Key Location(s): Right lower extremity **CASE 1**

Presented by Hal Weitzbuch, MS, MD; Anjeli K. Isaac, MD; and George Engel, MD

History of Present Illness

61 year old Pakistani man presented with a mass on his right shin that he believed was present for approximately a year and a half. He denied any pain, pruritus, or associated symptoms. There was no discharge from the lesion. However, he did note that there was more rapid growth over the previous six months. He has no previous history of skin disease or cancer and denied trauma at the site.

Past Medical History

Carotid artery disease, hypertension, hyperlipidemia, rheumatoid arthritis

Medications/Allergies

Metoprolol, rosuvastatin, aspirin, methotrexate, clopidogrel
NKDA

Review of Systems

Denied fevers, chills, productive cough, shortness of breath, nausea, vomiting, weight changes, urinary urgency, dysuria, hematuria, and headaches

Physical Exam

Lymph: No cervical, occipital, supraclavicular, axillary, inguinal, or popliteal lymphadenopathy

Skin: Right proximal tibial tuberosity with 4.5cm x 5cm nontender, non-pulsatile, mobile tumor with superficial, central, fluid-like, compressible component; no transillumination

Histopathology

RIGHT TIBIA, EXCISION:
Skin and subcutaneous tissue with a proliferating pilar (trichilemmal) tumor and a cavernous hemangioma; negative margins. The specimen reveals a well circumscribed tumor surrounded by fibrous stroma. The tumor itself is composed of variably sized lobules of squamous epithelium. There is trichilemmal keratinization noted and the occasional presence of squamous eddies. Additionally, focal areas display nuclear atypia and occasional mitotic figures. Infiltration of the squamous lobules into the surrounding interstitial tissue is not identified. Adjacent to the tumor there are dilated vessels consistent with a cavernous hemangioma. Although focal areas of cellular atypia and occasional mitoses are noted, the focal nature of these findings favors a proliferating pilar tumor over a malignant proliferating pilar tumor.

Radiology

Right tibia/fibula radiograph: subcutaneous soft tissue mass located in the anterior proximal shin with punctate calcifications and areas of low densities, just below the anterior tibial tuberosity; there is no underlying bone involvement

Diagnosis

Proliferating trichilemmal cyst with overlying hemangioma

Treatment and Course

At the first visit, the patient consented to a biopsy; however, when aspirating during local anesthetic injection, 15cc of blood was obtained. The patient was then referred to surgery-oncology and was then sent for a CT scan of his right shin. However, upon contacting the patient at a later time, it was found that he was lost to follow-up and never received his radiologic imaging. His CT scan was rescheduled and an incisional biopsy was eventually performed. After preliminary histopathology results were not conclusive for a proliferating trichilemmal cyst, he was scheduled for surgical excision with margins by the dermatologic surgical service. He underwent excision and reconstruction with multiple flaps given the difficult location of the lesion, and he is recovering with no signs of recurrence.

Discussion

Proliferating trichilemmal cysts (PTC) are usually found on the scalp of older women. They vary in size but may grow up to 25 centimeters in diameter and are almost always found as a solitary lesion. Other locations where these cysts have been reported to grow are the face, trunk, scapula, thorax, leg, groin, forearm, and in the lumbar area. As in our patient, there has been another case of a PTC documented on the shin. PTCs have also developed within a nevus sebaceous and an epidermal nevus. However, this is the first case to our knowledge of a PTC associated with a cavernous hemangioma.

PTCs have been called many other names including proliferating trichilemmal tumor, pilar tumor, subepidermal acanthoma, invasive hair matrix tumor of the scalp, giant hair matrix tumor, hydatidiform keratinous cyst, trichochlamydocarcinoma, proliferating follicular cystic neoplasm, and proliferating trichilemmal cystic squamous cell carcinoma (PTCSCC). As evidenced by the variety of names used, there has been some disagreement with regard to the diagnosis of this entity. Probably the most troubling controversy regarding PTCs is the questionable potential for malignancy that the lesion might possess. Throughout the literature, there are numerous articles describing malignant and metastatic masses that were diagnosed in conjunction with a PTC. For example, some authors describe PTCs that transformed into SCCs, while others describe PTCs that coexist with malignant trichilemmal cysts. On the other hand, some, including Mones and Ackerman in particular, counter that these lesions are malignant from the outset and use PTCSCC to describe them.

