Chicago Dermatological Society

April 2009
MONTHLY EDUCATIONAL CONFERENCE

Program Information
Continuing Medical Education Certification
and
Cases Submitted by Members

Saturday, April 18, 2009

The Hyatt Lodge at McDonald's Campus
Oak Brook, Illinois

Program

Registration

12:00 p.m.	Registration opens
12:00 p.m. - 1:00 p.m.	Buffet lunch & visit with exhibitors *Prairie Ballroom Foyer*

Program Activities

1:00 p.m. - 2:50 p.m.	**General Session** *Prairie Ballroom*
1:00 p.m.	Announcements and CDS business *Warren Piette, MD*
1:05 p.m.	Monitoring Activities of Autoimmune Bullous Diseases *Lawrence Chan, MD*
1:45 p.m.	Case discussions *Moderated by: Warren Piette, MD*
2:50 p.m. - 3:10 p.m.	**Break and visit with exhibitors** *Prairie Ballroom Foyer*
3:10 p.m. - 5:00 p.m.	**General Session** *Prairie Ballroom*
3:10 p.m.	Regression in Melanoma *Christopher Shea, MD*
3:35 p.m.	Case Discussions *Moderated by: Warren Piette, MD*
4:15 p.m.	Interactive Purpura *Warren Piette, MD*
5:00 p.m. - 6:00 p.m. *Prairie Ballroom Foyer*	**Reception for CDS Members and Guests**

CME Information

This activity is jointly sponsored by the Chicago Medical Society and the Chicago Dermatological Society.

This activity has been planned and implemented in accordance with the Essentials Areas and Policies of the Accreditation Council for Continuing Medical Education (ACCME) through the joint sponsorship of the Chicago Medical Society and the Chicago Dermatological Society. The Chicago Medical Society is accredited by the ACCME to provide continuing medical education for physicians. The Chicago Medical Society designates this educational activity for a maximum of three (3) *AMA PRA category 1 credits*™. Each physician should claim only those hours of credit that he/she actually spent in the educational activity.

Commercial Support: There are no educational grants associated with this meeting. One or more companies may pay a fee to be present as exhibitors. The program content is free from any commercial or corporate influence.

Guest Speakers

Lawrence S. Chan, MD – Chairman, Department of Dermatology, University of Illinois at Chicago

Christopher R. Shea, MD – Chief, Section of Dermatology, University of Chicago Medical Center

Warren Piette, MD – Chairman, Division of Dermatology, Stroger Hospital of Cook County

Speaker CME Disclosure of Financial Interests

Dr. Chan: Publication, Mason Publishing (American Society of Microbiology Press)
Dr. Shea: No significant financial interests to disclose.
Dr. Piette:

CME Credit Documentation

Course Director: Warren Piette, MD

Target Audience: Practicing dermatologists, dermatology residents and fellows.

Objectives: At the conclusion of this learning activity, the participant should be able to:

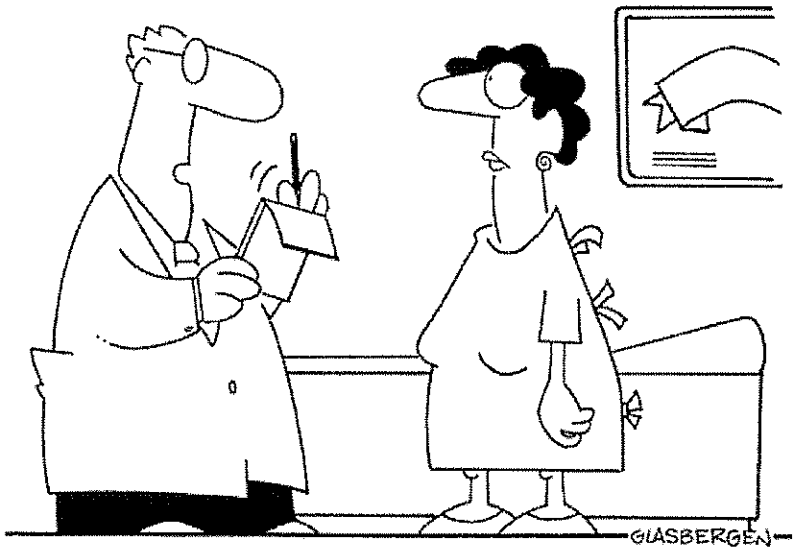
1. Discuss differential diagnoses for unusual presentations of common dermatoses
2. Describe therapeutic options for patients with common, but recalcitrant, dermatologic syndromes
3. Describe the clinical progressions of autoimmune bullous, purpura and vasculitis diseases

Following the meeting, the Chicago Medical Society will send you a certificate documenting your attendance at this conference and the number of Category 1 CME credits you earned.
It is essential that you sign the CME sign-in sheet located at the Chicago Dermatological Society registration desk. Do so before you leave the conference! If you have any questions about your credits, please contact the Chicago Dermatological Society at 847/680-1666, or by email: RichardPaul@DLS.net

Evaluation Forms

Please complete and return your meeting evaluation form. This feedback is an important part of the CME process and helps us to design programs in the future that better meet the needs of our members. Note that the form will be scanned by computer; keep your responses within the spaces provided, and avoid making any extraneous marks on the sheet. Thank you!

