SYSTEMIC LUPUS ERYTHEMATOSUS JOURNAL pdf

1: Systemic Lupus Erythematosus | West Indian Medical Journal

The author reviews environmental, genetic, epigenetic, and hormonal factors in systemic lupus erythematosus, its diverse organ manifestations, and the myriad immune abnormalities that characterize.

Submit manuscript at https: Open Access is a global, peer reviewed, Open Access online journal that provides a central point for publication of basic, clinical, translational, and epidemiological studies of all aspects of lupus and related diseases. Open Access journals were developed in response to the need for a barrier-free forum for publication of groundbreaking studies in lupus. The journal publishes research works on lupus from fields including, but not limited to: It will provide an integrative journal for molecular pathology and digital pathology ,creating an open case discussion platform to be used during daily diagnostic work. Open access is using Editorial Tracking System for quality in review process. Editorial Tracking is an online manuscript submission, review and tracking system used by most of the best open access journals. These lesions later become as a red, inflamed patch with a scaling and crusty appearance. A rim darker than the normal skin while the centre areas may appear lighter in colour. Many different body systems, including joints, skin, kidneys, blood, heart, and lungs Are affected by the Symptoms of these diseases. Advances in Lupus Treatment Several advances have been taken place from last 50 years which has improved the human health. Advances like faster diagnosis, renal dialysis and transplant, and better general care including lowering of blood pressure and treating osteoporosis have had significant roles in this. Advances in Lupus Diagnosis To diagnose lupus is critical because signs and symptoms vary from person to person. It may vary over time and overlap with those of many other disorders. No test can diagnose lupus. However the combination of blood and urine tests, signs and symptoms, and physical examination observations leads to the diagnosis. However, Lupus does appear in certain families, where when one of two identical twins has lupus, results in an increased chance that the other twin will also evolve the disease. These findings, as well as others, strongly prove that genes are involved in the expansion of lupus. Lupus anticoagulant is a misnomer, which is actually a prothrombotic agent. Lupus anticoagulant antibodies in living systems will result in an increase in inappropriate blood clotting with an individual. It is sometimes mistaken for lupus though as many as one in four people with lupus could also have symptoms that are similar to FM. It is found to be rare, but characteristic thing in which patients present with edematous erythematous plaques , usually on the trunk. Related Journals of Lupus Tumid Current Problems in Dermatology , Anais Brasileiros de Dermatologia Brazil, Acta Dermatovenerologica Alpina, Panonica et Adriatica Slovenia, Acta Dermato-Venereologica, Supplement, Experimental Dermatology, Supplement Subcutaneous Lupus It is considered to be clinically distinct subset of cases of lupus erythematous that is mostly observed in white women having age between 15 to 40, consisting of skin lesions that are scaly and develop as polycyclic annular lesions or psoriasiform plaques. It is said to be chronic , unabating form of lupus erythematosus with the fingertips, rims of ears, calves, and heels affected especially in case of women. Lupus Cerebritis Cerebritis is found to be an infection of the brain that normally results to the generation of an abscess within the brain itself. Cerebritis usually arise as a result of an underlying condition, which prevails the inflammation of the brain tissue. It is commonly found in patients with lupus. Lupus cerebritis may transpire in adults and children. This state is in the differential diagnosis for many neurologic state and conditions. Sun exposure also plays a critical role. In contrast, some of the medications used to treat lupus may cause side effects that include hives. But there is a difference between positive and negative feelings which may lead to treatable illness called clinical illness. Symptoms of clinical illness are loss of interest in activities and responsibilities that used to be important. Reports on systemic lupus erythematosus SLE , systemic lupus erythematosus SLE , Life threatening pneumonia in a lupus patient, hypertrophic lupus erythe matosus are in this zone.

SYSTEMIC LUPUS ERYTHEMATOSUS JOURNAL pdf

2: Systemic Lupus Erythematosus | Dermatology | JAMA | JAMA Network

(11) Antinuclear antibodies and lupus erythematosus cells are the consequences rather than the causes of SLE. 1 From the Medical Research Council Environmental Radiation Research Unit, Department of Medical Physics and the Department of Dermatology, The General Infirmary, Leeds, England.