Histologically, some of the atypical features that might designate the lesion as malignant are abnormal mitoses, high mitotic index, marked cellular pleomorphism, infiltrating margins, and necrosis. Several authors have performed molecular studies on these lesions to determine malignancy. Jaworski, Sleater, and Hashimoto all found abnormal ploidy, and Takata noted the same p53 mutation in anaplastic as well as more typical areas of a PTC. However, while many common features of malignancy are present using molecular studies, few cases reported actually metastasized. Furthermore, the well-circumscribed pattern, smooth borders, and lack of a precursor epidermal lesion favor a benign classification.

It is important to note that cases have been described with very atypical histopathological features and large sizes which did not recur after resection while other cases that showed minimal atypia and were quite small have led to recurrence, metastasis, and ultimately death. According to Mones and Ackerman, the only benign form of a PTC is a "proliferating trichilemmal cystic acanthoma". Based on these findings it seems that the most beneficial treatment is to resect all PTCs with clear margins and to follow-up all cases postoperatively with skin and lymph node exams regularly, with a low

threshold for re-excision. As the histological appearance may not correlate with the clinical behavior, it is still quite difficult to determine which lesions might benefit from chemotherapy, radiotherapy, or nodal dissection. Per Holmes, "[the] chance for metastasis may not be very great but the possibility does exist."

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Key location(s): Forehead, left hand **CASE 4**

Presented by Julia M. Kasprzak, MD and Warren Piette, MD

History of Present Illness

35 year-old homosexual male with HIV/AIDS, last CD4 count 137, presented with 1 month of intermittent fevers, right throat mass with localized neck pain, odynophagia (10/10 intensity), and neck pain that was worse with movement. Two weeks prior to presentation, he noted tender skin lesions on scalp, arms, hands, chest, and legs.

Past Medical History

HIV/AIDS (last CD4 137, VL 86,941); 3 months prior to admission: RPR negative
History of Mononucleosis
History of primary varicella in childhood
Last PPD negative (unknown date)
Hepatitis A, hepatitis B, influenza vaccines (2009); Pneumococcal vaccine (2010)

Medications/Allergies

Atazanavir 300 mg PO daily
Ritonavir 100 mg PO daily
Emtricitabine/tenofovir (Truvada) 200/300 mg PO daily
TMP/SMX 160/800 mg PO q48H
Methocarbamol 1500 mg PO q8H
Ibuprofen 800 mg PO q8H prn pain
Tramadol 50 mg PO daily to TID prn pain
NKDA

Social History

Smokes tobacco, occasional alcohol use, regular marijuana use; lives alone, no shelters, never incarcerated, no recent travel, no pets

Review of Systems

Positive for generalized intermittent headaches; 18 pound weight loss over 1 month

Physical Exam

Vitals: 98.7, 61, 135/68, 18, 97% RA
HEENT: Right tonsil enlarged, 5mm stellate purple discoloration overlying, no erosions or exudate
Neck: Right neck tender to palpation, no masses
Lymph: Bilateral cervical and retroauricular lymphadenopathy, no axillary nodes
Skin: Dorsum of hands, palms, arms, back, chest, face, forehead and scalp with red papules with overlying scale, some with distinct collarette of scale; back and chest with scattered folliculocentric pustules; scalp with papulonodules, tender to palpation; beard with confluent scale

Laboratory Data

The following initial labs were abnormal:

Albumin	3.7 g/dL	[3.8 – 5.2 g/dL]
Total Bilirubin	1.6 mg/dL	[0.2 – 1.2 mg/dL]
Hemoglobin	10.7 g/dL	[11.7 – 14.9 g/dL]
RPR	1:8	
Passive Particle Agglutination	REACTIVE	

