

		Notes	Morphology	Labs	Management
CCLE	Discoid LE – Young females – Associated with SLE – Rarely resolves – Diagnosed by skin biopsy – Rare but causes concern as the face is the most commonly affected site and because of permanent scarring that may be seen.	Site: Sun exposed areas. Above head and neck. Possible M.M involvement: SSC arise from long standing DLE In scalp: Cause scarring permanent alopecia must be treated aggressively by: intralesional or systemic steroid	Red indurated plaques that evolve with atrophy, scarring and pigment changes. Hyperkeratosis characterized by follicular plugging. An erythematous papule or plaque with slight to moderate scaling. As the lesion progresses, the scale may thicken and become adherent to underlying epidermis (carpet-tack sign), and run into hair follicle *pigmentary changes: Hypo pigmentation in the central or inactive area and hyper pigmentation at the active border. (annular plaque)	In generalized form associated with SLE: +ve ssDNA +ve ANA ↑ ESR Leukopenia	Super-potent topical steroids [1st line] Anti-malaria[multiple] Retinoid *Avoid strong steroid if the lesions in the face, around the eye and the axilla to avoid atrophy *Resistant cases: immunosuppressive
	Hypertrophic LE	Site: Extremities Diagnosed by clinical appearance & biopsy	Hypertrophic lesions		
	LE-LP Overlap	Site: Extremities, palms and soles M.M involvement: Oral ulcers Nail involvement Diagnosed by biopsy; shows LP features			
	Chilblain LE	Site: Rim of ears Fingertips Calves and heels			
	Tumid LE	Site: Trunk	Edematous erythematous plaques.		Anti-malaria
	LE Panniculitis: – Doesn't resolve – Rx only reduces scars	Site: Fatty regions & proximal extremities	Starts as non-tender subcutaneous nodules without skin changes and if left untreated causes atrophy and scarring		Single; Intra-lesional steroids Multiple; systemic
SCLE	SCLE: –Young females – Associated with photosensitivity & arthralgia – 10–15% associated with SLE	Site: Sun exposed areas [face & upper trunk] A variant of LE in b/w SLE and DLE in photosensitive pts w/ +ve Anti-Rho(60%) & Anti-LA(40%) but not ANA Associations: – neonatal LE – Complement deficiency syndrome – Drug induced SCLE	Annular, papulosquamous or psoriasiform lesions Heals without scarring	+ve ANA +ve Anti-RO	Sun protection & sunscreens Anti-malaria
ACLE	ACLE: – 90% associated with SLE		Localized or generalized erythema Bullous and others		

		Signs and Symptoms	Notes	Labs	Management
SLE	<p>– ACR Diagnosis Criteria [4 out of 11]:</p> <ol style="list-style-type: none"> 1.DLE 2.Malar rash 3.Oral ulcer 4.Photosensitivity 5.CNS [seizures or psychosis] 6.Serositis [pleuritis or pericarditis] 7.Renal [proteinuria >0.5g/day] 8.Arthritis 9.Blood: hemolytic anemia, leukopenia, thrombocytopenia 10.Serology: +ve ANA 11.Serology: +ve Anti-dsDNA, anti-smith, anti-phospholipids 	<p>Cutaneous Manifestations:</p> <p>Malar rash Bollous LE Psoriatic plaques on elbows and knees M.M painless ulcers; oral, nasal and vaginal Fingertips edema and erythema with periungual telangiectasia Lupus hair and diffuse hair thinning.</p> <p>Systemic Manifestations:</p> <p>Joints→ Migratory arthralgia and arthritis. Vascular→ thrombosis & thromboembolism Cardiac→ pericarditis most commonly, myo/endocarditis Renal→ Acute nephritis or nephrosis. CNS→ Vasculitis manifested as hemiparesis, convulsions & psychosis + Sneddon syndrome Blood→ITP, coombs +ve hemolytic anemia, neutropenia, lymphopenia.</p>	<p>Drugs that induce SLE: Hydralazine, procainamide, sulfonamide or anti-convulsants.</p> <p>Associations: Renal involvement [anti-dsDNA & anti-C1q] Thrombosis [anti-phospholipids] Drug induced [ANA& Anti-histone]</p> <p>Diagnosis: Serology + skin biopsy Typical presentation (unwell, butterfly rash provoked by sun) To confirm: ANA + anti-dsDNA.</p>	<p>ANA Anti-dsDNA Anti-smith Anti-phospholipid ↓C2 & C4 ↑ESR CBC U&E MSU</p>	<p>Cutaneous symptoms: Locally: Sun protection Topical or intra-lesional steroids Topical calcineurin inhibitor Topical retinoid Systemic: Anti-malaria</p> <p>Systemic symptoms: Anti-malaria Systemic steroids</p>

NOTES on SLE:**Rash:**

Macular rash that affects the face at T zone & spares the nasolabial folds, so called “butterfly area” which differentiates it from rosacea and seborrheic dermatitis. It is provoked by sunlight. and in hands, it spares the joint area usually (between joints)
Seen in acute flare-ups and resolves after 6 wks without scarring or hyper-pigmentation.

Lupus Hair:

- a. Short hair along the frontal hair line.
- b. Permanent hair loss & scarring is unusual (seen in CDLE)
- c. The lesion is less florid

Serology [To diagnose a Lupus case]: IMP [MCQ]

ANA – Seen in all lupus [most sensitive for lupus]
Anti-dsDNA + Anti-Smith [Specific for SLE]
Anti-dsDNA [Renal involvement in SLE]
Anti-ssDNA [Association with DLE]
Anti-RO + Anti-LA [Association with SCLE]
Anti-phospholipids [Association with Anti-phospholipid syndrome]
Anti-nRNP [Association with mixed connective tissue disease]

Before starting treatment with Hydroxychloroquine: IMP [MCQ]

1. Refer patients for ophthalmology clinic:
It causes ocular muscles weakness and visual field defect. [after treatment follow-up twice a year]
2. Check G6PD levels
It causes aplastic anemia in patients with G6PD deficiency.
– If hydroxychloroquine is contraindicated use another anti-malaria or other treatment modality.