Multimedia Abstract Systemic lupus erythematosus is an autoimmune disease with a significant genetic component to susceptibility. Some environmental risks are known, and identification of specific genetic factors promises to define new molecular targets for therapy. Broad immunosuppression will be replaced by early, selective, and individualized intervention. Mortality rates will decline, and insights into therapy may apply to other autoimmune conditions. Systemic lupus erythematosus SLE is a multisystem autoimmune disease involving both humoral and cellular aspects of the innate and acquired immune systems. Lupus is characterized by autoantibodies with a spectrum of specificities that participate in disease pathogenesis. Lupus occurs worldwide and affects females more commonly than males Lupus, predominantly a disease of younger women, shortens life expectancy, creates significant morbidity, and accounts for substantial total health care expenditures. Major Clinical and Research Advances Clinical management of SLE is based on use of nonsteroidal anti-inflammatory drugs NSAIDs , the addition of hydroxychloroquine and other agents originally developed as antimalarials, targeted and judicious use of glucocorticoids, including large intravenous doses, and aggressive use of other immunosuppressive agents, such as cyclophosphamide. Vigorous management of comorbid conditions, including hypertension and infection, has decreased mortality in persons with SLE. Autoreactivity encompasses a broad range of specificities that can include inciting antigens and other antigens through spreading of the immune response. Nucleosomes, apoptotic material, and efficient pathways for routine, nonimmunogenic clearance appear pivotal in pathogenesis of SLE. Equally, effector pathways for inflammation are critical for the development of end-organ damage. Current Scientific Foundation Lupus involves abnormal activity of the immune system in response to environmental stimuli encountered by the genetically susceptible host. Family studies emphasize the heritability of the SLE diathesis, but susceptibility is polygenic, involving multiple genes with a threshold effect. Deficiencies of complement and other opsonins, genetic variants of IgG and C-reactive protein receptors, and inflammatory cytokine promoter variants have been implicated as components of genetic susceptibility factors. The presence of autoantibodies and autoreactive T cells indicates broad involvement of the immune system, and noninflammatory mechanisms also contribute to vascular and organ injury. Animal models and clinical observations suggest that different sets of genes can produce similar clinical phenotypes. Consequently, identification of both environmental events and genetic susceptibility factors is critical for understanding SLE. Cutting-Edge Research Substantial investigative efforts are focused on studying SLE multiplex families and affected sibling pairs to establish regions of linkage in the genome with the SLE phenotype. Although apoptosis per se does not appear to be grossly defective in SLE, the processing of apoptotic cells and debris contribute to immune dysregulation. Apoptotic material may alter the local tissue environment and the presentation of self as antigenic. Therefore, the determinants of tolerance and the pathways that circumvent tolerance are central to the lupus diathesis. The strong heritability, measured by the risk of disease among siblings, and the convergence of several investigative groups on specific genetic regions of interest underscore the promise of this approach. Nonetheless, the task of unraveling this complex, and perhaps heterogeneous, disease is daunting. Effective collaborations with large cohorts of both simplex and multiplex families will be essential. Appropriate understanding of "phenotype" and access to state-of-the-art informatics tools are essential for this undertaking. The ability to take the discoveries from genetics, functional genomics, and pathophysiology to the bedside will require appropriate clinical tools to evaluate efficacy and outcomes. Many of these tools are at hand, and they must be woven into an overall effort addressing new therapies. Forecast for Research Advances The next 25 years will contain remarkable progress in the understanding and management of SLE. Identification of susceptibility genes and their contribution to disease pathways will provide insight into the understanding of environmental triggers. Assessment of individual genetic "portfolios" with gene

array technology, combined with advances in knowledge about exogenous stimuli, will facilitate prevention of SLE. New markers of immune activation and deviation will enable early therapeutic intervention. Biotechnology will provide more effective means of immunomodulation, perhaps through antigen-specific tolerance induction, selective deletion of activated immune cells, or interruption of inflammatory cascades. Glucocorticoid use will decline and alkylating agents will no longer be part of the therapeutic armamentarium. Early, effective interventions will reduce comorbidities, which will be attenuated further by aggressive management of the causes of morbidity. Gene therapy for such a complex genetic disease will be used first for drug delivery, not germ line modification. Discoveries in one autoimmune disease will have lessons and applications for other diseases. More targeted therapies will replace broad, nonspecific immunosuppression for most treatment.