The following labs were negative:
Histoplasma urine antigen, cryptococcal blood antigen, DFA VZV, DFA HSV 1/2, blood cultures (4/4), stool culture, respiratory AFB (x2), urine culture, throat culture, wound culture forehead negative for bacteria and herpes, hepatitis B and C

Histopathology
LEFT DORSUM HAND AND CENTRAL VERTEX SCALP, PUNCH BIOPSIES:
Ulcerated skin with superficial and mid dermal acute neutrophilic and chronic lymphoplasmacytic inflammation; AFB, GMS, PAS negative.

Radiology
CT Neck with and without contrast: Bilateral tonsillar enlargement, right greater than left; edema of right pyriform sinus; 1cm abnormal fluid collection lateral and posterior to right tonsil consistent with abscess; 5mm collection in enlarged right cervical lymph node consistent with lymphadenitis; Bilateral small caliber cervical lymph nodes

Treatment and Course
The patient was admitted to the hospital after being seen in the ED. He was initially started on fluconazole for possible esophageal candidiasis and placed in respiratory isolation for possible disseminated HSV/VZV. He was given morphine for pain. ENT performed a parapharyngeal needle aspiration, but no fluid returned. In order to rule out lymphoma, ENT planned for right cervical lymph node biopsy. On hospital day #3, the RPR titer returned at 1:8 and the PPA was reactive. The patient called a few of his partners, and one reported a "recent rash". The lymph node biopsy was cancelled. The patient was treated with one dose of benzathine penicillin G 2.4 MU intramuscularly and discharged the next day. Two weeks later, he was seen at the HIV clinic. He felt better at that time and was reportedly taking all of his HIV medicines. His RPR was re-checked at that visit and returned at 1:32.

Diagnosis
Secondary syphilis with syphilitic angina (tonsillitis)

Discussion
It is estimated that up to 22% of secondary syphilis cases have oropharyngeal involvement, which can occur in the absence a generalized rash. Tonsillar involvement is a rare, but reported, manifestation of the disease. Patients present with tender regional lymphadenopathy and ulcerations on the tonsil; bilateral and unilateral involvement of the tonsil has been reported. Unilateral disease such as in this patient can be worrisome for other disease processes such as squamous cell carcinoma, lymphoma or cervical lymphadenitis. Histologic examination of tonsillar tissue can help rule out malignancy and in syphilis it will show plasma cell and lymphocytic perivascular infiltrate with reactive hyperplasia and may show spirochetes identified by tissue stain. It was not performed in this case since the patient improved after receiving benzathine penicillin G.

This case illustrates the importance of recognition of oropharyngeal involvement in syphilis to aid in the diagnosis and treatment of this disease. It is especially important to consider secondary syphilis of the tonsil in high-risk populations including those with HIV. Co-infection with HIV and syphilis is very common and oropharyngeal involvement in syphilis is increasing due to greater incidence of oral sex practices. All patients with syphilis should be tested for HIV and all patients with HIV should be tested for latent syphilis. The treatment of choice is intramuscular benzathine penicillin G 2.4 MU and it is

recommended that serological tests are repeated for at least one year after treatment to ensure eradication.

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Key Location(s): Fingers **CASE 7**

Presented by Morayo Adisa, MD and Warren Piette, MD

History of Present Illness:
58 year old woman admitted for evaluation of restrictive cardiomyopathy was consulted for an abdominal fat pad biopsy. The patient had multiple past hospitalizations for CHF exacerbation. She reported a history of increased easy bruising over the last 2 years. She reported that simple actions, such as rubbing her eyes, made her lids very bruised for days and she sustained significant bruising of her fingers from opening jars or bottles.

