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Clinical Focus on Dermatology

Objective Structured Clinical Examination (OSCE)



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CHAPTER

4 Autoimmune Connective Tissue Disorders

Sudip Kumar Ghosh, Sharmistha Panja, Nazish Nalyer

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CASE 1: SYSTEMIC LUPUS ERYTHEMATOSUS

A 30-year-old woman presented with an unexplained fever persisting for 1 month. She also exhibited a fixed, erythematous, photosensitive patch on her malar regions, sparing the nasolabial folds. Additionally, she had a morbilliform erythematous rash with erosion on the trunk, back, and extremities **(Fig. 1)**, along with painless oral ulcers. Antinuclear antibodies (ANA) were positive, with a titer of 1:320, and had leukopenia [white blood cell (WBC) count 3500/μL].



FIG. 1: Clinical presentation of case 1.

Questions

1. What is the diagnosis?
2. What are the other oral manifestations of systemic lupus erythematosus (SLE) besides oral ulcers?
3. What are the poor prognostic factors associated with this condition?
4. How does the childhood-onset variant differ from the adult-onset variant of this disease?

Answers

1. SLE.
2. Oral manifestations in lupus patients may include cheilitis on the lips, gingivitis, glossodynia, fissured tongue, along with red and brown macules, telangiectasia, and cobblestone-like changes on the gums. The palate may display red dots, red macules, honeycomb-like plaques, and cobblestone patterns. On the buccal mucosa, red and brown macules can be seen, often accompanied by white streaks. Xerostomia, oral pigmentation, and altered salivary flow may also be associated features.
3. Poor prognostic factors at the time of diagnosis include young age at onset, male sex, certain ethnicities, low socioeconomic status, creatinine levels > 1.4 mg/dL, hypertension, massive proteinuria, nephrotic syndrome, hypoalbuminemia, anemia (hemoglobin levels below 12.4 g/dL), thrombocytopenia, lymphocytopenia, hypocomplementemia, and the presence of antiphospholipid antibodies.
4. In childhood-onset SLE, certain clinical manifestations are more common than in adults. These include malar rash, mucocutaneous involvement, such as ulcers, renal complications such as proteinuria and urinary cellular casts, seizures, headaches, cognitive dysfunction, mood disorders, psychosis, thrombocytopenia, hemolytic anemia, fever, and lymphadenopathy. In contrast, adults with SLE are twice as likely to present with Raynaud's phenomenon, pleuritis, arthralgia, photosensitivity, and sicca syndrome compared to children and adolescents.

CASE 2: SCLERODERMA

A 38-year-old woman presented with progressive skin tightening, starting in the fingers and extending to the forearms, upper arms, neck, and face. Additionally, she had resorption of terminal phalanges and fixed flexion deformity of the hands **(Fig. 2)**. The affected skin appeared shiny and taut, with a loss of normal skin creases, and no hair. Depigmented areas with perifollicular pigment retention were also observed.



FIG. 2: Clinical presentation of case 2.

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Questions

1. What is the diagnosis?
2. What are the hand changes associated with systemic sclerosis?
3. What is salt and pepper pigmentation?
4. Method used to semiquantify skin thickness in scleroderma.
5. Name four important scleroderma-like conditions and outline their distinguishing features.

Answers

1. Diffuse cutaneous systemic sclerosis.
2. Hand changes may include sclerodactyly, swelling of the hands, Raynaud's phenomenon, fingertip "rat bite" ulcers, stellate scars, joint contractures with flexion deformities, hyperpigmentation, salt-and-pepper pigmentation, calcinosis cutis, parrot beak nails, and resorption of digits.
3. The "salt-and-pepper appearance" is characterized by vitiligo-like depigmentation with retention of perifollicular pigmentation. The depigmentation may also spare regions over superficial veins and skin creases. This distinctive pigmentation pattern may be the sole visible clinical manifestation of systemic sclerosis sine scleroderma.
4. The method used to semiquantify skin thickness in scleroderma is the modified Rodnan skin score. This score is derived from clinical palpation of 17 specific body areas, including the fingers, hands, forearms, upper arms, chest, abdomen, thighs, lower legs, and feet. Each site is subjectively assessed for thickness on a scale of 0–3: 0 = normal (A), 1 = mild (B), 2 = moderate (C), and 3 = severe (D). The highest possible score is 51.
5. Four important scleroderma-like conditions are detailed in **Table 1**.