SYSTEMIC LUPUS ERYTHEMATOSUS JOURNAL pdf

3: Research Advances in Systemic Lupus Erythematosus | Dermatology | JAMA | JAMA Network

Systemic lupus erythematosus (SLE) is a clinically heterogeneous disease which is autoimmune in origin, and characterized by the presence of autoantibodies directed against nuclear antigens. It is, by definition, a multi-system disease, and patients can present in vastly different ways.

Summary What is lupus? Lupus is an autoimmune disease. This means that your immune system attacks healthy cells and tissues by mistake. This can damage many parts of the body, including the joints, skin, kidneys, heart, lungs, blood vessels, and brain. There are several kinds of lupus Systemic lupus erythematosus SLE is the most common type. It can be mild or severe, and can affect many parts of the body. It usually goes away when you stop taking the medicine. Neonatal lupus, which is rare, affects newborns. It is probably caused by certain antibodies from the mother. Anyone can get lupus, but women are most at risk. Lupus is two to three times more common in African American women than in Caucasian women. African American and Hispanic women are more likely to have severe forms of lupus. The cause of lupus is not known. What are the symptoms of lupus? Lupus can have many symptoms, and they differ from person to person. Some of the more common ones are Pain or swelling in joints Muscle pain Fever with no known cause Red rashes, most often on the face also called the "butterfly rash" Chest pain when taking a deep breath Hair loss Pale or purple fingers or toes Sensitivity to the sun Swelling in legs or around eyes Mouth ulcers Swollen glands Feeling very tired Symptoms may come and go. When you are having symptoms, it is called a flare. Flares can range from mild to severe. New symptoms may appear at any time. How do I know if I have lupus? So it may take months or years for a doctor to diagnose it. Your doctor may use many tools to make a diagnosis:

SYSTEMIC LUPUS ERYTHEMATOSUS JOURNAL pdf

4: Lupus | Lupus Symptoms | SLE | MedlinePlus

Systemic Lupus Erythematosus Systematic Lupus Erythematosus (SLE) is a chronic disease with a significantly improved life expectancy due to early recognition, diagnosis, monitoring, and therapy of patients.