Cases Submitted from the Community



"I'm prescribing a squiggly line, two slanted loops, and something that looks like a P or J."

Form Entry Detail: Case Discussion Submission - April 2009 Conference

Entered: 04/07/2009 @ 10:06 AM - Edit Entry

First Name: Gina Clementz

Last Name: Marafino

Degree: MD

Practice name: Dermatology Partners of North Shore

City: Chicago

Practice type: Private practice

Case title: aggressive right hand dermatitis

Key locations: right palm

History: 15 year girl with acute onset itchy right palm X 1 week, long history of atopic dermatitis, seen again within second week of symtoms due to rapid progression of appearance and intensity of symptoms (pruritis, sting)

Past history: atopic dermatitis

Medications & Allergies: nkda

Family history: may provide this info at end if you want to stump the audience (pet hedgehog x 6 months no other family members affected patient is primary caregiver of hedgehog)

Physical exam: Dr marafino 1st visit 1: R hand palm: dried vesicles within a 1.5 x 1.5 cm pink plaque, rare small pustule interdigital, KOH -, fungal culture performed (photo 1) Dr. Marafino 2nd: annular erythema with pustules 3 x 3 cm (photo 2)

Laboratory data: fungal culture T. mentagrophytes

Diagnosis: withheld

Discussion: Diagnosis: T. manum (T. mentag.) from pet hedgehog...several cases have been described to date (most outside of the US)...now that exotic pets are more common in the USA cases such as this one are possible....Hedgehogs are also known to harbor Salmonella. The patient just started lamisil 250 qd and soaking in Domboros.

Other comments or notes:

Last Updated: 04/07/2009 @ 10:15 AM CST by Gina Clementz Marafino

Members24x7 Administration: Chicago Dermatological Society

Form Entry Detail: Case Discussion Submission - April 2009 Conference

Entered: 04/09/2009 @ 04:51 PM - Edit Entry

First Name: Edward

Last Name: Peterka

Degree: MD, MS

Practice name: Cottage Professional Building

City: Galesburg

Practice type: Private practice

Case title: Porphyria Cutanea Tarda

Key locations: Face and hands

History: The patientis a 54 year old white male who presented with sores and blisters on the dorsum of both hands. He stated that the condition began in Summer 2007 and Spring 2008. He first noted the blisters on the dorsum of his hands and forehead. He reported that he had Hepatitis C and that his urine was dark. He gave no history of drug ingestion. He gave a history of alcoholism.

Past history: As above.

Medications & Allergies: None

Family history: None

Physical exam: The patient presented as a well-developed and well-nourished 54 year old white male in no acute distress. He was pleasant, cooperative, friendly, oriented to time and place and was healthy appearing. His scalp and forehead showed crusted lesions. He showed skin fragility, hyperpigmentation, and hirsutism of the face.

Laboratory data: Fasting blood sugar (done previously) was 83 mg/DL (normal 70-99). Porphyrin studies showed a Uroporphyrin 4887.3 mg per 24 hours (normal 3.3-29.5), Heptacarboxyporphyrin 2289.4 mg per 24 hours (normal 6.8 or less), Hexacarboxyporphyrin 186 mg per 24 hours (normal 0.9 or less), Pentacarboxyporphyrin 248.7 mg per 24 hours (normal 4.7 or less, Coproporphyrins 846.6 mg per 24 hours (normal 155 or less). Porphyrin totals 8290.3 mg per 24 hours (normal 12-190. Additional labs: Alkaline Phosphatase 356 (normal 38-126) Gamma GT 586 (normal 15-73) AST 71 (normal 17-59) ALT 58 (normal 21-72) Hemoglobin 14.2 (normal 13.0-16.5) Hematocrit 40.3 (normal 38.0-50.0) Ferritin 387 (normal 18-464) Total Iron Binding Capacity 136 (normal 49-181) Percent saturation 56 (normal 15-62) HIV antibody screen showed none detected.

Diagnosis: Porphyria Cutanea Tarda, possible Variegate Porphyria

Discussion: The patient was started on Silymarin 140 mg per capsule-taking one capsule four times daily. He was also advised to abstain from alcohol and avoid iron in his vitamins.

Other comments or notes: Photo files coming under separate cover

Last Updated: 04/09/2009 @ 04:08 PM CST by Edward Peterka

Members24x7 Administration: Chicago Dermatological Society

Form Entry Detail: Case Discussion Submission - April 2009 Conference

Entered: 04/12/2009 @ 11:02 PM - Edit Entry

First Name: Kathleen

Last Name: Remlinger

Degree: MD

Practice name: Fox Valley Dermatology

City: Aurora

Practice type: Private practice

Case title: Darier's

Key locations: Back, neck, arms, chest

History: 12 year history of pruritic eruption that had been treated with topical steroids (low through high potency), pimecrolimus, 12% lactic acid, 20% urea, oral antihistamines. Condition waxes and wanes, but doesn't clearly respond to specific therapies.

