

1: Skin signs of rheumatic disease | DermNet New Zealand

Recent advances in the understanding of cutaneous findings in cutaneous lupus erythematosus (CLE) and systemic LE (SLE), dermatomyositis, systemic sclerosis, and rheumatoid arthritis in terms of clinical evaluation, triggers of disease, outcome measures, systematic clinical studies, and treatment will be discussed.

Unlike DLE, there is no scarring. The cutaneous manifestations of SLE include malar erythema, photosensitivity, oral ulcers, discoid plaques, bullae, purpura, calcinosis cutis, and alopecia. The butterfly rash malar erythema is the most common expression of SLE Fig. Treatment includes sun protection; intralesional, topical, and systemic corticosteroids; antimalarials; dapsons; and immunosuppressants. Scleroderma Scleroderma is an autoimmune skin disease that can be localized or generalized. The localized form, known as morphea, begins as erythematous patches that evolve into dusky, hypopigmented, indurated plaques with violaceous borders, usually on the trunk. Differential diagnosis includes diabetic sclerodema, scleromyxedema, and chronic graft-versus-host disease. Treatment includes vasodilating drugs, phototherapy UVA1 for limited disease, methotrexate, and cyclophosphamide. Affected patients, usually men, often have vesicles and crusted plaques on the penis circinate balanitis and erythematous pustules and papules on the palms and soles keratoderma blennorrhagicum that can mimic pustular psoriasis. Differential diagnosis includes psoriasis, juvenile plantar dermatoses, rheumatoid arthritis, ankylosing spondylitis, and gout. Treatment includes topical corticosteroids, cyclosporine, or acitretin for refractory disease. Erythema Chronicum Migrans Erythema chronicum migrans, the hallmark of Lyme disease, reflecting early infection with the tick-borne spirochete *Borrelia burgdorferi*, develops as a red macule or papule at the site of the tick bite and gradually enlarges to an annular, reddened plaque Fig. Late sequelae include meningoencephalitis, myocarditis, and peripheral neuropathy. Differential diagnosis includes cellulitis, spider bite, erythema multiforme, and erythema annulare centrifugum. Other features include scaly, telangiectatic plaques with atrophy and hypopigmentation poikiloderma on the face, neck, trunk, and extremities; malar erythema; and nail abnormalities periungual telangiectases and cuticular hypertrophy. Diagnostic criteria include the aforementioned changes plus elevated creatine kinase or aldolase level, positive Jo-1 antibody, and electromyographic changes. In adults, dermatomyositis has a strong association with neoplasm, usually an adenocarcinoma of the breast, gastrointestinal tract, or lung. Differential diagnosis includes SLE and photosensitive drug eruption. Biopsy reveals a characteristic neutrophilic infiltrate, and direct immunofluorescence demonstrates deposition of IgA at the dermal-epidermal junction. Most patients have an asymptomatic gluten-sensitive enteropathy or, less commonly, thyroid disease. Differential diagnosis includes linear IgA dermatosis, bullous pemphigoid, scabies, contact dermatitis, and bullous lupus erythematosus. Treatment includes dapsons, sulfapyridine, and a gluten-free diet. Acrodermatitis Enteropathica Acrodermatitis enteropathica is an inherited or acquired condition characterized by pustules, bullae, scaling in an acral and periorificial distribution, and concomitant zinc deficiency. When inherited, acrodermatitis enteropathica results from a mutation in SLC39A, which encodes an intestinal zinc transporter. In adults, disease can occur after total parenteral nutrition without adequate zinc supplementation; with alcoholism, other malabsorption states, or inflammatory bowel disease; or as a consequence of bowel surgery. Most patients have diarrhea. Differential diagnosis includes other nutritional deficiencies, such as niacin or biotin deficiency, and necrolytic migratory erythema. Treatment is zinc supplementation. Necrolytic Migratory Erythema Necrolytic migratory erythema glucagonoma syndrome is a rare disease characterized by erythematous, scaly plaques on acral, intertriginous, and periorificial areas, in association with an islet cell tumor of the pancreas. Associated signs include hyperglycemia, diarrhea, weight loss, and atrophic glossitis. Treatment is removal of the tumor. Treatment of the hepatitis C infection often leads to resolution of the vasculitis. Click to Enlarge Lichen planus Fig. Lichen planus also occurs with primary biliary cirrhosis and hepatitis B virus immunization. Oral erosive lichen planus is the most common expression of lichen planus in hepatitis C patients. Treatment includes topical and intralesional corticosteroids, topical immunomodulators, and phototherapy. Necrolytic acral erythema, characterized by pruritic keratotic plaques on the upper and

