ALOPECIA

Female or male pattern	Telogen Effluvium	Alopecia areata	Tinea capitis	Scarring alopecia
Increased DHT	Warfarin, heparin, lithium, valproate, hypothyroidism, hyperparathyroidism, pituitary disease Stress Postpartum	Autoimmune: hypothyroidism pernicious anemia	Fungal Scales and hairs broken off 2 millimeters above the scalp	Trauma Infection: Syphilis HIV, TB, H Zoster Discoid Lupus Sarcoid
midline part	Thining; no patches	patchy	patchy	Variable pattern
minimal shedding; negative pull test	prominent shedding positive pull test	prominent shedding positive pull test	Prominent shedding Positive pull test	Broken hairs Negative pull test
minodxidil 2% BID Finasteride 1mg in men.	Drug avoidance Time	Treat underlying disorder Intralesional steroids if < 50%. Oral steroids for more than 50%.	Griseofulvin 1200 mg/day for 6 weeks. 300mg/day for 1 week every 3 weeks for a total of 3 weeks.	Treat underlying condition.

DRUG ERUPTIONS.on a spectrum:
Erythematous m-p eruption
To
Erythema Multiforme: target & iris lesions
To
Stevens-Johnson Syndrome: Oral, ocular, facial and trunk involvement. Discrete dark red macules, sometimes with necrotic center. Blister formation occurs. However, less than 10% of skin surface becomes detached. Mortality is 5%.
To
Toxic Epidermal Necrolysis: TEN. Same distribution as SJS. Severe blistering with detachment in sheets involving more than 30% of the area detaching. Mortality is 50%. **Rx is to recognize the drug relationship, discontinue it, hospitalize the patient, and Rx with IVIG.**

Differential Diagnosis of SJS and TEN:
Staph Scalded Skin Syndrome (caused by staph toxin), exfoliative erythroderma, paraneoplastic pemphigus, acute exanthematus pustulosis. Skin biopsies of SJS and TEN show full thickness epidermal necrosis, not seen in these other conditions.

Drug Eruptions: Work up is to 1) Obtain a CBC for eosinophilia, and 2) a skin biopsy for eosinophils in skin. This would confirm drug etiology.

Urticaria: Pruritic, raised hives. Hours or days post initiation of therapy. Antibiotics, radiocontrast. Cutaneous mast cell in superficial dermis.

Angioedema. Deeper dermis. ACE inhibitors. NSAIDs. ACE inhibitor reactions occur in .02% to 0.1% of new users.

Drug Exanthems: M-P eruptions. Antibiotics.

Erythroderma: Redness over 50% of body. 10% due to drugs, the remainder due to atopic dermatitis, psoriasis, and malignancy.

Hyersensitivity Vasculitis. Age 16+, use of drug in temporal relation to symptoms, palpable purpura, maculopapular rash, biopsy showing **neutrophils around an arteriole or**

venule.

Hypersensitivity Syndrome: Rash, fever, often hepatitis, arthralgias, hematologic abnormalities, and lymphadenopathy. **Sulfa drugs (e.g., Trimethoprim/Sulfamethoxazole) and Anti-epileptics (phenytoin, carbamazepine, phenobarb) and sulfas** are most common offenders.

Phototoxic drug eruptions: Warn the Patient !! These look like exaggerated sunburns and are most frequently caused by: **NSAIDs, quinolones, tetracyclines (remember doxycycline!!), amiodarone, & phenothiazines.**

Fixed drug eruptions: mouth, face, genitalia, acral areas. Causes: phenolphthalein (laxatives), tetracyclines, barbiturates, sulfonamides, NSAIDs, and salicylates.

DRUGS EXACERBATING PSORIASIS:
Lithium
Beta Blockers
Anti-malarials.
NSAIDS
Captopril
Withdrawal of Prednisone (used for another illness)

Sunblock should be applied about ½ hour before sun exposure.
It should be re-applied every two hours.
4 inch wide brim hat is best sun protection for face and neck.
The most important feature of clothing protection is the tightness of the weave.
A wet t shirt can magnify the sun exposure.

Erythema Nodosum correlations (from UpToDate)
Sarcoid >>> CXR
Inflammatory Bowel Disease
TB >>> PPD
Strep infections >>> Streptozyme & ASO titer.
OCP's & other drugs
Behcet'd disease (rare)
Other autoimmune or CTD

In all patients with urticaria, **avoid** Aspirin, NSAIDs, and Opiates. These exacerbate the urticaria via non-immunologic histamine release.