Past history: Knee replacement (2002), hysterectomy (2000), appendectomy (1958), arthritis.

Medications & Allergies: Meds: ASA 81 mg, Fosamax, istacol. Allergies: Duricef (unknown reaction), many analgesics (mainly GI symptoms, hallucinations etc).

Family history: Seasonal allergies and asthma. No similar skin disorder.

Physical exam: Back and arms with cruted keratotic papules scattered on arms and upper left back but clustered on lumbar skin. Keratoses on palms with V notch on left index fingernail and red and white streaking of some fingernails. Blotchy erythema in V of chest.

Laboratory data: Pathology (lower back 2-27-09) vertical parakeratosis overlying suprabasilar clefts. Grains and corps ronds present. Superficial perivascular lympphohistoiocytic infiltrate in dermis.

Diagnosis: Darier's disease.

Discussion: Pruritus and lesions on arms and chest improved with fluocinonide cream. Patient was started on vitamin A 10
000 IU BID for 2 weeks then once daily.

Other comments or notes: Treatment suggestions.

Last Updated: 04/12/2009 @ 11:02 PM CST by Kathleen Remlinger

Form Entry Detail: Case Discussion Submission - April 2009 Conference

Entered: 04/12/2009 @ 11:07 PM - Edit Entry

First Name: Kathleen

Last Name: Remlinger

Degree: MD

Practice name: Fox Valley Dermatology

City: Aurora

Practice type: Private practice

Case title: Pityriasis rubra pilaris

Key locations: Scalp, chest, abdomen, arms, buttocks

History: Patient developed very pruritic eruption 2 and a half years ago. She was originally treated with topical steroids, and then with dapsone for 3 months. In June 2007 she was hopitalized with congestive heart failure and pancreatitis. She received antibiotics and a liquid diet and her skin cleared, but flared soon afterward.

Past history: Vitiligo for 15 years. Diabetes mellitus, arthritis, hypertension, thyroid disorder, colitis, congestive heart failure, cholecystectomy, appendectomy, hysterectomy, knee replacements.

Medications & Allergies: Furosemide, glipizide (since 2007), lisinopril (for over 10 years), Januvia, coumadin, propranolol, methimazole (stopped 2009), Advair, Klorcon, Detrol LA, protonix, Celexa

Family history: Seasonal allergies.

Physical exam: Erythema and scaling in scalp and posterior neck. Erythema with fine scale on mid chest with coalescing erythematous macules under breasts. Marked palmar hyperkeratosis with onycholysis and yellowing of nails. Pink scaly papules on arms, some with follicular accentuation, especially in left antecubital fossa. Legs show erythema and dyspigmentation with hyperkeratosis of soles. Buttocks show pink ill-defined scaly patches.

Laboratory data: CMP: glucose 176, BUN 34, creatinine 1.21, AST 52 (10-35), HbA1C 7.1. The following were normal: CBC with differential, lipid panel, electrolytes, calcium, total protein, albumin, total bilirubin, ALT, alkaline phosphatase, urine microalbumin, T4, TSH. Pathology (left inner forearm 12/07): epidermis is mildly atrophic with minimal spongiosis and focal parakeratosis around a dilated follicle with focal plugging. PAS stain negative. Mild chronic dermatitis and pityriasis rubra pilaris can be considered.

Diagnosis: Pityriasis rubra pilaris

Discussion: Patient was treated with acitretin 25mg daily with initial improvement, but then worsening in folds. Latter was treated with mixture of Naftin and Pandel cream. Narrow band UVB was started 2-5-08. Patient was hopitalized March 2008 with MRSA so went off acitretin and phototherapy. Skin flared October 2008 and patient was restarted on acitretin 25mg daily to every other day. She developed retinoid dermatitis and was switched to therapy with methotrexate February 2009 with a good response.

Other comments or notes: Diagnosis (PRP versus psoriasis), and treatment suggestions.

Last Updated: 04/12/2009 @ 11:54 PM CST by Kathleen Remlinger

Members24x7 Administration: Chicago Dermatological Society

Form Entry Detail: Case Discussion Submission - April 2009 Conference

Entered: 04/15/2009 @ 09:29 PM - Edit Entry

First Name: Kathleen

Last Name: Remlinger

Degree: MD

Practice name: Fox Valley Dermatology

City: Aurora

Practice type: Private practice

Case title: Pruritus

Key locations: Trunk and extremities

History: Patient has 20 year history of itching all over body, especially severe since 2003. Notes small itchy lesions that resolve with hyperpigmentation.

Past history: Hypothyroidism, hypercholesterolemia, arthritis of knee, seasonal allergies, appendectomy.

