP	No abnormalities	
AST	14 U/L	[0-40 U/L]
Uric Acid	10.6 mg/dL	[3.0-7.0 mg/dL]
Synovial WBCs	508,000	
Gram stain	Negative	
Synovial fluid	Monosodium urate crystal	

Histopathology
Right anterior thigh (formalin-fixed H&E):
Acute mixed lobular and septal panniculitis with suppuration, associated with amorphous eosinophilic material in the subcutis. Special stains for fungus, bacteria and acid fast bacilli are negative.

Diagnosis
Gouty panniculitis

Treatment and Course
The patient was treated empirically for acute gouty arthritis with secondary infection with IV ceftriaxone, oral prednisone 30 mg daily, and colchicine 0.6 mg twice daily. On day three of admission, his leg nodules were no longer symptomatic. He was discharged on a two-week course of IV antibiotics, prednisone taper, and colchicine. Upon follow up, his lower leg lesions were still present, though asymptomatic. The patient was tapered off of prednisone and started on allopurinol 300 mg daily for therapy of chronic gout.

Discussion
While gouty tophi are relatively common, gouty panniculitis is a rare cutaneous manifestation that may precede or follow the onset of arthritic symptoms. Since Niemi et al. first described gouty panniculitis in 1977, there have been only a few case reports.

Gouty panniculitis is characterized clinically by indurated subcutaneous nodules with a predilection for the lower extremities, with or without pain. Histology shows a mainly lobular panniculitis accompanied by deposits of monosodium urate crystals in the subcutaneous tissue. Confirmation of negatively birefringent crystals in biopsy specimens is dependent on alcohol-based fixation. In formalin-fixed material, the crystals dissolve leaving behind characteristic, amorphous pink areas with needle-like clefts, which correspond to the sites of crystal deposition surrounded by inflammatory changes. In our case, alcohol fixation was not obtained, but monosodium urate crystals were visualized in the patient's synovial fluid aspirate.

The mechanism of gout-induced panniculitis is not clear. Some have postulated that the lobular subcutaneous tissue reaction could be triggered and perpetuated by arterial blood supply disruption caused by monosodium urate crystals. Microtrauma to the walls of terminal blood vessels, as well as a communication loss between the vessels and the dermis, makes the subcutaneous tissue more vulnerable. Risk factors for this specific presentation of gout are at this time unclear; however, early case reports suggested a link with hypertension, hypertension-related chronic renal insufficiency, elevated serum uric acid levels, and venous stasis.

There is no specific therapy for gouty panniculitis given its rarity. Therapeutic interventions have mirrored those used for both acute gouty arthritis and chronic tophaceous gout, including acute symptomatic management with anti-inflammatories such as NSAIDS, prednisone, and colchicine, followed by chronic uric acid stabilization with xanthine oxidase inhibitor such as allopurinol and febuxostat. Rheumatology literature suggests that relatively high doses of allopurinol (600-1200 mg/day) for two to three years could lead to progressive improvement in the subcutaneous lesions and possibly prevent new lesions. Surgical resection of symptomatic lesions has also been suggested.

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Key locations: Face, chest, back **CASE 5**

Presented by Anand Haryani MD and David Reid MD

History of Present Illness
A 30-year-old G1P1 woman presented with a six-month history of sudden-onset, eruptive, nodulocystic acne on her face, chest, and back. She reported no previous history of acne, and only recently tried over-the-counter acne face washes without improvement. She denied any new dietary or occupational exposures. During review of systems, patient described a 20-pound weight gain over the previous year along with deepening of her voice, decreased sexual desire, increase facial hair growth, and hair loss on her scalp. Incidentally, she was recently seen in the emergency department for persistent lower abdominal pain, which was attributed to her history of uterine fibroids. At that time, she also reported a one-year history of oligomenorrhea and was referred to gynecology for further evaluation.

Past Medical History
Uterine fibroids

Medications/Allergies
None/NKDA

Social History
No tobacco use. No alcohol use. No illicit drug use.