Rosacea is treated with oral tetracycline or metronidazole. Steroids will cause worsening of the Rosacea when it is withdrawn.

Dermatitis herpetiformis — Dermatitis herpetiformis is a condition characterized by pruritic papulovesicles over the external surface of the extremities and on the trunk. The diagnosis is confirmed histologically by the demonstration of granular IgA deposits along the subepidermal basement membrane. Similar to celiac disease, antibodies against tissue transglutaminase (anti-tTG) are elevated in patients with the disease. Compared to endomysial antibodies, anti-tTG antibodies had a sensitivity of 98 percent and specificity of 89 percent in a study involving 61 patients with dermatitis herpetiformis who were compared to 84 controls [87].

Dermatitis herpetiformis is common among patients with celiac disease. A population-based study in Finland that involved 147,000 people found that dermatitis herpetiformis was present in 24 percent of the 398 diagnosed patients with celiac disease (a celiac disease prevalence of 1:369) [88]. This estimate may underestimate the prevalence of celiac disease among patients with dermatitis herpetiformis since approximately 85 percent of patients with dermatitis herpetiformis have evidence of celiac disease on mucosal biopsy.
Dermatitis herpetiformis and celiac disease are associated with the same HLA-DQ alpha beta heterodimers, and dermatitis herpetiformis shares an association with other autoimmune conditions [89-91]. Although the celiac disease in patients with dermatitis herpetiformis is often

asymptomatic, the skin lesions in most patients respond to gluten withdrawal [92].

INFLAMMATORY FACIAL LESIONS

Rosacea	In 50+ age groups. Facial flushing from hot liquids or ETOH. Erythema, telangiectasias, papules, pustules,	Rhinopyema in men
Dermatophytosis or Tinea Faciei	Asymmetry, Erythematous, annular lesions.	KOH prep shows hyphae
Staph aureus	folliculitis	
Acne		
Allergic contact dermatitis	Pruritic. Papules. Vesicles. Clears in a few weeks.	
SLE	Similar to rosacea. Symmetric, macular erythema. Photosensitivity.	Systemic Sx's. Positive ANA
Discoid lupus erythematosis	discoid hyper and hypo pigmented plaques on scalp and sun exposed areas. PASTE: Plugging, Atrophy, Scaling, Telangiectasia, Erythema.	
Dermatomyositis	Heliotrope over eyelids	
Primary amyloid	Pinch purpura	
Seborrheic dermatitis	well demarcated confluent macular erythema with greesy scale. Scalp, eye-borws, chin, naso-labial creases and chest.	

RHEUMATOLOGY AND SKIN DISEASES

DISEASE	RHEUMATOLOGIC SITE	SKIN ABNORMALITIES
Psoriatic arthritis	Fingers (DIP joints), asymmetric oligoarthritis. Hyperuricemia.	Onychodystrophy, onycholysis, nail pitting, subungal keratosis, tophi
Gout	Gout of big toe	tophi, olecrenan bursa, infrapatellar bursa, achilles tendon, subcu tissue on extensor surface of forearm, helix of ear.
Dermatophyte infection	no joint disease (otherwise it resembles psoriatic arthritis)	sub ungual hyperkeratosis, thickening, onycholysis (DX: KOH prep on nail clippings or fungal cultures)
rheumatoid arthritis	PIP joints	rheumatic nodules (where ???), rheumatoid vasculitis, pyoderma gangrenosum.
Sarcoid	Inflamed joints: Swollen, warm, tender, & painful. Distribution: knees, ankles, elbows, wrists, small joints of hands.	Lupus pernio (violaceous indurated lesions with a predilection for nose, ears, lips, and face), skin plaques, papules, subcu nodules, and erythema nodosum. (No nail changes.)
Dermatomyositis (and internal malignancies)	Proximal Muscle Weakness	Periorbital: Heliotrope Rash. Knuckles: Red, scaly plaques (Goitren's papules)

Treatments for acne by type September 22, 2004

Acne Type	Treatment
Comedonal	Topical Tretinoin; Topical salicylic acid
Mild-moderate inflammatory	Topical benzyl peroxide & erythromycin
Moderate-severe inflammatory	Oral Tetracycline
Very severe intractable inflammatory	Oral isotretinoin (**get pregnancy test**); oral antibiotics

DIFFERENTIAL DIAGNOSIS OF A MALAR RASH:
SLE or Cutaneous SLE (has interface changes on biopsy)
Acne Rosacea (or just "rosacea", which is acneiform and erythematous)
Dermatomyositis

DRUGS & LUPUS IN DERMATOLOGY

LUPUS CONDITION	DRUG causing the induction
Cutaneous lupus erythematosus *	**HTZ; calcium channel blockers**
SLE (Serositis)	Hydralazine, minocycline, INH, procainamide.