Past Medical/Surgical History
Diastolic heart failure with restrictive pattern, history of carpal tunnel release surgery, gout, arthritis

Medications/Allergies
Bumetanide, carvedilol, metolazone, aldactone, allopurinol
NKDA

Review of Systems
She denied any history of GI bleeds or neuropathy

Physical Exam
Skin: Bilateral distal phalangeal tips with bulbous skin changes with wrinkling and yellowish to orange papules and plaques on the lateral aspects

Laboratory Data
The following were abnormal:

PTT	36.5 secs	[23.6 – 36.4 secs]
GFR calculated	78 mL/min/1.73m^2	[≥ 90 mL/min/1.73m^2]
Total protein	6.0 g/dL	[6.4 – 8.3 g/dL]
Albumin	3.4 g/dL	[3.8 – 5.2 g/dL]
LDH	254 U/L	[130 – 240 U/L]
Urine protein	10	[Negative]
Immunoglobulin M	34 mg/dL	[77 – 220 mg/dL]
Kappa/Lambda ratio	1.34 mg/dL	[1.41 – 2.83 mg/dL]
Serum protein electrophoresis	Low gamma-globulin level	
Serum immunofixation electrophoresis	Lambda light chain identified	
Urine Immunofixation electrophoresis	Lambda light chain identified	

The following were normal or negative: CBC with the exception of the neutrophil and lymphocyte counts, basic metabolic panel, prothrombin time, urinalysis except as indicated above, immunoglobulin G, and immunoglobulin A

Histopathology
ABDOMINAL FAT BIOPSY, INCISIONAL BIOSY:
Adipose tissue shows slight expansion of fat cell walls. The blood vessel walls have deposits of pale pink homogenous material consistent with amyloid. Congo red stain for amyloid is positive.

RIGHT FOURTH LATERAL DIGIT, PUNCH BIOPSY:
There are dilated vessels in the upper half of the dermis. Amyloid, crystal violet and elastin stains are negative.

Radiology
Cardiac MRI/MRA: Diffuse concentric hypertrophy of the mid and basal portions of the walls of the left ventricle, including the interventricular septum (IVS); extensive diffuse hyperenhancement of the walls of left and right ventricles and IVS, with relative sparing of the endocardial and epicardial portions; imaging findings suggest myocardial infiltrating process, including amyloidosis among other etiologies; findings reflect restrictive physiology; EF = 74%; normal pericardial thickness

Diagnosis
Primary systemic amyloidosis with cutaneous manifestations

Treatment and Course
The patient was under the care of the hematology/oncology and cardiology services, but she has not been seen in follow-up since her discharge from the hospital.

Discussion
Amyloidoses are characterized by the extracellular deposition of amyloid proteins which are altered autologous protein fibrils with anti-parallel β-sheet conformation. The amyloidoses may be classified into hereditary, localized and systemic forms. The systemic form can be further subclassified into primary and secondary forms. Primary systemic amyloidosis is the most common form and is almost always associated with a plasma cell dyscrasia with 15 – 20% of patients reported to have concomitant multiple myeloma. It is characterized by the deposition of amyloid light chain (AL) protein in the skin (within the dermis), kidneys, heart, GI tract, skeletal and smooth muscles, carpal ligaments and nervous system. Kidneys being the most common organ affected. The infiltration of the organs leads to the impaired function of the affected organs and the extent of symptoms corresponding to the degree of organ infiltration.

Cutaneous manifestations of primary systemic amyloidosis vary and can be seen in approximately 29 – 40% of cases. Pruritus (due to hepatic infiltration), petechiae, ecchymoses, waxy papules, morphea–like plaques, alopecia, nail dystrophy (onycholysis, brittle nails, nail banding), macroglossia and pigmentary changes are some of the more commonly reported cutaneous clinical manifestation of this entity. The petechiae and ecchymosis are postulated to be due to amyloid protein deposition within the cutaneous blood vessels causing vascular fragility. This explains the easy bruisability experienced by our patient. The skin findings may be one of the first presenting signs of this multisystemic disease, in which case, could provide an opportunity for early diagnosis and possibly early therapeutic intervention. In other cases, cutaneous manifestations may develop after other organ involvement. Unfortunately, our patient does not recall if the cutaneous lesions preceded her other systemic symptoms or not.