Physical Exam

Skin:	Forehead, cheeks, chin, anterior neck, upper back, and chest with numerous erythematous papules, pustules, and tender subcutaneous nodules
Abdomen:	Non-tender, non-distended, no palpable masses or organomegaly
Pelvic Exam:	Within normal limits per gynecology

Laboratory Data
The following labs were remarkable/abnormal:

Pregnancy Test (Urine)	Negative	
Prolactin	6.08 ng/ml	[3.85 – 8.78 ng/ml]
Follicle-stimulating hormone	5.29 mIU/ml	[4.54-22.5 mIU/ml]
Luteinizing hormone	2.19 mIU/ml	[2.12-10.89 mIU/ml]
Testosterone (serum total)	356.2 ng/dl	[10.0-75.0 ng/dl]
Dehydroepiandrosterone sulfate (DHEA-S)	2461.30 ng/ml	[180-3910 ng/ml]

Imaging
Transvaginal ultrasound: 4.3 x 3.5 x 4.1 cm lobulated, solid mass with increased vascularity on the left adnexa.

CT pelvis with contrast: Left adnexal solid mass measuring 4.6 x 3.4 cm. No evidence of intra-abdominal metastasis.

Histopathology
Left adnexal mass:
Mixed population of polygonal to ovoid cells with eosinophilic, finely granular, and clear cytoplasm, with a predominance of clear cells. Cells show ample cytoplasm, small round to oval

nuclei, and small nucleoli. Tumor cells are immunoreactive with calretinin and inhibin, and nonreactive with antibody to epithelial marker AE1/AE3.

Diagnosis
Steroid cell tumor, not otherwise specified

Treatment and Course
The patient underwent a successful laparoscopic left salpingo-oophorectomy. Following the procedure, total testosterone normalized to13 ng/dl and DHEA-S decreased to 1700 ng/ml.

For her acne, the patient was started on doxycycline, benzoyl peroxide gel, and clindamycin gel, however she did not receive the doxycycline due to financial reasons. One year after surgical removal of the steroid cell tumor and the initiation of acne treatments as above, she has hypertrophic and ice-pick scarring on the face, back, and chest. There is no evidence of active acneiform lesions at this time.

Discussion
Steroid cells tumors, a subtype of sex cord tumors, are rare growths that occur primarily in adult women and comprise 0.1% of all ovarian tumors. There are three categories of ovarian steroid cell tumors: stromal luteoma, Leydig cell tumor, and steroid cell tumor, not otherwise specified (NOS). Steroid cell tumors, NOS, comprise 60% of all steroid cell tumors and occur in younger, women of reproductive-age (mean age of 43 years) with 23 cases reported in prepubescent girls. The majority of tumors are low-grade, well circumscribed, unilateral, solid, and only slightly larger than the normal ovary. Approximately one-third of steroid cell tumors, NOS are malignant, with extra-ovarian spread of the tumor, and carry a poor prognosis.

Hirsutism and virilization, the most common symptoms, occur in over 50% of patients. In the first three months of tumor growth, patients develop signs and symptoms of androgen excess: oligomenorrhea, regression of female external genitalia, and abnormal hair growth. Amenorrhea, hirsutism, acne, clitoromegaly, male pattern baldness, sterility, and deepening of the voice occur later in the disease process.

Estradiol secretion by steroid cell tumors, NOS occurs in 6-23% of cases. Excess estrogen can result in menorrhagia. Other associations include Cushing's syndrome, which is found in 17% of cases and is due to an increase in cortisol. Hypertension and hypokalemia may develop due to elevated serum pro-renin. Of note, one-quarter of steroid cell tumors, NOS do not produce any hormones.

An adult female patient presenting with severe, sudden-onset acne should be evaluated for an underlying endocrine disorder, particularly an androgen-secreting tumor or hypercortisolism. Androgens, including testosterone, dihydrotestosterone, and the weaker dehydroepiandrosterone sulfate, act on androgen receptors in sebaceous gland epithelial cells (sebocytes, keratinocytes, and dermal fibroblasts) affecting sebocyte proliferation, differentiation, activity, and homeostasis. The greatest effect is seen in areas of skin prone to acne including the face, neck, upper chest, and back. Additionally, androgens promote the inflammatory response, further contributing to acne formation and progression.