*Biopsy shows an "interface dermatitis"; this is the best method of diagnosis.

ACNE ROSACEA is made worse by
*Hot beverages (cool liquids will quiet rosacea)
Alcohol
Caffeine
Spicy Foods
Sunlight

PRIMARY CARE MEDICINE — MKSAP XIII 12/18/03. **NON-FUNGAL**

DERMATOLOGIC CONDITIONS: ATOPIC DERMATITIS (ECZEMA) ET AL.

Entity	Sx's	Setting	Mechanism	RX
Atopic Dermatitis = Eczema	flexure areas. Papules: Tiny intensely pruritic; Lichenification. With Inc Temp and lymphadenopathy, can be life threatening: Exfoli-ative Erythroderma	house dust	Atopy = "Genetic predisposition to an immediate (Type I) hypersensitivity rx'n to antigens." S Aureus secretes super antigens that stimulate CYTOKINES	Topical steroids up to 4 months (this does NOT cause atrophy). Wet wraps to stop excoriation.
Numular or discoid eczema	Estensor surfaces;trunk, buttocks. Coin shaped.			
Allergic contact dermatitis (irritant contact dermatitis is a separate entity)	As with Eczema but not life threatening.	Poison ivy or oak, nickel, topical lidocaine, neomycin, soaps	absorption thru stratum corneum	sx'c. Steriods may be given topically or orally.
Hyperimmune gloublin E syndrome	As with Eczema	Recurrent resp tract infections; recurrent soft tissue abscesses from S. aureus	Markedly elevated IgE	??
Gluten sensitive enteropathy	Desquamative dermatitis with more pronounced scaling	diarrhea	auto-immune	??
Zinc deficiency	pruritic papules with lichenification	zinc deficiency from mal-absorption	immune abnormality	Zinc replacmeent

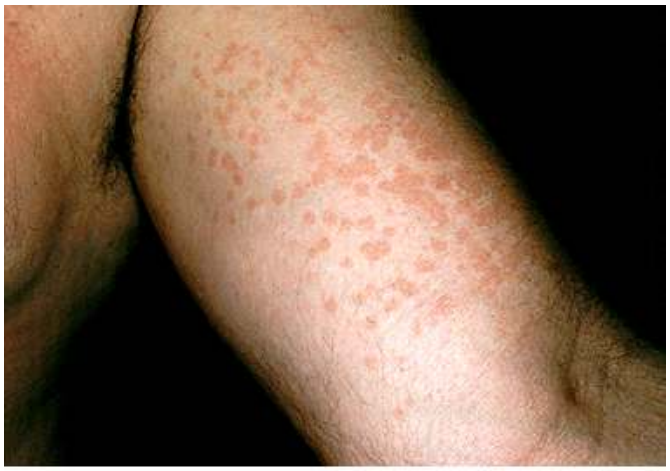
NON-FUNGAL DERMATOLOGIC CONDITIONS (CONT'D)