TABLE 1: Scleroderma-like conditions.

Scleroderma	Poorly defined woody and nonpitting induration of the skin, with inconstant overlying erythema or hyperpigmentation. It first involves the neck and then spreads to the shoulders, the upper trunk, and the arms symmetrically. It can reach the face, and rarely the buttocks and thighs. Interestingly, the swelling spares the hands and feet. Extracutaneous involvement is rare. Histopathological examination shows swollen collagen bundles and mucin accumulation
Scleromyxedema	Also known as papular mucinosis. Symptoms may include induration, sclerodactyly, and limited mouth opening. Excessive serum paraprotein, particularly immunoglobulin G (IgG) λ, may be present, along with significant dermal mucin deposition
Eosinophilic fasciitis	• This condition primarily affects the extremities and trunk, typically sparing the fingers • Characterized by local edema and a painful and symmetrical stiffening of the limbs, progressing rapidly to fibrosis, which can limit joint movements • Low-grade myositis may be present, and histopathological examination reveals dermal and hypodermal sclerosis with a normal epidermis, accompanied by occasional muscle inflammation. Peripheral blood eosinophilia is a common finding

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Nephrogenic systemic fibrosis	This condition arises following gadolinium exposure in patients with end-stage renal disease. It typically presents with symmetric painful skin thickening of the extremities, including the fingers, with pronounced induration, joint contractures, and decreased mobility. The face is almost always spared. In some cases, muscle and lung involvement can occur. Histopathological findings include fibroblast proliferation, mucin deposition, and dermal spindle cell proliferation, which are characteristic features

CASE 3: DERMATOMYOSITIS

A 35-year-old woman presented with difficulty combing her hair and rising from a squatting position for the past 6 months. She exhibited multiple violaceous papules on the knuckles of her hands **(Fig. 3)**, periorbital violaceous swelling, and dysphagia to solid foods. Laboratory tests revealed elevated muscle enzymes, but her antinuclear antibody test was negative.



FIG. 3: Clinical presentation of case 3.

Questions

1. What is the diagnosis?
2. What are the mucocutaneous manifestations of this condition?
3. What dermoscopic findings can be observed in the proximal nail folds of these patients?
4. How does juvenile dermatomyositis differ from adult-onset dermatomyositis?
5. What is antisynthetase syndrome?

Answers

1. Adult-onset dermatomyositis.
2. Mucocutaneous signs of dermatomyositis are outlined in **Table 2**.

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TABLE 2: Mucocutaneous changes of dermatomyositis.

Heliotrope sign: • *Gottron's papules* (slightly elevated, violaceous, pink, or dusky red papules located over the dorsal side of the metacarpal or interphalangeal joints) and *Gottron's sign* (A macular rash without papules with the same distribution as Gottron's papule) • *Heliotrope rash:* Periorbital red or violaceous erythema of one or both eyelids, often with edema • *Photodistributed poikiloderma:* Facial erythema, shawl sign, and V-neck sign • *Holster sign:* Symmetric poikiloderma of the hips and lateral thighs below the greater trochanter • Psoriasiform lesions • *Calcinosis cutis:* Particularly in juvenile dermatomyositis • *"Mechanic's hands:"* A hyperkeratotic eruption on the ulnar aspect of the thumb and radial and palmar aspect of the index finger, with desquamation and rhagades	*Less common associations:* • Cutaneous erosions or ulcerations • Flagellate erythema • Tender palmar macules and papules, hyperkeratotic palmar papules • Erythroderma • Cutaneous eruptions resembling pityriasis rubra pilaris (Wong variant) • Vesiculobullous lesions • Lipoatrophy (especially in juvenile dermatomyositis) • Small vessel vasculitis (notably in juvenile dermatomyositis) • Panniculitis
Nail-fold changes: Dystrophic and ragged cuticle (Samitz sign), cuticular hypertrophy, and nail-fold telangiectasias	
Scalp and hair changes: Erythematous or violaceous patch, poikiloderma, and scaling, nonscarring alopecia	
Oral mucosal changes: Oral ulcers, ovoid palatal patches, gingival erythema, hemorrhage, vesicles, leukoplakia-like plaques, and telangiectasias	