Early diagnosis and treatment of androgen excess can prevent irreversible signs of virilization, such as deep voice and short stature. Unfortunately, diagnosis of steroid cell tumors is difficult for several reasons: nonspecific symptoms (abdominal pain, distention, and bloating), non-palpable tumor, and negative imaging studies. Several medications cause similar virilizing findings.

Recognizing these common medications (androgenic steroids, progesterones, oral contraceptives, and dehydroepiandrosterone) can help elucidate a diagnosis.

Women with virilizing signs and symptoms require a bimanual exam, serum testosterone, DHEA-S, and imaging of the adrenals and ovaries. Serum testosterone greater than 200 ng per dL (6.94 nmol per L) is strongly indicative of a virilizing tumor. DHEA-S is elevated in adrenal pathology and is mostly within normal range for steroid cell tumors, NOS. A younger patient presenting with virilization should be screened for congenital adrenal hyperplasia with serum 17-hydroxyprogesterone. When no obvious mass is seen on imaging studies but strong clinical suspicion persists, percutaneous sampling of all four adrenal and ovarian vessels can localize small steroid-producing tumors, though the success rate is under 50%. In addition, several radiolabeled steroid scans, [131I]aldosterol, [131I]iodocholesterol, and 75Se, can be used to detect ovarian androgen-producing tumors.

Like other ovarian tumors, steroid cell tumors, NOS are managed surgically. Conservative surgery with unilateral oophorectomy can be performed for young patients or those who wish to maintain fertility with stage I disease. For women who have completed childbearing or have advanced disease, total abdominal hysterectomy with bilateral salpingo-oophorectomy and complete surgical staging is indicated. Hormone levels should be monitored postoperatively, as they are expected to return to normal levels within days following tumor resection. Based on trials for metastatic ovarian stromal cell tumors, adjuvant chemotherapy comprised of bleomycin, etoposide, and cisplatin (BEP) should be used in cases of malignancy.

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Presented by Mariam Mafee MD and Jerry Feldman MD

SKINTERMISSION

CASE 6

Presented by Mariam Mafee MD, Shilpa Mehta MD, and Warren Piette MD

A 31-year-old African American man presented with bilateral upper eyelid swelling.

UNKNOWN

Key locations: Upper and lower extremities **CASE 7**

Presented by Tarana Mohammadi MD and Warren Piette MD

History of Present Illness
A 52-year-old man presented to the emergency department with a two-month history of dry, cracking skin on his lower legs. His condition worsened over the past one to two weeks, with painful fissuring, scaling, and swelling. It made ambulation difficult. He denied pruritus. He had never experienced anything similar. The patient was initially managed conservatively with emollients and leg elevation. However, several days later, he again presented to the emergency department with similar complaints.

Past Medical History
Hypertension, chronic kidney disease, hearing impairment

Medications
Amlodipine

Allergies
Ceftriaxone

Family History
No history of skin disease

Social History
Smoker (approximately one half pack per day for 20 years)
Social alcohol
Denied illicit drug use

Review of Systems
Denied fevers, chills, night sweats, nausea, vomiting, and abdominal pain. Endorsed intermittent diarrhea for about two months, associated with a 15-pound weight loss.