Tissue biopsy with use of various stains such as crystal violet, PAS, thioflavin T fluorescence and Congo red to demonstrate the presence of the amyloid deposits within the tissue provide definitive diagnosis of amyloidosis. Fine needle fat pad biopsy and rectal biopsy are the biopsies of choice with a positive result in 80 – 90% of cases. Skin biopsy of involved or uninvolved areas may be useful in providing a definitive diagnosis. Congo

red and crystal violet stains of one of our patient's finger lesion did not support a diagnosis of cutaneous amyloidosis.

There have been case reports of similar appearing finger tip changes reported in about 7 patients in the literature with multiple myeloma and acral localized cutis laxa. The skin lesions in these patients appeared as asymptomatic, soft redundant, loose and wrinkled skin particularly on the fingertips. Tissue biopsies in these cases showed the presence of amyloid material around blood vessels and within the dermis. The amyloid material in each of these cases was highlighted with Congo red and crystal violet stains. In addition, the same tissue samples showed diminution of elastic fibers in the dermis and focal clumping of the elastic fibers around the dermal vessels and appendages supporting a diagnosis of cutis laxa. However, despite the strong clinical resemblance of our patient's distal finger tips to those reported in the literature to be associated with multiple myeloma associated acral localized acquired cutis laxa, the tissue investigations failed to reveal the presence of amyloid deposition and qualitative and quantitative alteration in the elastic fibers of the distal fingertips. Instead, dilated lymphatic channels representing either lymphangioma or lymphedema. This is to my knowledge, the first case of primary systemic amyloidosis associated distal phalangeal lymphangioma. Unfortunately our patient has been lost to follow-up, so further clinical and tissue characterization of this entity cannot be undertaken.

The prognosis for primary systemic amyloidosis is poor with mortality attributed to cardiac or renal end organ failure. Congestive heart failure in particular portends a poor prognosis.

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Key Location(s): Left lower extremity **CASE 10**

Presented by Rachel Pritzker, MD and Warren Piette, MD

History of Present Illness

55 year old female presented with a history of an ulcerating leg lesion for 3 years. She stated that it began as a small "ingrown hair" red tender bump which slowly enlarged and subsequently ulcerated. The lesion was very painful, but otherwise asymptomatic. The patient recalled no trauma to the area, but eventually admitted to injecting cocaine into the lesion every two weeks. She had been seen by an infectious disease physician at an outside hospital and given minocycline, trimethoprim/sulfamethoxazole, and mupirocin ointment intermittently for 2 years without effect. The longest duration she was on one of these medicines was over 2 months. She has no history of blood clots.

Past Medical History

None

Medications/Allergies

Mupirocin ointment, minocycline, trimethoprim/sulfamethoxazole
NKDA

Social History

Cocaine abuse; no alcohol or other illicit drug use; no recent travel

Physical Exam

Skin: Left upper leg with several deep round ulcerations with fibrinous, purulent, and necrotic material at the base; surrounding ulcerations there are several linear firm hyperpigmented scars; there is a mild violaceous and hyperpigmented non-contiguous livedoid pattern in the background of the involved area; right upper leg with hyperpigmented patches and no ulcerations; arms and legs with many scars overlying veins in linear distribution

Laboratory Data

The following were abnormal:

Hepatitis C Antibody	Positive	[Negative]
Beta 2 Microglobulin	2.40 mg/L	[0.04 – 2.30 mg/L]

The following were normal: CMP, CBC, PT, PTT, pANCA, cANCA, β2-glycoprotein, anti-cardiolipin antibody, lupus anticoagulant, ANA, Hepatitis B panel

Histopathology

LEFT ANTERIOR THIGH, PUNCH BIOPSY:
Skin with acanthosis and dermal fibrosis. No vascular or granulomatous changes found. No polarizable foreign material identified. GMS, PAS, and AFB stains negative.