3. Dermoscopy reveals the presence of dilated vessels, avascular areas, elongated capillaries, tortuous vessels, "bushy capillaries," giant capillaries, hemorrhages, disorganized vascular architecture, interfollicular scales, peripilar casts, and tufting.
4. Juvenile dermatomyositis is associated with a higher frequency of neck muscle involvement, vasculopathy, calcinosis, cutaneous ulcers, and gingival telangiectasia, but it has fewer instances of interstitial lung disease (ILD), pulmonary arterial hypertension (PAH), malignancy associations, and myositis-specific antibodies compared to adult-onset dermatomyositis. Additionally, juvenile dermatomyositis generally has a better prognosis.
5. Antisynthetase syndrome is an autoimmune disorder marked by the presence of autoantibodies against specific aminoacyl transfer ribonucleic acid (tRNA) synthetases. Clinical manifestations include ILD, nonerosive symmetric polyarthritis, myositis, Raynaud's phenomenon, unexplained fever, and/or mechanic's hands. The typical "triad" of arthritis, myositis, and ILD is observed in up to 90% of cases.

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CASE 4: SJÖGREN'S SYNDROME

A 30-year-old woman presents with increased eye dryness, a dry mouth, and palpable purpura **(Fig. 4).** Schirmer's test (without anesthesia) revealed abnormal results (≤3 mm in 5 minutes). Additionally, she tested positive for anti-SSA (Ro) and/or anti-SSB (La) antibodies.



FIG. 4: Clinical presentation of case 4.

Questions

1. What is the diagnosis?
2. What skin changes have been reported in this syndrome?
3. How is this condition diagnosed?
4. What treatment options are available?

Answers

1. Primary Sjögren syndrome.
2. Other skin changes associated with Sjögren syndrome include hypergammaglobulinemic purpura, annular erythema, livedo reticularis, erythema multiforme, cutaneous vasculitis, erythema nodosum, erythema perstans, vitiligo, Raynaud phenomenon, nodular cutaneous amyloidosis, alopecia, papillary atrophy of the tongue, angular cheilosis, eyelid dermatitis, subcorneal pustular dermatosis, ulcerative lichen planus, and erythema elevatum diutinum.
3. Sjögren syndrome is primarily a clinical diagnosis, supported by several tests.
   - *Schirmer test* evaluates tear production by measuring the wetting of a standardized filter paper placed between the bulbar and lachrymal conjunctiva or inferior fornix of each eye; a result of wetting < 5 mm in 5 minutes confirms impaired tear production, suggestive of Sjögren syndrome.
   - *A salivary gland biopsy*, typically taken from the minor salivary glands inside the lip, often reveals a focal, periductal lymphocytic inflammatory infiltrate.
   - *Tests assessing salivary gland function and imaging techniques, such as sialography, scintigraphy, and high-resolution ultrasound*, can further confirm salivary gland pathology.

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- Additionally, *sialometry* is considered positive if <1.5 mL of saliva is collected over 15 minutes under unstimulated conditions.
- The presence of autoantibodies, such as anti-Sjögren's syndrome-related antigen A (anti-Ro/SSA), anti-Sjögren's syndrome type B (anti-La/SSB) antibodies, rheumatoid factor, and ANA, supports the diagnosis.