Entity	location/Sx's/ setting	Mechanism	RX	other
Comedonal acne	Face. Puberty, PCO, Cushings.	Propionibacterium acnes	benzoyl peroxide + topical ab'c (erythro, or clinda)	
Papular and Pustular acne	""	"". inflammatory response from fats of bacteria	Above plus Topical (1) retinoid related cpds: isotretinoin, tazarotene gel (short acting), adapalene or (2) salicylic a, or azelaic a. (for those intolerant to the retinoid cpds).	May need oral tetrac 500 bid, doxyc 100 bid, minoc 100 qd, erythro 1 gm bid, tmp-smz ds bid.
Sever pustular acne or nodulocystic acne			oral isotretinoin or minoc PLUS topical azelaic a. for 6 months; then azelaic a alone..	mino regimen has fewer side effects.
impetigo and furunculosis	pustular crusted grouped lesions	Staph A and, less frequently, grp A Strep	Cephalexin, erythro, dicloxacilllin orally or mupirocin (bactroban) ointment.	
Folliculitis	"hot tub folliculties", HIV	Staph A, Pseudomonas, candida	Topical Ab'cs and hot compresses. Eliminate nasal carriage with mucopiricin.	HIV has Staph A, or eosinophilic pustular lesions.
Psoriasis	Scalp, extensor surfaces, low back, intergluteal, behind ears. Discrete, raised plaques, white scale. (Unlike eczema psoriasis does not weep.)	Extra-cutaneous: Nail abnormalities(pittin g, hyperkeratosis). Arthritis, Sausage digits (Pencil in cup x-ray), sacroiilitis, HLA B27 +.	(cont.) Koebner: due to skin trauma. Auspitz Sgn: punctate bleeding on scale removal. Distinguish from atopic derm (lichenified but not raised or scaly)	Sunlight, topical steroids, tar. Sever: refer. Oral steroids, if withdrawn quickly, will cause a flare.
Guttate psoriasis	trunk and prox limbs: diffuse small red discrete papules.	(Cont.:) Distinguish from pityriasis rosea, drug eruptions,	(cont.) and 2ndary syphillis.	
Generalized pustular (von Zumbusch's psoriasis)	trunk: 2 - 3 mm pustules, limbs, palms, soles, NOT the face.	Systemic toxicity and onycholysis		
psoriatic erythroderma	ubiquitous. Less scaling.	PUVA therapy as a form of Koebner phenom.		MF, Sezary S,
Squamous cell ca	superficial, discrete, hard on a red base.	(Cont)Months later: deeply nodular, ulcerated.	Punch biopsy then complete excision.	

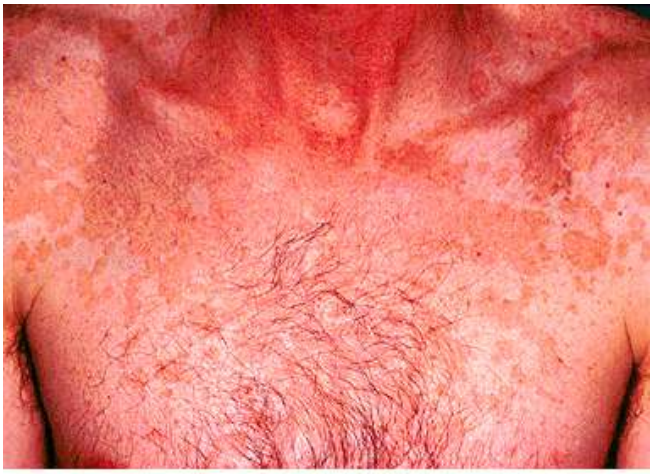
Entity	**location/Sx's/ setting**	**Mechanism**	**RX**	**other**
Basal cell ca	Waxy, semi-translucent, nodule. Central depression. Telangiectasias.	(Cont.) May be pigmented. May develop a rolled edge.	Shave biopsy. If aggressive histology, Mohs, micrographic surg, rad ther.	
Urticaria. Acute, chronic, cold, warm (cholinergic), exercise)	hives, pruritic, sharp borders. Occur in 25% of people. Trauma, sunlight, cold, heat. Dermatographism.	NOT food. In chronic urticaria, obtain Complement levels, C4 and C1 inhibitor level and function. An incr. ESR or eosinophilia point towards other entitis.	H1 competitors: hydroxyzine (Atarax, Vistaril) or diphenhydramine (Benadryl) 25 - 50 mg Q6H. Cyproheptadine (Periactin) may be especially useful for treatment of cold urticaria, 2 - 4 mg Q6-8hrs. H2 antagonists: Ranitidine (Zantac) or Cimetidine (Tagamet)	chronic: 4-36 hrs, recur-rences 1/3 persist beyond 6 weeks and may last 10 years in 40% where there is recurrence.