Microbiology

LEFT ANTERIOR THIGH, TISSUE CULTURE:
Mycobacterium chelonae, identification by innogenetics line probe assay; no antibiotic sensitivity done; AFB culture no growth after 6 weeks; no fungus isolated
BACTERIAL WOUND CULTURE:
Streptococcus viridans

Diagnosis

Non-healing ulceration from skin-popping illicit drugs with superimposed *Mycobacterium chelonae* infection

Treatment and Course

The patient was advised to stop injecting illicit drugs in the area and DuoDERM® was placed on the area. She noted significant improvement with the occlusive dressing. When the culture came back positive for *Mycobacterium chelonae*, she was seen by our infectious disease department. She was started on clarithromycin 500mg PO BID and minocycline 100mg PO BID (as she was unable to tolerate doxycycline previously). After 3 weeks on this treatment, she self discontinued due to a constant dysgeusia. She was then switched to azithromycin 600mg PO daily with continuation of minocycline. Overall, within this four month time course, there has been 50% improvement in the initial ulcerations.

Discussion

Mycobacterium chelonae is member of the group of nontuberculous rapid growing mycobacterium including *Mycobacterium fortuitum* and *Mycobacterium abscessus*. It exists as a saprophyte and found ubiquitously in water, soil, aerosols, and animals. Tap water is considered the major reservoir for most human pathogens and the source for many outbreak scenarios of mycobacterial skin disease. Recent scenarios of outbreaks of rapidly growing mycobacterial infection in immunocompetent hosts include infection after tattoo placement, liposuction, and nail salon services. Infections typically occur after trauma or surgery. Isolated infections have been described in cases of Mohs surgery, punch biopsy, acupuncture, mesotherapy, cosmetic injections, laser resurfacing, breast reconstruction, insulin injections, and body piercing.

Clinical signs of mycobacterial infection are classically painful red nodules that ulcerate and drain, but folliculitis, furuncles, abscess, cellulitis, nodules, draining lesion, ulcerations have also been described. Moreover, in any therapy resistant nodule or ulceration, mycobacterial infection should be suspected.

The detection of atypical mycobacterium is frequently difficult. On histopathology, a variety of patterns can be seen from suppurative granulomas to diffuse, nonspecific, chronic inflammatory changes. In many cases the traditional Ziehl-Neelsen histochemical stain has low sensitivity (less than 15% positivity) due to large bacterial load needed for detection and bacterium unable to survive processing techniques. Tissue culture for atypical mycobacterium is still the gold standard for cutaneous lesions. It is important to inform the lab that atypical mycobacterium is considered, as culture should be done on special broth and solid agar medium at lower temperatures than other bacterium. *M. chelonae* and other rapid growing mycobacterium usually grow within 7 days. Further, newer techniques of rapid identification and speciation on tissue specimens include 16S ribosomal DNA sequencing and polymerase chain reaction (PCR)-restriction length polymorphism analysis (PRA). Our patient's diagnosis was made by this PCR technique on the cultured tissue.

There are no standardized treatment recommendations for treatment of *M. chelonae* as there are no large, randomized controlled trials. It is recommended to obtain susceptibility testing for all nontuberculous mycobacterium species to guide therapy. First line treatment for localized rapid growing mycobacterial disease is usually clarithromycin 500mg PO BID for six or more months. There have been several cases of

clarithromycin resistant strains and many recommend using clarithromycin in combination with a second antibiotic to reduce the chance for this resistance to develop. Other antibiotics used to treat *M. chelonae* include azithromycin, amikacin, tobramycin, linezolid, ciprofloxacin, doxycycline and sulfonamides. Side effects from clarithromycin are most commonly nonspecific gastrointestinal symptoms and include alteration in taste and smell (inducing a metallic taste) for the duration of treatment. Other side effects include jaundice, cirrhosis, renal failure, dizziness, headaches, delirium, ototoxicity, and a false positive urine drug screen for cocaine.

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