

## Internal Medicine Dermatology

### P1600

#### **Sunitinib-induced hand-foot syndrome: A new, distinct form of hand-foot syndrome**

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The tyrosine kinase inhibitor (TKI) sunitinib is approved for the treatment of metastatic renal cell carcinoma and GIST refractory to or recurrent after imatinib therapy. However, its use is limited by a variety of adverse events, most notably the hand-foot syndrome. We propose that this distinct form of hand-foot syndrome differs from the classic hand-foot syndrome caused by anti-metabolite chemotherapy. A 67 year old Vietnamese male began to have painful lesions on the soles, palms, and fingers 2 weeks after initiating sunitinib 50 mg orally daily. He had a history of metastatic GIST status post partial gastrectomy, radiation therapy, and imatinib therapy. He denied symptoms. On his soles the patient had thick, indurated, well-demarcated 0.5 to 3 cm yellow to flesh-colored tender plaques with erythematous halos concentrated over pressure points (Figure 1). The hand lesions were beefy-red, partially eroded, and crusted (Figure 2). The pain and rash improved rapidly in 1 week with discontinuation of sunitinib but recurred when it was restarted at 37.5 mg daily. Finally, the patient tolerated sunitinib 25 mg/day without recurrence of the rash. While relatively common in clinical trials, the HFS caused by sunitinib has not been well-characterized. Our case suggests that the HFS induced by sunitinib and a similar TKI, sorafenib, may be distinct from the classic HFS (also called palmo-plantar erythrodysesthesia) caused by anthracycline and anti-metabolite chemotherapeutic medications. In classic chemotherapy-induced HFT, initial diffuse erythema of the palms and soles can progress to swelling, tingling, dysesthesia, desquamation, or bullae over pressure points (Figure 3). The plantar lesions of TKI-associated HFS are unique in that instead of being diffuse, they tend to be well-demarcated, localized over pressure areas, and have yellow painful hyperkeratosis surrounded by a rim of erythema. On the hands, the lesions of TKI-induced HFS are also well-demarcated and commonly form bullae that erode and heal rather quickly.

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*Commercial Support: None Identified*

## Internal Medicine Dermatology

**P1601**

### **Systemic amyloidosis presenting with widespread hemorrhagic bullae: Masquerading as purpura fulminans**

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**Background:** Primary systemic amyloidosis (PSA), associated with multiple myeloma (MM), presents with diverse clinical manifestations secondary to infiltration of numerous organ systems by immunoglobulin light chain derived amyloid. The skin is involved approximately 25-40% of the time in PSA and a large variety of lesions occur. Common lesions include skin colored papules on the head, neck, or anogenital area and waxy, translucent, or purpuric lesions that resemble nodular amyloidosis. Infiltration of blood vessel walls results in periorbital and pinch purpura secondary to increased pressure or mild trauma. We describe a patient who presented with widespread hemorrhagic bullae.

**Observation:** A 70 year-old woman with history of diabetes mellitus II, chronic renal insufficiency, hypertension, and eczema treated with systemic corticosteroids had noticed increased fragility of her skin with easy bruising and tearing for two years. She presented with shortness of air, fatigue and scattered purpura. She rapidly deteriorated and required intubation. During resuscitation she developed widespread hemorrhagic bullae and erosions. Work-up revealed acute worsening of renal failure, anemia, thrombocytopenia, and decreased cardiac function. She was also found to have pneumonia, bacteremia, and legionellosis. Initial clinical differential diagnosis included purpura fulminans, vasculitis, or vasculopathy. Skin biopsies showed intradermal blisters with extravasated erythrocytes and deposition of amorphous material around adnexal structures and within blood vessel walls. Congo red stain was negative, but amyloid P immunoperoxidase was positive. Immunofixation electrophoresis and urine immunofixation revealed monoclonal IgA lambda gammopathy and bone marrow biopsy was consistent with MM. The patient died of a stroke shortly after initiation of chemotherapy.

**Comment:** Because of the acute development of widespread hemorrhagic bullae in addition to multiple organ failure, this case of bullous amyloidosis was initially felt to be purpura fulminans. Multiple myeloma was not considered until amyloid was seen in the skin biopsies. Although cutaneous involvement occurs frequently with PSA it is rare to develop hemorrhagic bullae. We present this interesting case to increase the awareness of this rare presentation of MM related amyloidosis and to illustrate that skin findings may provide the initial impetus to search for underlying systemic disease.

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## Internal Medicine Dermatology

**P1602**

### **Paraneoplastic dermatoses associated with gynecologic and breast malignancies**

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Paraneoplastic dermatoses are a heterogeneous group of skin disorders that manifest an underlying internal malignancy. Early recognition of these cutaneous hallmarks offers an opportunity for early diagnosis, treatment of the internal malignancy and monitoring for tumor recurrence. The nine most common paraneoplastic and metastatic cutaneous manifestations of malignancies found in women with gynecologic or breast disease are reviewed. A case presentation of multicentric reticulohistiocytosis associated with endometrial cancer is presented followed by a review of multicentric reticulohistiocytosis, dermatomyositis, malignant acanthosis nigricans, erythema gyratum repens, hypertrichosis lanuginosa acquisita, Sweet's syndrome, Paget's disease, extramammary paget's disease and Sister Mary Joseph's nodule.