Medications & Allergies: Meds: Levothyroxine, fluoxetine (2004). More recently also on hydrocodone with acetaminophen, atorvastatin, trazodone, glucosamine. Allergies: ASA

Family history: No family history of asthma, psoriasis, other skin condition.

Physical exam: 52 year old Southeast Asian woman. Mild ill defined hyperpigmentation of lids and right lateral cheek. Reticulated hyperpigmentation on trunk and extremities with sparing in butterfly pattern on back and sparing below breasts. A few 3mm erythematous papules with central erosion on right antecubital and on left back. Mild dermatographism.

Laboratory data: CBC normal except mildy increased absolute eosinphil count 561 (15-500). ESR 38. Urinalysis trace leukocyte esterase. Urine culture with mixed organisms. The following were normal or negative: CMP, lipid panel (on atorvastatin), anti-mitochondrial antibodies, ferritin, ANA, IgE, chest x-ray, patch testing to 61 allergens, PPD. Pathology (2-26-07) L arm 1: perivascular inflammation with patchy parakeratosis and dermal fibrosis. Left arm 2&3: papillary dermal hyaline deposits. L hip: psoriasisform epidermal hyperplasia with perivascular inflammation.

Diagnosis: Withheld

Discussion: Patient gets some relief with class I and II topical steroids. She has also tried Lidamantle cream, Protopic ointment, Elidel cream, antihistamines, doxepin, naltrexone 50mg daily, and narrow band UVB without improvement. Patient was started on cyclosporine with marked improvement in pruritus though patient continues to get some itchy papules.

Other comments or notes:

Last Updated: 04/15/2009 @ 09:28 PM CST by Kathleen Remlinger

Members24x7 Administration: Chicago Dermatological Society

Form Entry Detail: Case Discussion Submission - April 2009 Conference

Entered: 04/15/2009 @ 10:02 PM - Edit Entry

First Name: Kathleen

Last Name: Remlinger

Degree: MD

Practice name: Fox Valley Dermatology

City: Aurora

Practice type: Private practice

Case title: Ulcerating bullous dermatosis on legs

Key locations: Legs

History: 53 year old Asian woman developed blisters evolving to very painful ulcers starting Christmastime 2007. She had traveled to Washington, DC and to New York in summer of 2007, but no foreign travel. Patient had been treated with potent topical steroids, terbinafine cream, and mupirocin ointment without improvement. Patient stated legs were infected.

Past history: Healthy.

Medications & Allergies: Meds: multivitamins. Allergies: NKDA

Family history: Negative for asthma, eczema, seasonal allergies, psoriasis, autoimmune disorders.

Physical exam: Legs inclulding feet showed several ulcers many with fibinoid debris and other with re-epithelialization. Some had violaceous borders. surrounding skin was mildly atrophic. Hyperkeratosis of soles. Marked onychauxis and subungual debris.

Laboratory data: Fungal culture negative. Bacterial culture: mixed skin flora including coagulase negative Staph. the following were normal or negative: CMP, CBC, ANA, anti-epidermal antibodies (9/08), direct immunofluorescence (10/08), vascular ultrasound. Pathology: L achilles (8-5-08) Surface of epidermal cyst or changes could represent chronic eczema. L lateral Achilles (8-13-08) subepidermal vesicular dermatitis with re-epithelialization.

Diagnosis: Withheld

Discussion: On 9-23-08 patient was given cyclosporine and trimethoprim-sulfamethoxazole by mouth, and lidocaine ointment topically. Largest ulcers were covered with hydrocolloid dressings. Two weeks later patient had improvement in old ulcers but continued to develop new ones. She began Bandaid antiseptic wash and developed contact dermatitis which improved with cessation of wash. Patch test confirmed allergy to benzalkonium chloride. New serpiginous lesions appeared around toes. A repeat biospy was performed 10-15-08. After results were received, cyclosporine was stopped, therapy was changed and patient's ulcers healed completely in 2 months.

Other comments or notes:

Last Updated: 04/15/2009 @ 10:14 PM CST by Kathleen Remlinger

Members24x7 Administration: Chicago Dermatological Society

Form Entry Detail: Case Discussion Submission - April 2009 Conference

Entered: 04/13/2009 @ 09:23 PM - Edit Entry

First Name: Gary

Last Name: Barsky

Degree: MD

Practice name:

City: Elmhurst

Practice type: Private practice

Case title: Severe childhood progressive pigmentary purpura

Key locations: left leg, thigh, and forearm

History: 9 y/Hispanic female born in USA while spending one month in Mexico at age 2 1/2 developed asymptomatic red light brown discoloration on the left mid leg that has extended gradually to most of the left lateral leg, left posterior thigh, and intermittently on the left forearm. The patient has had no treatment until one month ago used halobetasol cream for two weeks with no improvement.The redness is less noticeable upon awakening and bathing makes it much more prominent. A thorough lab workup showed a positive ANA 1:1280 The patient was seen three weeks ago by Loyola University Rheumatology who said she doesn't have SLE but lab tests are pending

Past history: NOrmal vaccination up to date and no history of having any chilhood illnesses .There is no family history of connective tissue disease. The mother hasa history of breast cancer

Medications & Allergies: no known medication alllergies

Family history: No connective disease or purpuric eruptions. THe mother has ahistory of breast cancer.