4. Treatment often requires a multidisciplinary approach. The treatment options are outlined in **Table 3**.

TABLE 3: Treatment of Sjögren syndrome.

Xerosis (dry skin)	Avoidance of soaps and harsh detergents; instead, use emollients and humectants to maintain skin hydration
Xerostomia (dry mouth)	Basic self-care measures to stimulate oral secretions prevent oral dryness and dental caries, as well as regular dental care. Treatment of oral candidiasis: Topical fluoride helps reduce the risk of dental caries. Chewing sugar-free gum, avoiding anticholinergic medications, using saliva substitutes, and pilocarpine or cevimeline can promote saliva production
Dry eyes	Large-frame glasses to minimize tear evaporation can be helpful. Medications used for the treatment of other conditions (e.g., antidepressants, antihistamines, anticholinergics, diuretics, and neuroleptics) may worsen symptoms of dryness, and alternatives should be sought whenever possible. Artificial tears, eyelid lubricants, sialogogues, topical cyclosporine (0.05%) ophthalmic solution, and tear duct plugs are recommended for symptom relief
Vaginal dryness	Lubricants can be used to improve comfort
Arthralgias and arthritis	Traditional nonbiologic disease-modifying antirheumatic drugs (DMARDs) such as hydroxychloroquine or low-dose weekly methotrexate and corticosteroids may be used depending upon the severity of symptoms
Neurological involvement, severe pulmonary disease, or vasculitis	These conditions may require immunosuppressive treatments, such as systemic steroids, leflunomide, azathioprine, mycophenolate, or rituximab. Gabapentin, pregabalin, venlafaxine, or duloxetine may be used for neuropathic pain

CASE 5: ROWELL SYNDROME

A 25-year-old woman with a 2-year history of SLE, with positive anti-nuclear antibody (ANA) (Titer: 1:320, Hep-2 cell line, IIF method; speckled pattern) and anti-Ro (SS-A) antibodies, presented with painful, pruritic, ring-shaped erythematous plaques over the different parts of the body, lasting 12 days. The lesions were symmetrically distributed across the face, neck, trunk, abdomen, and extremities. Some exhibited a "targetoid" appearance **(Fig. 5)**. Additionally, she had lesions consistent with discoid lupus erythematosus (LE) over the scalp. A lesional biopsy of the truncal lesion suggested erythema multiforme; however, direct immunofluorescence (DIF) testing was negative.

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FIG. 5: Clinical presentation of case 5.

Questions

1. What is the provisional diagnosis?
2. What are the typical clinical presentations of a patient with Rowell syndrome?
3. What are the important serological markers of Rowell's syndrome?
4. What are the common differential diagnoses of Rowell syndrome?
5. What are the therapeutic options available for Rowell syndrome?

Answers

1. *Provisional diagnosis*: Rowell syndrome (characterized by erythema multiforme-like lesions along with LE and distinctive immunological findings) is a rare condition whose recognition as a distinct entity is controversial and lacks broad acknowledgment. Currently, it is regarded as a separate subtype of chronic cutaneous LE.
2. It mostly affects middle-aged women, patients experience recurring episodes of annular lesions primarily on the limbs, with lesser involvement on the face, neck, chest, and mouth, in addition to typical features of any LE variant. The palms and soles are rarely affected. Initial lesions manifest as erythematous papules, progressing to annular lesions that may develop vesicles at the periphery, often mimicking "target-like" lesions. Typically, the condition resolves without scarring, but severe cases may result in bullae, necrosis, and ulceration. Erythrocyanosis, chilblains, and Raynaud phenomenon are also observed in patients with this variant.
3. The patient shows at least one positivity among speckled ANA, anti-Ro/SSA, and anti-La/SSB antibodies.
4. The differential diagnosis for Rowell syndrome includes conditions such as erythema multiforme, other variants of LE, drug-induced skin reactions such as Stevens–Johnson syndrome/toxic epidermal necrolysis (SJS/TEN), and other autoimmune or inflammatory skin disorders.