SUPERFICIAL FUNGAL INFECTIONS and WARTS

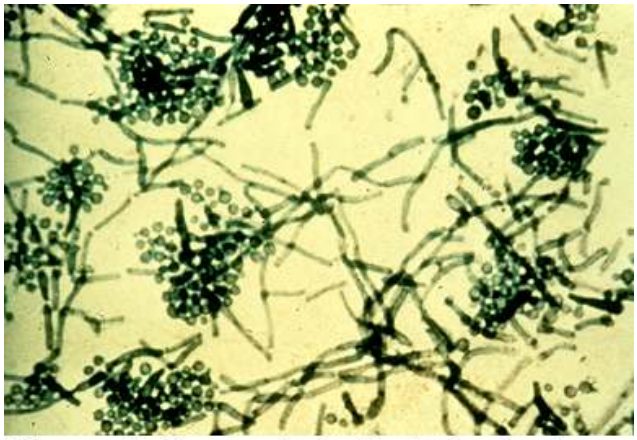
Entity	Location/Sx's/ Setting	Mechanism	Rx	Other
Tinea pedis	Bottom of feet, between toes, pruritic, red, vesicles. Maceration, fissuring, moccasin type with fine scale on entire sole.	Fungal inf'n with moisture	Antifungal creams. Terbenafine cream. Clotrimazole. Oral terbenafine or itraconazole.	Complicated tinea pedis: dermatophytid rx'n, recurrent cellulitis
Tinea versicolor	Chest, back, lower neck, proximal arms. Light brown or reddish brown macules, NON-pruritic.	lipophilic yeast Malassezia furfur or Pityrosporum orbiclase, ovale or Malassezia ovalis.	2.5% selenium sulfide. Selenium shampoo. Ketoconazole 2% shampoo. Orally: itraconazole 200 mg to block recurrences.	KOH prep: spores and hyphae (spaghetti and meatballs).
Onychomycosis	thickened colored nails. Ddx: ½ non-fungal: psoriasis (pitting), warts, lichen planus, bacteria.	A FUNGAL INFECTION. Send nail clippings for culture.	Laquer of Ciclopirox 8% laquer: cure of 9%. Oral Rx (success > 70%): Itraconazole (CHF has occurred) or terbinafine (hepatotoxicity has occurred).	
Warts	Transmission: Skin-to-skin or fomites. HPV. Scraping will reveal thrombosed capillaries beneath the wart (seeds).	Ddx: lichen planus, seborrheic keratosis, acrocordon, corns, syphyllis.	2/3 resolve in 2 years. If painful etc: salicylic a or lactic a (over the counter) resolves 70% of warts. Snip excision.	Liquid nitrogen near digital nerves can cause neuropathy.



Tinea versicolor Multiple small salmon-colored macules are evident on the arm that coalesce in some areas. Courtesy of Beth G Goldstein, MD and Adam O Goldstein, MD.



Tinea versicolor Multiple light brown coalescing macules are present on the chest. Courtesy of Beth G Goldstein, MD and Adam O Goldstein, MD.



Microscopic tinea versicolor Examination of scale with 10 percent potassium hydroxide reveals both hyphae and spores in the characteristic "spaghetti and meatballs" pattern. Courtesy of Beth G Goldstein, MD and Adam O Goldstein, MD.

Etiology: Malassezia furfur

Diff Dx: Seborrhea, eczema, pityriasis rosea, secondary syphillis.

Rx: Any anti-fungal agent.

SOME BLISTERING CONDITIONS IN ADULTS September 22, 2004

Disease	Setting	Location	Lesion	Lvl	Pathology	Treatment
Allergic Contact Dermatitis	Nickel, Chrome, poison ivy or sumac	Exposed surface	Erythema, bilsters, pruritic	Intra epid	Intra-epidermal spongiosis	Steroids
Pemphigus (vulgaris et al)	50-60 y.o. Rarely lymphomas, myasthenia gravis, thymoma, and drugs (captopril & penicillamine)	Mouth is 1st site, then scalp, face, chest, groin.	Vesicles & flaccid bullae, easily broken and extended (+Nikolsky's sign)	Intra epid	Separation of epithelial cells from each other (acantholysis); IgG Abs to desmoglein 3 is shown as + immunofluorescence to surrounding keratinocytes.	Prednisone, very hi dose, e.g., 150mg/d + immunosuppressive (azath,mtx,ctx). Plasmapheresis. IVIG
Bullous pemphi-goid	the most common of the blistering diseases. >60	Chest, abdomen, oral cavity (16%)	Tense bullae on a red base; some: urticarial plaques. Pressure applied to blister fails to extend it (-Nikolsky sign)	Sub epid	Often with Eos. Immunofluoresecence: Linear deposition of IgG and C3 at B Mb. Bullous pemphigoid antigen 1 and 2 (BPAG1 and BPAG2).	oral prednisone at < 1mg/kg (less than Rx of pemphigus). OR Tetracycline, with or without niacinamide.
Dermatitis herpeti-formis	**Celiac Sprue, ie., Gluten sensitive enteropathy (> 50%).** *	**Extensor surfaces, buttocks & trunk. Sometimes scalp & face. Symmetric.**	**Pruritic papulovesicles** (-Nikolsky sign)	Sub epid	blisters and clusters of neutrophils at the dermal papilla; Granular deposits of IgA at B Mb	**Avoid gluten (treats both skin and GI disease)** Dapsone.
Herpes	Adults	Dermotomes (zoster) or clusters (simplex)		Intra epid		
Paraneo-plastic phemphi-gus	Adults > 50	Oral & trunk	Papules & Bullae +Nikolsky	Itra epid	Intra-epidermal IgG Abs to desmoglein H3 and plakin proteins.	