Physical exam: Deep red blanchable nontender erythema over the left leg and left posterior thigh. Negative lymphadenopathy. Normal oral mucosa.

Laboratory data: ANA 1:1280 and 1;640 pattern pending.CBC CMP normal
, Connective tissue profile pending Two skin biopsies have been performed and should be ready for viewing

Diagnosis: withheld

Discussion: Does positive ANA have significant relevance to the progressive pigmentary purpura and what further workup should be performed

Other comments or notes:

Last Updated: 04/13/2009 @ 09:52 PM CST by Gary Barsky

Gary Jay Barsky, M.D.
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Chicago Dermatological Society April 18th, 2009 Monthly Conference

Case presented for diagnosis and treatment options

The patient is a 56-year-old female born in Greece who has been in good general health up until 2 years ago when she developed asymptomatic red-brown patches over the back of her left hand and her left posterior arm. This was untreated and stable until August 2008 when it started spreading to other scattered areas on her body. She saw multiple dermatologists since then who gave her treatments such as Lustra AF cream none of which helped. In the last 3 months, she has had treatments with the fraxel laser and an IPL vascular setting which have not helped. She is in good general health , without any medication allergies, on no internal medications nor herbal dietary supplements. She has no history of connective tissue disease. She has a history of intermittent low platelet counts.The review of systems is otherwise negative and normal.

The family history is negative for any connective tissue disease, significant diseases, or similar significant skin problems.

The physical examination reveals a pleasant, healthy-appearing, an alert and oriented individual x3. There are blanchable nonpalpable red-brown purpuric patches over the dorsal left hand, right foot, and under the right arm.

A hematological workup within the past 2 weeks was normal. Platelet function normal, anti-cardiolipin antibody negative, ANA negative

This is a case for diagnosis and treatment options

Gary J. Barsky M.D.

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FAX

BARSKY

0 - 837 - 8145

A-A(-) A(+)
PFS ~1

I saw [redacted] in consultation. This is a 56 year-old female who apparently has a history of thrombocytopenia dating back at least 10 years. She has not had a previous hematologic evaluation. She has a four-to-five year history of noting "blood spots" on the left side of her face and over her right eyelid after she vomits. Apparently, she used to vomit frequently with her monthly menses. She is now in menopause and she has not vomited since last summer. She feels that this rash looks like red blood and not a bruise. She has not had any other signs of bleeding. She denies epistaxis. Her menstrual periods have always been quite light. She has not had any bleeding with prior childhood surgery.

She also has had various rashes on her hands and arms. The most prominent one is on the dorsum of her left hand. She has been seen by multiple dermatologists without a clear diagnosis. She has recently received some laser therapy to the dorsum of the left hand to try and eliminate the discoloration. Last summer in Greece, her platelets were 95,000. In September in your office, her platelets were 134,000. Her CMP was normal. Her ESR was 4 and her CRP was within normal limits.

Past medical history: None.

Past surgical history:

1. Tonsillectomy.
2. Appendectomy.

Past radiation history: None.

Gynecologic history: Gravida 4, Para 2 with 2 miscarriages both early in pregnancy. She is menopausal.

Medications: Multivitamin, calcium, occasional Motrin. She took one Motrin yesterday.

Allergies: Penicillin which apparently caused a urinary tract infection.

Social history: She is a married female who owns a boutique. She has smoked a quarter pack of cigarettes a day for 30 years. She denies alcohol use.

Family history: She has two daughters who are well without a history of bleeding. She has a brother and two sisters alive and well without a history of bleeding. Both parents are alive with a history of cardiac disease.

Review of systems: She denies anorexia or weight loss. There have been no fevers, chills or sweats. She has occasional headaches. There has been no change in vison or hearing, no sores in her mouth and no shortness of breath, cough or chest pain. Her bowels are regular without black or bloody stools. There has been no hematuria or dysuria and no joint swelling or pain. She has occasional numbness in her right hand with cold exposure.

On physical exam, she is 5 feet 5 inches tall and weighs 147 pounds. The pulse is 72. The blood pressure is 130/80. She is oriented x 3. She appears well. Pupils are equal and reactive. Extraocular motion is intact. Sclerae are anicteric. There is no cervical, supraclavicular, axillary or inguinal adenopathy. Lungs are clear. Heart is regular without murmur. The abdomen is soft and nontender. There is no hepatosplenomegaly. Extremities: There is no clubbing, cyanosis or edema. Neurologically, she is intact. On the dorsum of her left hand, there is a flat brown/purple discoloration overlying the base of the fourth and fifth fingers. She has no petechiae or purpura on her body.