Physical Exam
First admission:
Skin: Lower legs: edema, tender fissuring and plate-like scales

Second admission:
Skin: Extensor forearms: diffuse adherent small, brown scales
Lower legs: thick, brown, plate-like scales and intervening fissures
Ankles and dorsal feel: fine wrinkling consistent with resolving edema
Plantar feet: thick hyperkeratosis
Lymph node: Left axillary lymphadenopathy

Laboratory Data
The following labs were remarkable/abnormal:

Hemoglobin	10.6 g/dL	[12.9-16.8 g/dL]
Hematocrit	33.4%	[38.1-49.0 %]
Creatinine	1.9 mg/dL	[0.6-1.4 mg/dL]

Imaging
Non-contrast CT scan of chest/abdomen/pelvis: left axillary adenopathy, with the largest lymph node measuring 70 x 41 mm. Mildly enlarged right axillary lymph nodes present (maximum 20 mm). Mildly enlarged submental lymph nodes are present (maximum 15 mm).

Histopathology
Left axillary lymph node core needle biopsy:
Lymphoid tissue with sheets of large atypical cells, which are anaplastic lymphoma kinase-1 (ALK-1) negative. Immunohistochemical stains reveal large cells that are strongly positive for CD4, CD5, CD43, and CD30. They have variable positive staining for CD3 and CD7 and are negative for CD20.

Diagnosis
Acquired ichthyosis secondary to systemic anaplastic large cell lymphoma (ALCL)

Treatment and Course
Bone marrow biopsy and aspirate showed trilineage hematopoiesis with orderly maturation and no atypical infiltrates. Flow cytometry did not detect an abnormal population of cells.

Hematology and oncology were consulted. The patient was to undergo another staging CT scan and begin chemotherapy, but instead, elected to transfer his treatment and care to an outside hospital.

Discussion
"Ichthyosis" originates from the Greek word for fish, *ichthys*, and is used to describe fish-like scales on the skin. Acquired ichthyosis resembles hereditary ichthyosis vulgaris, with generalized scaling of the skin, but develops later in life. As in the genetic form, it can vary in severity, ranging form minor dryness to dramatic desquamation with plate-like scales. Acquired ichthyosis is commonly distributed on the legs and can be associated with neoplasms, infections, endocrine disorders, inflammatory disorders, malnutrition, or drugs. Cutaneous manifestations can present either before or after identification of the associated disease. The mechanism for the development of acquired ichthyosis is unknown. Secretion of epidermal growth factors or an autoimmune reaction against the skin have been postulated.

Francesco Ronchese first made the association of ichthyosis and malignancy in 1943 when he described a patient diagnosed with Hodgkin disease, who later developed ichthyosis. Hodgkin lymphoma remains the most common malignancy associated with acquired ichthyosis. Various other lymphoproliferative disorders, such as mycosis fungoides, non-Hodgkin lymphoma (reticulolymphosarcoma), and multiple myeloma have rarely been reported. Non-lymphoproliferative malignancies, including dysgerminoma of the ovary, leiomyosarcoma, transitional cell carcinoma of the kidney, and hepatocellular carcinoma, have also been associated with acquired ichthyosis. Consequently, acute acquired ichthyosis prompts investigation for malignancy, especially Hodgkin lymphoma. If associated with lymphoma, the ichthyosis often improves with treatment of the underlying malignancy.

Anaplastic CD30 positive, large-cell lymphoma (ALCL) is a subset of non-Hodgkin lymphoma. Cutaneous ALCL falls along a spectrum of disease with lymphomatoid papulosis. Overall, these diseases have a good prognosis, with a 10-year disease-related survival exceeding 85 percent. Primary systemic ALCL, including primary nodal, behaves more aggressively. Systemic ALCL is further subdivided into ALK-1 positive or negative, depending on whether or not neoplastic cells express ALK-1, which indicates a t(2;5) chromosomal translocation. Both subtypes are treated as aggressive lymphomas. However, ALK-positive ALCL more commonly affects children and young

adults and responds well to chemotherapy, whereas ALK-negative ALCL usually affects patients over age fifty-five and is less responsive to chemotherapy, thus portending a worse prognosis. Nine cases of ALCL-associated acquired ichthyosis have been reported in the literature. Three of these cases had only systemic ALCL (no cutaneous manifestation of the lymphoma), as in the patient presented. The remaining six cases had both cutaneous and systemic ALCL tumors (which sometimes appeared later). No cases of primary cutaneous ALCL have been reported in association with acquired ichthyosis. Among the patients that survived their lymphoma, the acquired ichthyosis regressed with chemotherapy and, in one case, its recurrence heralded a return of lymphoma.