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5. The majority of individuals diagnosed with Rowell syndrome exhibit positive responses to treatment. Therapy for Rowell syndrome typically encompasses a variety of medications commonly employed in the management of LE, such as topical and oral steroids, hydroxychloroquine, dapsone, and immunosuppressive drugs, such as azathioprine and cyclosporine.

CASE 6: MORPHEA

An 11-year-old girl presented with skin hardening and hair loss over the left side of her scalp for the past 4 years **(Fig. 6)**. Upon examination, a brown-colored, poorly defined, linear band-like hardened and thickened plaque, approximately 4 × 10 cm in size, was observed over the left frontotemporal region, accompanied by scarring alopecia. The lesion extended onto the forehead, involving the left eyebrow. Examination of the face, eyes, oral mucosa, and other parts of the body revealed no abnormalities. There was no history of any abnormalities in the central nervous system (CNS). Baseline investigations, including magnetic resonance imaging (MRI) and computed tomography (CT) scans of the brain, were unremarkable. Histopathological analysis revealed dermal sclerosis, along with significant atrophy of eccrine glands and remnants of atrophic hair follicles.



FIG. 6: Linear band like plaque accompanied by scarring alopecia.

Questions

1. What is the diagnosis?
2. What is the literal meaning of it?
3. What is the clinical presentation?
4. What are the differential diagnoses?
5. What are the treatment options for this condition?

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Answers

1. En coup de sabre (ECDS)
2. ECDS, French for "the cut of a sword," represents a subtype of linear morphea, a localized form of scleroderma primarily affecting the frontotemporal and forehead regions.
3. It predominantly occurs in young children and women (female to male ratio of 2:1). ECDS typically presents unilaterally and progresses slowly. It may manifest as a linear band resembling a port wine stain or multiple small plaques that merge together. During the inflammatory phase, it exhibits erythematous or lilac-colored borders and may extend to involve the eyebrows, nose, and other facial areas, including the oral mucosa. As inflammation subsides, a linear, hardened, and thickened plaque develops, often accompanied by scarring alopecia. In severe cases, ECDS can affect the underlying muscles and bones, resulting in significant disfigurement. Rarely, it may lead to ocular complications and seizures due to involvement of the eyes and central nervous system, respectively.
4. Differential diagnoses to consider include traumatic injury, linear facial lichen sclerosus, linear cutaneous LE, and verrucous epidermal nevus.
5. Treatment of ECDS poses a considerable challenge. In active lesions, topical or intralesional corticosteroids are often the treatment of choice. Other options include topical calcineurin inhibitors, vitamin D analogs, phototherapy (such as NB-UVB, UVA-1, and BB-UVA), and immunosuppressants. In cases of severe cosmetic disfigurement, reconstructive surgery may be necessary.

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Sudip Das MD(Dermatology and Venereology) (AIIMS New Delhi) FRCP(Edinburgh) is a renowned dermatologist and academician, currently serving as a Professor and Head of the Department at Calcutta National Medical College and Hospital, Kolkata, West Bengal, India. With an impressive record of 134 publications and 397 presentations, he has made significant contributions to Dermatology Research and Education. Dr Das has been honored with several prestigious awards, including the IADVL Best Teacher's Award (2018), the Ganapati Panja Award (2021), and notable orations like the Dr RK Panja Oration (2023) and the CSRAO Memorial Oration (2024). He has served in various leadership roles, including Vice-President of Indian Society for Pediatric Dermatology (ISPD), Past National Coordinator for Psoriasis, and Managing Editor of the Indian Journal of Dermatology (IJD). His areas of expertise include psoriasis, CADR, vitiligo, chemical peels, hair disorders, and leprosy. A visionary leader, Dr Das continues to inspire through his research, mentorship, and global dermatological collaborations.

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