lower extremities, is a distinctive finding in hepatitis C infection and can resemble a deficiency dermatosis. Porphyria cutanea tarda is discussed later. Click to Enlarge Hereditary Hemorrhagic Telangiectasia Hereditary hemorrhagic telangiectasia Osler-Weber-Rendu syndrome is an autosomal dominant disorder characterized by numerous telangiectases on the skin and oral mucosa Fig. Telangiectases can involve the lungs, liver, brain, eyes, and gastrointestinal tract; hemorrhage can occur at any site. Pulmonary arteriovenous fistulae and central nervous system angiomas can also occur. Differential diagnosis includes generalized essential telangiectasia. Treatment includes estrogen therapy or oral contraceptives in postpubertal women, laser cauterization, selective embolization, and supportive care. Muir-Torre Syndrome Muir-Torre syndrome is a disorder characterized by one or more sebaceous tumors adenoma, epithelioma, carcinoma and one or more internal neoplasms, usually colorectal or genitourinary, rarely lymphoma. Treatment is isotretinoin and regular GI and genitourinary evaluation. Click to Enlarge Peutz-Jeghers Syndrome Peutz-Jeghers syndrome is an autosomal dominant disease characterized by lentiginos on the skin periorbital region, dorsal surfaces of the fingers and toes and mucosa lips, buccal mucosa and hamartomas of the stomach, small intestine, and colon. The polyps are usually benign with low malignant potential, but patients have a 10 to 18 times greater lifetime risk of cancer, especially GI malignancies. Treatment includes regular and routine endoscopy and symptomatic treatment for hypogeusia and diarrhea. Pyoderma Gangrenosum Pyoderma gangrenosum is a neutrophilic dermatosis characterized by painful ulcers with boggy, undermined edges and a border of gray or purple pigmentation Fig. The ulcers often follow trauma pathergy and begin as pustules or nodules that ulcerate and extend centrifugally. Fifty percent of patients have underlying rheumatoid arthritis or inflammatory bowel disease or, less often, a paraproteinemia, usually an IgA gammopathy. Click to Enlarge Differential diagnosis includes infection, vasculitis, spider bite, and factitious disorder. Treatment includes treatment of underlying disease if applicable, local wound care, systemic and intralesional corticosteroids, cyclosporine, and infliximab. Renal Disease Nephrogenic Systemic Fibrosis Nephrogenic systemic fibrosis, also known nephrogenic fibrosing dermopathy, is a recently described disorder that resembles scleroderma. Nephrogenic systemic fibrosis occurs in patients who have end-stage renal disease and are on dialysis and occasionally in patients with acute renal failure or after kidney transplantation. Nephrogenic systemic fibrosis is characterized by thick, indurated plaques on the extremities and the trunk. Disease can be progressive, leading to joint contractures. Autopsies have demonstrated that disease is not limited to the skin; visceral organ and muscle fibrosis has been noted. The cause remains unclear, but the MRI contrast agent gadolinium might have a role in the pathogenesis of this condition. Treatment includes immunosuppressive agents, phototherapy, topical steroids, retinoids, and photopheresis, all with little benefit. Patients have a significantly increased risk of renal oncocytoma and chromophobe renal carcinoma. Spontaneous pneumothorax can occur secondary to rupture of pulmonary cysts. Mutations in the folliculin gene on chromosome 17 are responsible for this syndrome. Endocrine and Metabolic Disease Porphyrias Porphyrias are inherited or acquired disorders of heme biosynthesis and can be erythropoietic, hepatic, or mixed in nature, each associated with a specific enzyme defect in the heme pathway. Porphyria cutanea tarda, the most common porphyria, is a hepatic porphyria with acquired and sporadic forms Fig. It is caused by a deficiency in uroporphyrinogen decarboxylase, leading to the accumulation of uroporphyrin in the urine and serum. Click to Enlarge Precipitating factors include alcohol ingestion, estrogen administration, certain hepatotoxins dinitrochlorobenzene, carbon tetrachloride , HIV infection, hemochromatosis, and hepatitis C infection. Manifestations of porphyria cutanea tarda include photosensitivity, skin fragility, bullae and erosions on sun-exposed skin especially dorsal hands , and hypertrichosis. Biopsy reveals a subepidermal bulla with festooning of the dermal papilla. Direct immunofluorescence reveals IgG and C3 at the dermal-epidermal junction and in vessel walls. Differential diagnosis includes bullous SLE, epidermolysis bullosa acquisita, pseudoporphyria, and variegate porphyria. Treatment includes phlebotomy and antimalarial drugs. Pseudoporphyria Pseudoporphyria mimics porphyria cutanea tarda without an enzyme defect; plasma and urinary porphyrins are normal. Medications NSAIDs [especially naproxen], furosemide, and tetracycline are the most common cause of pseudoporphyria. Less common causes are tanning bed use and hemodialysis. Differential diagnosis is the same as for porphyria cutanea tarda. Treatment includes removal of the cause. Box 1 outlines the most common cutaneous

manifestations of diabetes, arranged by frequency of occurrence most to least frequent.