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## Internal Medicine Dermatology

### P1603

#### **Leukemia cutis occurring as the initial manifestation of chemotherapy-associated monocytic leukemia**

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A 66 year old male presented to our institution with a recent, rapid onset of a diffuse nodular rash. The patient's past medical history was significant for small cell lung cancer, treated the year prior with lobectomy and four cycles of carboplatin/VP-16 chemotherapy. Approximately 6 weeks prior to the eruption of his rash, he developed DIC and was hospitalized. At that time, a visceral lesion was found on imaging and presumptive diagnosis of metastatic lung cancer was made. The patient was treated once again with chemotherapy. Approximately 3 weeks after his last cycle of chemotherapy, the patient noted development of red, raised nodules on his right upper flank and right temple.

The patient was transferred to our institution and examination revealed multiple, well-circumscribed flesh colored and red-purple nodules ranging between 3-8 mm in size, occasionally coalescing into plaques. Nodules were most prominent over the face, scalp, abdomen, and extremities. Several of the nodules had begun to ulcerate, particularly on the scalp and pretibial surfaces. Skin biopsies revealed dense sheets of atypical mononuclear cells throughout the deep dermis and subcutaneous adipose tissue. The epidermis was unremarkable, and a grenz zone was evident in the superficial dermis. The atypical cells expressed CD68, lysozyme, CD43, and CD4, but lacked expression of CD2, CD3, CD5, CD7, CD20, or myeloperoxidase. CD56 expression was indeterminate. The histopathologic findings were compatible with an extramedullary myeloid cell tumor with monocytic differentiation. Peripheral blood smear and bone marrow biopsy subsequently confirmed acute monocytic leukemia.

The patient was initially treated with chemotherapy. Shortly thereafter, the patient developed purpura fulminans, and flow cytometry revealed relapsed acute monocytic leukemia. The patient elected to return home with hospice care and passed away. Leukemia cutis characteristically has a poor prognosis, and leukemia associated with prior chemotherapy (as was the case with our patient) is characteristically difficult to treat. The rapid and extensive nature of this patient's skin eruption was the first clue to the diagnosis of acute monocytic leukemia in this patient, and skin biopsy was helpful in establishing the diagnosis.

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# Internal Medicine Dermatology

**P1604**

## **Localized neuropathic itch syndromes**

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Background: Localized itch of non-pruritoceptive origin is often neuropathic and may be referred to as neuropathic itch syndrome.

Objective: To describe the characteristics of neuropathic itch syndrome using nerve conduction studies.

Methods: Included in the study were 88 consecutive patients with anogenital pruritus (54), brachioradial pruritus (25) and scalp dysesthesia (9). Nerve conduction studies included measurements of distal sensory and motor latency, conduction velocity, and F-responses.

Results: A neuropathy was demonstrated in 29/36 patients (80.5%) with anogenital pruritus, 8/14 patients (57.1%) with brachioradial pruritus and 4/9 patients (44.4%) with scalp dysesthesia. Limitations: Nerve conduction studies were not available for all patients included in the study.

Conclusion: A considerable proportion of patients with brachioradial pruritus, anogenital pruritus and scalp dysesthesia have abnormal nerve conduction results suggesting a neuropathic origin. A neuropathic itch syndrome should be included in the differential diagnosis of localized chronic itch.

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## Internal Medicine Dermatology

**P1605**

### **Hemodialysis-associated steal syndrome: A case report**

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An arteriovenous fistula construction is needed to be performed in patients with chronic renal failure who should undergo long-term hemodialysis. However, a severe complication of a arteriovenous fistula, "hemodialysis -associated steal syndrome", can develop in 5% to 10% of the patients. It is caused by the arterial insufficiency occurring distal to the arteriovenous fistula due to the diversion of the blood flow into the fistula over the physiological degrees. Clinically, it can manifest with coolness, paresthesia, and absence of distal pulses, or more severe signs like cyanosis and gangrene. A 62-year-old diabetic man presented with a 1-month history of painful ulcers on his left hand fingers. He had a history of chronic renal failure, and had been undergoing hemodialysis therapy via left arm brachio-cephalic arteriovenous fistula for 3 years. Dermatologic examination revealed several round, crusted ulcers of 0.5-1 cm in diameter with a minimal surrounding erythema located on the second and third fingers of the left hand. The fingers were edematous, pale and cold, and the radial pulse diminished. Arterial Doppler USG examination proved the decreased distal arterial blood flow, thus a diagnosis of hemodialysis-associated steal syndrome was made. After the ligation of the arteriovenous access, a significant clinical improvement was achieved. Since it can result in severe morbidity including tissue or limb loss, a prompt diagnosis and correct management is mandatory in ischemic steal syndrome, which should always be kept in mind in the differential diagnosis of painful hand ulcers in a hemodialysis patient.

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