Before ALCL was recognized as a class of lymphoma, most cases were called atypical Hodgkin lymphoma. This may account for some of the previous association with Hodgkin lymphoma and acquired ichthyosis.

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Key location: Distal extremities **CASE 8**

Presented by Katie Manno MD and Warren Piette MD

History of Present Illness
A 45-year-old man presented with a six-day history of cough, shortness of breath, headache, generalized body aches, fever, chills, nausea, and diarrhea. He had just returned from Missouri one week earlier, where he was working for a brick recycling company. He denied any other recent travel, outdoor recreational activities, known arthropod bites, or exposure to pets. He also denied recent exposure to large bodies of water or soil, and he had no sick contacts. In the emergency department, his chest x-ray demonstrated a right lower lobe infiltrate. Therefore, the patient was admitted for community acquired pneumonia and started on ceftriaxone and azithromycin, with addition of doxycycline to cover for intracellular organisms.

On hospital day three, the patient spiked a fever of 101 degrees Fahrenheit, which coincided with worsening mentation and nuchal rigidity. He then developed a new rash on the hands and feet. Due to progressive worsening of symptoms, the patient was intubated, and lumbar puncture was subsequently performed. As the patient was rolled to his right side for the procedure, a tick was spotted on the inner helix of the left ear.

Past Medical History
Asthma, COPD, hypertension

Medications/Allergies
None/NKDA

Social History
Born and raised in Chicago
Occupation: Intermittent construction jobs
Active heroin user
Previous tobacco user (20 pack-years, quit 2014)
No history of alcohol abuse

Physical Exam
Vitals: T 101.8, BP 92/71, HR 112, O2 sat 99-100% on ventilator
Upper and lower extremities: hundreds of petechiae, several scattered small comma-shaped and branching purpura on dorsal and palmar hands up to forearms, as well as dorsal and plantar feet to the calves

Laboratory Data
The following labs were remarkable/abnormal:

WBC	19.3 k/μL	[4.4 – 10.6 k/μL]
Platelet	23 k/μL	[161 – 369 k/μL]
PT/INR	17.1/1.41 secs	[11.8-14.3 secs]
PTT	35.5 secs	[24.2-36.8 secs]
BUN/Cr	107/3.1 mg/dL	[8 – 20 mg/dL] / [0.6 – 1.4 mg/dL]
AST/ALT	369/105 U/L	[0 – 40 U/L] / [5 – 35 U/L]

Microbiology
Legionella urine Ag: negative
Coxiella burnetii IgM/IgG: none detected
Ehrlichia chaffeensis IgM/IgG/PCR: negative
Francisella tularensis Ab: negative
Rickettsia rickettsia IgM: 1:128(<1:64)
Rickettsia rickettsia IgG: 1:128(<1:64)
Rickettsia typhi IgM: 1:128(<1:64)
Rickettsia typhi IgG: none detected

Imaging studies
Chest radiograph: Right lower lobe infiltrate

Diagnosis
Rocky Mountain Spotted Fever

Treatment and Course
The patient was treated with doxycycline for a total of 10 days. He regained consciousness and was extubated on hospital day six. He was sent to rehabilitation on hospital day 15, with subsequent full recovery. The tick found on the patient's ear was sent to the CDC and identified as *Dermacentor variabilis*.

Discussion
Rocky mountain spotted fever (RMSF) is a tick-borne illness that was first described in 1899. The causative pathogen is *Rickettsia rickettsii*, which is transmitted most commonly by the *Dermacentor variabilis* (dog tick), *Dermacentor andersoni* (wood tick), or *Rhipicephalus sanguineous* (brown dog tick). According to the Center of Disease Control data from 1997-2002, it was estimated that the annual incidence of RMSF is 2.2 cases per million. More than half of the RMSF cases are reported from only five states: North Carolina, South Carolina, Tennessee, Oklahoma, and Arkansas. However, there have been cases reported from all 48 contiguous states except Vermont and Maine.