In our office today, the white count is 6.4, hemoglobin of 13.4 and platelets of 115,000. Her smear was reviewed. The red cells were normochromic and normocytic. There was no significant aniso or poikilocytosis. The white blood cells matured normally without atypical forms. There were no Jolly bodies noted. The platelets were mildly decreased. The platelet morphology was notable for many very large platelets. Only a few of them were larger than a red blood cell. They had normal coloration.

Assessment:

1. Thrombocytopenia. This has been mild and chronic for many years. The etiology of this is not certain. It is possible that she could have a mild chronic ITP. She could have an anticardiolipin syndrome. The very large appearing platelets raise the possibility of congenital thrombocytopenia. There are three disorders that often present with quite large platelets. One is called Bernard-Soulier, the other is Gray Platelet Syndrome and the May-Hegglin anomaly. These disorders are often associated with a mild bleeding diathesis. The May-Hegglin Syndrome generally has white cells present with Jolly bodies and the Gray Platelet Syndrome has abnormally colored platelets. Neither of these are apparent on her peripheral smear and she does not seem to have a bleeding diathesis. Her menses have always been light and she has not had bleeding with any surgical procedures.
2. Bloody rash on her face after vomiting. This is quite difficult for me to picture as it is not apparent at this time. She does not seem to think that this was an ecchymotic or purpuric type of rash.
3. Rash over her arms and the dorsum of her hand. This also is not petechiae or purpuric. The diagnosis has been elusive despite seeing many dermatologists as well as having a skin biopsy. It is unlikely that she has a leukocytoclastic vasculitis with a normal sed rate and CRP.

Plan: I have ordered an anticardiolipin antibody panel and an ANA to screen for an autoimmune disease. I would like to check a platelet function screen; however, she took a Motrin today. I have instructed her not to take Motrin for the next two weeks and then to obtain a platelet function screen. If she has

abnormal platelet function, I will need to more seriously consider one of the rare congenital syndromes that are associated with large platelets and platelet function abnormalities. If this is normal, it will rule out these disorders. I do not know how to tie in her unusual skin rash with her thrombocytopenia at this time. Because her thrombocytopenia is quite mild and chronic, I do not feel that a bone marrow biopsy or any further workup is necessary at this time. I would certainly be happy to reevaluate her if she has a sustained decline in her count.

Members24x7 Administration: Chicago Dermatological Society

Form Entry Detail: Case Discussion Submission - April 2009 Conference

Entered: 04/15/2009 @ 02:30 PM - Edit Entry

First Name: Ana

Last Name: Eng

Degree: MD

Practice name: Elmhurst Dermatology

City: Elmhurst

Practice type: Private practice

Case title: dermatofibroma or dermatofibrosarcoma protruberans

Key locations: upper back

History: 47 year old lady with a lesion on the back for one year. It was removed twice 5/23/2008 & 7/11/2008 with lesions measuring 10x10x3mm and 15x10x3 mm respectively. This was interpreted as "dermatofibroma" twice.

Past history:

Medications & Allergies:

Family history:

Physical exam: A 1 cm growth on the upper back

Laboratory data:

Diagnosis: dermatofibroma or dermatofibrosarcoma protruberans

Discussion: The two pathologic tissues show very partial superficial specimens. These were both interpreted as dermatofibroma.

Other comments or notes: Problems: 1. Both previous surgeries show partial superficial tissues. 2. There were no concern on possible dermatofibrosarcoma protuberans by both the clinician and pathologist.

Last Updated: 04/15/2009 @ 02:19 PM CST by Ana Eng

CHICAGO DERMATOLOGICAL SOCIETY

DATE: April 18, 2009

PATIENT: VS

PRESENTED BY:
Harry Goldin, M.D., Tomas Victor, M.D., Thomas Cibull, M.D.

HPI: 68 year old white man who lives in the Ukraine presented in February 2009 with a:

- 40+ year history of red patches on the legs that come and go.
- 2 year history of recurrent leg ulcers.
- 1 year history of an ulcer on the right helix with secondary loss of tissue of the after standing near an air conditioner.

8 years ago he was diagnosed with cryoglobulinemia.
Past treatments have included:

- Plasmaphoresis in 11/06 and 2/07 which were not helpful
- Prednisolone 5 mg in October and November 2008.

He denies Raynauds's phenomena.

His condition worsens after exposure to cold. He notes a recent flare which occurred during the flight from the Ukraine to the USA.

PMH: Hypertension.

MEDS: Hypertension medication.

PHYSICAL EXAM:
There are retiform, hyperpigmented patches on the arms, trunk and legs.
There are red macules and papules-some crusted- on the legs.
There are red macules on the dorsum of the hands.
There is a "bit out" scar on the right ear with surrounding petichiae.
There is a violaceous macule on the left helix.
There is erythema on the shaft of the penis.