*Celiac sprue is associated with other autoimmune endocrinopathies: Thyroid dis, DM 1, SLE, Sjogren's, sarcoid, vitiligo, alopecia areata,. Also, NH Lymphomas & GI Lymphomas.

SUB-EPIDERMAL BLISTERING DISEASES ONLY (duplicates part of prior table)
September 22, 2004

Disease	Setting	Location	Lesion	Lvl	Pathology	Treatment
Bullous pemphigoid	the most common of the blistering diseases. >60	Chest, abdomen, oral cavity (16%)	Tense bullae on a red base; some: urticarial plaques. Pressure applied to blister fails to extend it (-Nikolsky sign)	Sub epid	Often with Eos. Immunofluorescence: Linear deposition of IgG and C3 at B Mb. Bullous pemphigoid antigen 1 and 2 (BPAG1 and BPAG2).	oral prednisone at < 1mg/kg (less than Rx of pemphigus). OR Tetracycline, with or without niacinamide.
Epidermolysis bullosa acquisita	Autoimmune disease: scar-ring alopecia, RA, SLE, IBD	Trauma areas: hands (dorsum), feet, knees, extensor surfaces; also, scalp, face, neck. Occ. mucosal involvement.	**Tense vesicles & bullae on a pale base; healing with scarring and milia.** (-Nikolsky sign)	Sub epid	Linear deposition of IgG at B Mb which maps to the blister base of sodium chloride-split skin.	
Dermatitis herpetiformis	**Celiac Sprue, ie., Gluten sensitive enteropathy (> 50%).** *	**Extensor surfaces, buttocks & trunk. Sometimes scalp & face. Symmetric.**	**Pruritic papulovesicles** (-Nikolsky sign)	Sub epid	blisters and clusters of neutrophils at the dermal papilla; Granular deposits of IgA at B Mb	**Avoid gluten (treats both skin and GI disease)** Dapsone.
Linear IgA bullous dermatosis	Vancomycin or NSAIDs	Trunk, knees, elbows, buttocks, mouth	Annular or grouped vesicles or bullae (-Nikolsky sign)	Sub epid	clusters of neutrophils at the dermal papilla; Linear deposition of IgA at BMb	D/C drug. Dapsone
Cicatricial pemphigoid	elderly.	Oral, nasopharyngeal, conjunctivae; 35% of patients: scalp, head, neck, trunk.	Blistering & Erosions (-Nikolsky sign)		Subepidermal blister with inflammatory cells; IgG and C3 on B Mb. Auto Ab's may occur against BPAG2 laminin 2, or type VII collagen.	

*Sprue is associated with other autoimmune endocrinopathies: Thyroid dis, DM 1, SLE, Sjogren's, sarcoid, vitiligo, alopecia areata,. Also, NH Lymphomas & GI Lymphomas.

	PCT: Porphyria Cutanea Tarda	AIP: Acute Intermittent Porphyria
Cutaneous	Blistering of sun exposed areas	
Liver	Increase AST, ALT; increased risk of hepatocellular ca Increase in porphyrins in liver.	Increase risk of hepatocellular ca,
Neurologic		Abd pain 90% vomit 65% constipation 70% muscle weakness 50% Limb, H & N pain 50% HPT 45% convulsions 15% respiratory paralysis 12%
precipitants	Sun, ETOH, hep C inf'n, estrogen use, pregnancy, smoking, hemodialysis, iron excess	Pre-menstrual, decreased calories, many drugs, surgery, infection, etoh excess, cigarettes.
urine	increase uroporphyrin	Increase ALA and PBG, aminolevulinic acid, and porphobilinogen
serum	increase porphyrin (best diagnostic test) > 10 ug/dL	
Other		HypoNatremia, SIADH
Treatment	Cease precipitants, particularly alcohol. Phlebotomy Low dose chloroquine	Avoid certain drugs: AceI, CCBs, sulfa, many many others. IV heme preparations. IV carbohydrates.