R. rickettsii infects endothelial cells of vessel walls and proliferates in the cytoplasm. The organism spreads from cell-to-cell via filopodia propulsion, causing injury to the microcirculation of various organ systems, including the brain, liver, skin, lungs, kidneys, and gastrointestinal tract. Vasculitis occurring in extracutaneous organs (e.g. brain or lungs) results in life-threatening complications.

The initial presentation of RMSF is often non-specific, consisting of fever and vague constitutional symptoms. The "classic triad" of fever, rash, and headache is present in less than 60% of patients with laboratory-confirmed disease. If cutaneous findings do occur, they typically appear two to four days after fever onset. In addition, a specific history of a tick bite within two weeks of illness onset is only reported in approximately 60% of cases.

A classic case of RMSF has distinct early and late cutaneous findings that may help to indicate the time course and severity of disease. Early disease manifests as erythematous macules on wrists, forearms, and ankles. Lesions spread to involve the palms and soles, and then move centripetally. The red-to-violaceous petechial and purpuric rash is usually seen on or after day six of symptoms and only occurs in 35-60% of patients. When the rash is more generalized, or the branching, petechial and purpuric eruption is appreciated, this is often an indication of progression to severe disease. Every effort should be made to initiate treatment prior to development of this severe stage. However, a rash may be completely absent or atypical in 10-20% of cases.

Diagnosis of RMSF is challenging, as the early clinical presentation is difficult to distinguish from other infectious and non-infectious diseases, and routine laboratory testing is non-specific. Confirmatory testing options include serologic testing, nucleic acid detection, immunohistochemical staining, and culture. The indirect immunofluorescence antibody (IFA) assay is considered to be the gold standard for diagnosis of RMSF, with an estimated sensitivity of 94 to100% after 14 days.

Prompt treatment is crucial, as the mortality rate of RMSF is nearly 30% without therapy. Because serum antibodies are often not detectable until 7-10 days after onset of illness, physicians should initiate anti-rickettsial treatment prior to complete work-up if clinical suspicion is high. Once rickettsial infection is suspected, both adults and children should begin empiric treatment with tetracycline antibiotics, particularly doxycycline. This class is considered the therapy of choice, because all pathogens causing tick-borne rickettsial disease are susceptible. Treatment should be continued for at least three days after the patient's fever resolves, with a minimum course duration of 5-7 days.

RMSF became a nationally reportable disease in 1989. When a potential case has been identified, the health-care provider should notify the local health department, who can then assist the provider in obtaining the appropriate diagnostic testing. A probable case of RMSF is defined as a patient with clinically compatible illness, and the presence of serum antibodies reactive with *R. rickettsia* at a titer considered indicative of current or previous infection. A diagnosis of RMSF is confirmed by a four-fold change in serum antibody titer as determined by IFA or ELISA, positive PCR, immunohistochemistry, or culture. Once a case of RMSF has been confirmed, it is then reported to the state health department.

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Key locations: Face, forearms, dorsal hands, lower legs **CASE 9**

Presented by Sangeetha Venkatarajan MD, Warren Piette MD, and Lisa Arkin MD

History of Present Illness
An 8-year-old Hispanic boy presented with a rash that developed one-month prior to presentation during the summer. The rash was associated with pruritus and burning. No other family members had similar findings. There were no previous episodes. He denied any eye pain, eye redness, lip changes, or oral pain.

Past Medical History
None

Medications/Allergies
None/NKDA

Review of Systems
Negative for fever, chills, nausea, vomiting, and diarrhea.