There are no ulcers on the finger tips or toes.
There is a 1.5 cm ulcer with a crusted surface on the right posterior ankle.
There is a 0.5 cm superficial ulcer on the left posterior ankle.

LABORATORY:
CBC: Hbg 11.4; WBC normal with normal diff
ESR: 12 (0-10); CRP: 1.2 (<1.0)
Rheum Factor: Negative
Urinalysis: 5-10 RBC/HPF (Normal: 0-3)
IgG, IgA, IgM: Normal
ANA, SSA, SSB: Negative
C3: 83 (90-180)
C4: 6 (16-47)
Hepatitis panel: Negative

Normal serum protein levels
Immunoelectrophoresis: Faint monoclonal free lambda light chain

Cryofibrinogen: Negative
Cryoglobulin qualitative: Positive
Marked cryoprecipitate with cryocrit 1.0%

Bone marrow biopsy: Consistent with monoclonal gammopathy of undetermined significance (MGUS).
Normocellular marrow with multilineage hematopoesis and mild interstitial plamacytosis (5-10%)and B-cell lymphocytosis (5-10%) associated with monoclonal plasma cell population and abnomal B-cells detected by flow cytometry.

Skeletal survey: Negative
Bence-Jones Protein: Negative

BIOPSY: Leukocytoclastic vasculitis.

DX: Cryoglobulinemia with vasculitis and monoclonal gammopathy.

Table 25.5 -- Classification of cryoglobulins.

CLASSIFICATION OF CRYOGLOBULINS.				
Subtype	**Molecular composition**	**Associations**	**Pathophysiology**	**Clinical manifestations**
I	Monoclonal IgM or IgG	Plasma cell dyscrasias, lymphoproliferative disorders	Vascular occlusion	Raynaud's phenomenon, retiform purpura, gangrene, acrocyanosis
II	Monoclonal IgM[*] (>IgG[*]) against polyclonal IgG	HCV, HIV, autoimmune connective tissue diseases, lymphoproliferative disorders	Vasculitis	Palpable purpura, arthralgias, peripheral neuropathy, glomerulonephritis
III	Polyclonal IgM[*] against IgG			

HCV, hepatitis C virus; HIV, human immunodeficiency virus.

[*] Typically have rheumatoid factor activity (i.e. are directed against the Fc portion of IgG).

Cases Presented by Dr. Piette

TABLE 1.

DIFFERENTIAL DIAGNOSIS OF PETECHIAL HEMORRHAGE – NON-PALPABLE, NON-RETIFORM AND ≤4 MM IN DIAMETER

Pathophysiology: hemostatically relevant thrombocytopenia (<50,000/mm^3, usually <20,000mm^3)

Major etiologies*

1. Idiopathic thrombocytopenic purpura
2. Thrombotic thrombocytopenic purpura
3. Disseminated intravascular coagulation
4. Other acquired thrombocytopenias, including drug-related
    a. Peripheral destruction (e.g. quinine, quinidine)
    b. Decreased production, idiosyncratic or dose-related (e.g. chemotherapy)
    c. Bone marrow infiltration, fibrosis or failure

Pathophysiology: abnormal platelet function

Major etiologies*

1. Congenital or hereditary platelet function defects
2. Acquired platelet function defects
    a. Aspirin, NSAIDs
    b. Renal insufficiency
    c. Monoclonal gammopathy
3. Thrombocytosis in myeloproliferative disease (often >1 000 000/mm^3)

Pathophysiology: non-platelet etiologies
Major etiologies* 1. Spiking elevations of intravascular venous pressure (Valsalva maneuver-like, e.g. repetitive vomiting, childbirth, paroxysmal coughing, seizure) 2. Fixed increased pressure (e.g. stasis, ligatures) 3. Trauma (often linear) 4. Perifollicular (vitamin C deficiency) **Pathophysiology: mildly inflammatory conditions** 1. Chronic pigmented purpura 2. Hypergammaglobulinemic purpura of Waldenström

TABLE 2.

DIFFERENTIAL DIAGNOSIS FOR MACULAR ECCHYMOSES – NON-PALPABLE AND NON-RETIFORM.

Ecchymoses (≥1 cm in diameter)

A. Pathophysiology: procoagulant defect plus minor trauma*

1. Anticoagulant use
2. Hepatic insufficiency with poor procoagulant synthesis
3. Vitamin K deficiency
4. Disseminated intravascular coagulation (some)

B. Pathophysiology: poor dermal support of vessels plus minor trauma*

1. Actinic (solar, senile) purpura
2. Corticosteroid therapy, topical or systemic
3. Vitamin C deficiency (scurvy)
4. Systemic amyloidosis (light chain-related, some familial types)
5. Ehlers-Danlos syndrome (primarily type IV)

C. Pathophysiology: other causes plus minor trauma*

1. Hypergammaglobulinemic purpura of Waldenström
2. Platelet function defects, including von Willebrand disease, medications, metabolic diseases
3 Acquired or congenital thrombocytopenia

TABLE 3.