2: Dermatologic Signs of Systemic Disease

Cutaneous Manifestations of Rheumatic Diseases has been fully revised and updated for the Second Edition, with nationally recognized experts contributing many sections. The text provides a comprehensive treatment of the dermatologic appearances of rheumatic diseases, with a special focus on how skin changes correlate with the underlying disease process.

It is important to correctly classify cutaneous lupus erythematosus CLE , as it helps determine the underlying type and severity of SLE. Occurs predominantly on sun-exposed skin with an insidious onset, unlike toxic epidermal necrolysis. Typically triggered or exacerbated by exposure to UV light. On recovery may have postinflammatory pigmentation changes without scarring. Start as macules or papules that progress to hyperkeratotic plaques. Photosensitive so plaques usually occur on sun exposed skin. Does not lead to scarring but can result in postinflammatory hypo- or hyperpigmentation. Monitor to exclude progression to SLE. Subacute lupus erythematosus Chronic CLE Discoid LE DLE - discoid plaques erythematous, well demarcated plaques covered by scale that become hyperkeratotic, leading to atrophy and scarring. Dyspigmentation of the peripheries is also common in certain ethnicities Asian, Indian. There is follicular involvement, causing reversible and irreversible scarring alopecia. Affects face, outer ears, neck, sun-exposed areas and lips. Has overlapping features with lichen planus. Most commonly, painless erythematous patches on oral mucosa that develop into chronic plaques that can centrally ulcerate. These also affect nasal, conjunctival and genital mucosa. Chronic discoid lupus erythematosus Many drugs are thought to induce SLE and drug-induced LE often includes cutaneous signs. Examples include hydralazine, isoniazid, chlorpromazine, procainamide, phenytoin, minocycline and anti-TNF medications. Some use the term lupus panniculitis to refer to subcutaneous involvement only, and lupus profundus when there is a combination of lupus panniculitis with discoid LE. Precipitated by exposure to the cold, so often presents in winter. Associated with nail fold telangiectasia. LE tumidus - variant of chronic CLE with succulent or indurated erythematous plaques without surface change. Rare forms of chronic cutaneous lupus erythematosus.

3: Cutaneous Manifestations of Rheumatic Diseases

What is rheumatic disease? Many autoimmune connective tissue diseases and vascular conditions in rheumatology have cutaneous manifestations.. Skin signs are useful in the diagnosis of rheumatic disease, as they often precede systemic symptoms.

4: Cutaneous Signs of Rheumatic Disease

Dermatologic manifestations are currently observed in autoimmune rheumatic diseases, particularly in the main systemic vasculitides, rheumatoid arthritis and relapsing poly-chondritis. In systemic vasculitides, some skin lesions correspond to a skin localization of the systemic vasculitis while.

5: www.enganchecubano.com: Customer reviews: Cutaneous Manifestations of Rheumatic Diseases

Rheumatologic diseases are highly associated with clinical symptoms of the skin, nails, and mucosal membranes. This book provides the practicing rheumatologist with a rapid, easy to consult reference to help interpret the nature of these cutaneous lesions and then quickly decide upon the.

6: Rheum2Learn Rheumatic Manifestations of Systemic Diseases

Skin manifestations can be hallmark features in some rheumatic diseases, especially lupus erythematosus, dermatomyositis, and systemic sclerosis.

7: Skin signs and systemic disease | DermNet New Zealand

Rheumatologic diseases, such as systemic lupus erythematosus (SLE, shown), have a wide variety of cutaneous manifestations that can be easily confused. Careful attention to characteristic physical examination findings can help determine the diagnosis even before a lengthy laboratory panel has been ordered.

8: Rheumatic Diseases - Types, Causes, Risks & Symptoms | Everyday Health

â€¢ Skin disease present at some time in the Skin disorders in RA â€¢ Rheumatoid nodules Cutaneous Manifestations of Rheumatologic Disease.

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