Physical Exam
Cheeks, temples: erythematous papules, some coalescing into thin plaques with crusting
Forearms, dorsal hands, posterior neck, upper chest, lower legs: erythematous papules, some with crusting, some with erosions
Back, abdomen, thighs, upper arms, buttocks: spared
Oral mucosa, lips, conjunctivae: clear

Laboratory Data
The following labs were remarkable/abnormal:

WBC	10.4 k/uL	[4.4-10.6 k/uL]
Hemoglobin	13.8 g/dL	[12.0-14.0 g/dL]
Hematocrit	41.4 %	[35.8-42.5 %]
Platelets	408 k/uL	[235-534 k/uL]
Absolute eosinophil count	1.5 k/uL	[0.0-1.0 k/uL]
LDH	267 U/L	[85-210 U/L]
ANA	Negative	

Diagnosis
Actinic prurigo

Treatment and Course
The patient was initially treated with an oral prednisone taper, starting at 0.5mg/kg/day, with mild improvement. He was then started on topical treatment with triamcinolone ointment and tacrolimus ointments with only modest improvement. In addition, daily photoprotection was recommended. His mother had difficulties bringing him to clinic, and treatment was sporadic. The lesions recurred when the topical treatment was weaned. Eventually, oral thalidomide 50mg daily was started with near resolution after one month, but recurred during a lapse off therapy. We plan to continue the patient on a maintenance dose of 25mg per day.

Discussion

Actinic prurigo (AP) is an uncommon, idiopathic photodermatosis with a female predominance. It most commonly occurs in Native Americans and those with Mestizo ancestry. AP is usually seen in skin phototypes III to V and typically begins in childhood, with an average age of onset between six to eight years of age. The course tends to be year-round, with acute flares during the summer.

The most accepted theory regarding pathogenesis for AP is a delayed hypersensitivity reaction to an unidentified autoantigen induced by ultraviolet (UV) radiation in genetically susceptible individuals. The presence of activated CD4-positive and memory T lymphocytes in the infiltrate, abnormal reactivity of AP lymphocytes against UV-irradiated keratinocytes, and a more intense proliferative response to isolated autologous skin antigens in AP patients support this hypothesis.

AP typically presents with intensely pruritic lesions localized to sun-exposed facial areas, neck, V-area of the chest, extensor arms and forearms, dorsal hands, and lower legs. Sun-protected sites are also involved in approximately 35-40% of patients. The lesions are typically flat, shiny, polygonal papules. However, cone-shaped papules with small hemorrhagic crusts can also be seen. Secondary changes may include edema, scaling, fissures, crusting, hyperpigmentation, ulceration, and scarring.

Histopathologic findings are non-specific. Hematoxylin and eosin-stained specimens show hyperkeratosis with orthokeratosis or parakeratosis, regular acanthosis, focal or multifocal spongiosis, thickening of the basal membrane, dense perivascular lymphocytic infiltrate in superficial and mid dermis, and papillary dermal edema.

Management of AP requires strict photoprotection including sun-protective clothing with a wide-brimmed hat and sunglasses, and high sun protection factor, broad-spectrum sunscreens. Patients should avoid working or standing by windows to minimize UVA exposure. Therapies including topical potent corticosteroids, topical calcineurin inhibitors, short courses of oral corticosteroids, pentoxifylline, cyclosporine, and azathioprine have all been attempted with varying results. Thalidomide has the best quality evidence available in the literature. In 34 patients with AP treated with thalidomide, Londoño et al. noted improvement in 32 patients within an average of 50 days. In a smaller study, Lovell et al. treated 14 AP patients with thalidomide and noted improvement in 11 patients. Teratogenicity is the most notorious side effect of thalidomide, for which enrollment in the Risk Evaluation and Mitigation Strategy (REMS) program is required for all patients. Other common side effects include constipation, fatigue and orthostatic hypotension. Thalidomide-induced neuropathy is dose-dependent, and longer courses at lower doses (i.e. 25mg once daily) may be more protective than shorter courses of thalidomide at higher doses.

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CASE 10

Presented by Barry Ladizinski MD MPH MBA, and Joerg Albrecht MD PhD

A 55-year-old homeless man with schizophrenia and ethanol abuse presented with large, minimally painful ulcerations on his scalp and neck.

UNKNOWN