INFLAMMATORY PURPURA: LESIONS WITH PROMINENT EARLY ERYTHEMA

Classic palpable purpura (round lesions, port-wine colored, partially blanchable)

I. Pathophysiology: leukocytoclastic vasculitis due to immune complex disease

- A. Small vessels only
  1. Idiopathic, infection or drug-associated IgG or IgM complexes
  2. Idiopathic IgA complexes (HSP), or IgA associated with drugs, infection
  3. Hypergammaglobulinemic purpura of Waldenström
  4. Urticarial vasculitis: often minimally purpuric
  5. Pustular vasculitis (e.g. bowel bypass syndrome)
- B. Small and medium-sized (macroscopic) vessels may be involved
  1. Mixed cryoglobulinemia
  2. Rheumatic vasculitides (LE, RA)

II. Pathophysiology: pauci-immune leukocytoclastic vasculitis

- A. ANCA-associated
  1. Wegener's granulomatosis
  2. Microscopic polyangiitis
  3. Churg–Strauss allergic granulomatosis (rarely)

B. Other

1. Erythema elevatum diutinum
2. Sweet's syndrome (vasculitis unusual)

III. Pathophysiology: not leukocytoclastic

A. Small vessels only

1. Erythema multiforme
2. Pityriasis lichenoides et varioliformis acuta (PLEVA)
3. Chronic pigmented purpura
4. Hypergammaglobulinemic purpura of Waldenström

Classic target lesion (usually erythema multiforme or Stevens Johnson syndrome)

TABLE 4.

DIFFERENTIAL DIAGNOSIS OF NON-INFLAMMATORY RETIFORM PURPURA (MINIMAL TO NO ERYTHEMA IN EARLY LESIONS)

Pathophysiology: occlusion probably due to microvascular platelet plugs

Major etiologies*

1. Heparin necrosis
2. Myeloproliferative disease with thrombocytosis
3. Paroxysmal nocturnal hemoglobinuria
4. Thrombotic thrombocytopenic purpura (though platelet plugs form primarily in visceral vessels, and skin hemorrhage is due more often to thrombocytopenia)

Pathophysiology: cold –related gelling or agglutination
Major etiologies* 1. Cryoglobulinemia, monoclonal (early lesions of mixed cryoglobulinemia are often inflammatory and leukocytoclastic due to immune complex deposition) 2. Cryofibrinogenemia (though most cryofibrinogens are incidental findings in hospitalized patients) 3. Cold agglutinins (rarely cause occlusion; usually cause hemolysis or are asymptomatic)
Pathophysiology:occlusion due to organisms growing in vessels
Major etiologies* 1. Vessel-invasive fungi (mucormycosis, *Aspergillus*, and others, usually in immunocompromised patients) 2. Ecthyma gangrenosum (often *Pseudomonas* or other Gram-negative bacilli proliferating in adventitia of subcutaneous arterioles) 3. Disseminated strongyloidiasis 4. Lucio phenomenon in leprosy

Pathophysiology: systemic alteration in control of coagulation
Major etiologies* 1. Protein C- and S-related a. Homozygous protein C or protein S deficiency b. Acquired protein C deficiency (some patients with sepsis-associated DIC) c. Coumadin necrosis (protein C dysfunction) d. Post-infectious purpura fulminans 2. Antiphospholipid antibody, lupus anticoagulant **Pathophysiology: vascular coagulopathy**
1. Atrophie blanche or livedoid vasculopathy (may also have a systemic prothrombotic component)
2. Malignant atrophic papulosis (Degos' disease). Antiphospholipid antibodys syndrome can mimic this disease
Pathophysiology: embolization or crystal deposition
Major etiologies* 1. Cholesterol emboli 2. Oxalate crystal deposition (rare)
3. Marantic endocarditis, atrial myxoma, hypereosinophilic syndrome: all rare
Pathophysiology: reticulocyte, red blood cell occlusion
High reticulocyte states 1. Sickle cell disease; occlusion worsened by red cell sickling 2. Other severe hemolytic anemias

Pathophysiology:uncertain
Cutaneous 1. Cutaneous calciphylaxis

TABLE 5.

DIFFERENTIAL DIAGNOSIS OF MIXED RETIFORM AND INFLAMMATORY MORPHOLOGY

Vasculitis-Leukocytoclastic

A. Primarily dermal vessel

1. IgA vasculitis

B. Dermal and subcutaneous vessels usually involved

1. Mixed cryoglobulinemia
2. Rheumatic vasculitides
3. Benign cutaneous polyarteritis nodosa
4. Microscopic polyangiitis
5. Wegener's granulomatosis
6. Churg–Strauss allergic granulomatosis

C. Other

1. Septic vasculitis

Vasculitis/Vasculopathy-Non-Leukocytoclastic

A. Livedoid vasculopathy

Non-vasculitic

A. Chilblains (pernio)

B. Pyoderma gangrenosum