Immediate access to this article To see the full article, log in or purchase access. He completed an internal medicine residency at Christiana Care Health Services and a rheumatology fellowship at Georgetown University School of Medicine. Address correspondence to James M. Reprints are not available from the authors. The authors indicate they do not have any conflicts of interest. The authors thank Cheryl Mongillo and Teresa Gill Cirillo for assistance in preparing the manuscript. Clinical manifestations of systemic lupus erythematosus. Guidelines for referral and management of systemic lupus erythematosus in adults. Treatment of systemic lupus erythematosus: Controlled trial with chloroquine diphosphate in systemic lupus erythematosus. Longterm ultraviolet-A1 irradiation therapy in systemic lupus erythematosus. Ultraviolet-A1 â€" nm irradiation therapy in systemic lupus erythematosus. Cyclosporine-A plus steroids versus steroids alone in the month treatment of systemic lupus erythematosus. Int J Clin Lab Res. Double blind, randomized, placebo controlled clinical trial of methotrexate in systemic lupus erythematosus. Bromocriptine in systemic lupus erythematosus: Effect of large doses of prednisone on the renal lesions of and life span of patients with lupus glomerulonephritis. J Lab Clin Med. Treatment of lupus nephritis: Am J Kidney Dis. Undiagnosed systemic lupus erythematosus in the community. Estimates of the prevalence of arthritis and selected musculoskeletal disorders in the United States. Systemic onset juvenile rheumatoid arthritis. Retrieved March 20, , from http: Incidence of systemic lupus erythematosus. Race and gender differences. A clinical and serological comparison of familial and nonfamilial systemic lupus erythematosus in Ireland. Family and twin studies in systemic lupus erythematosus. Genome scan of human systemic lupus erythematosus by regression modeling: Am J Hum Genet. General symptomatology and diagnosis of systemic lupus erythematosus in adults. Gilboe IM, Husby G. Application of the revised criteria for the classification of systemic lupus erythematosus on a cohort of Norwegian patients with connective tissue disease. Agespecific incidence rates of myocardial infarction and angina in women with systemic lupus erythematosus: Outcome in systemic lupus erythematosus: Contribution of traditional risk factors to coronary artery disease in patients with systemic lupus erythematosus. Clinical and immunological profile of SLE: Marini R, Costallat LT. Young age at onset, renal involvement, and atrial hypertension are of adverse prognostic significance in juvenile systemic lupus erythematosus. Rev Rhum Engl Ed. Childhood-onset systemic lupus erythematosus: Immunological and clinical differences between juvenile and adult onset of systemic lupus erythematosus. Association between clinical factors, socioeconomic status, and organ damage in recent onset systemic lupus erythematosus. Overview of the therapy and prognosis of systemic lupus erythematosus in adults. Incidence studies of systemic lupus erythematosus in southern Sweden: Morbidity and mortality in systemic lupus erythematosus during a 5-year period. A multicenter prospective study of 1, patients. Clinical characteristics and outcome of southern Chinese males with systemic lupus erythematosus. SLE mortality in an oriental population. A multicentre study of Danish patients with systemic lupus erythematosus. Disease mortality and clinical factors of prognostic value. Survival analysis of European Spanish patients with systemic lupus erythematosus. Trends in the incidence and mortality of systemic lupus erythematosus, â€" Mortality studies in systemic lupus erythematosus. Results from a single center. Improved survival over 24 years. Hospital experience and expected mortality in patients with systemic lupus erythematosus: The revised criteria for the classification of systemic lupus erythematosus. Updating the American College of Rheumatology revised criteria for the classification of systemic lupus erythematosus [Letter]. Evaluation of the ARA lupus criteria data set in pediatric patients. Usefulness of antinuclear antibody testing to screen for rheumatic diseases. Guidelines for the initial evaluation of the adult patient with acute musculoskeletal symptoms. Guidelines for clinical use of the antinuclear antibody test and tests for specific autoantibodies to nuclear antigens. American College of Pathologists. Arch Pathol Lab Med.

SYSTEMIC LUPUS ERYTHEMATOSUS JOURNAL pdf

5: Systemic lupus erythematosus

Skip to Journal menu Skip to Issue articles. select article Understanding the role of environmental factors in the development of systemic lupus erythematosus.

Signs and symptoms Common symptoms of SLE [8] SLE is one of several diseases known as " the great imitator " because it often mimics or is mistaken for other illnesses. Diagnosis can thus be elusive, with some people having unexplained symptoms of SLE for years. Common initial and chronic complaints include fever , malaise , joint pains , muscle pains , and fatigue. Because these symptoms are so often seen in association with other diseases, these signs and symptoms are not part of the diagnostic criteria for SLE. When occurring in conjunction with other signs and symptoms see below , however, they are considered suggestive. Males tend to have more seizures , kidney disease , serositis inflammation of tissues lining the lungs and heart , skin problems , and peripheral neuropathy. The three main categories of lesions are chronic cutaneous discoid lupus, subacute cutaneous lupus, and acute cutaneous lupus. People with discoid lupus may exhibit thick, red scaly patches on the skin. Similarly, subacute cutaneous lupus manifests as red, scaly patches of skin but with distinct edges. Acute cutaneous lupus manifests as a rash. Some have the classic malar rash or butterfly rash associated with the disease. More than 90 percent of those affected will experience joint or muscle pain at some time during the course of their illness. Fewer than ten percent of people with lupus arthritis will develop deformities of the hands and feet. People with SLE may have an association with antiphospholipid antibody syndrome [22] a thrombotic disorder , wherein autoantibodies to phospholipids are present in their serum. Abnormalities associated with antiphospholipid antibody syndrome include a paradoxical prolonged partial thromboplastin time which usually occurs in hemorrhagic disorders and a positive test for antiphospholipid antibodies; the combination of such findings have earned the term " lupus anticoagulant -positive". Another autoantibody finding in SLE is the anti-cardiolipin antibody , which can cause a false positive test for syphilis. It involves either the mitral valve or the tricuspid valve. Atherosclerosis also occurs more often and advances more rapidly than in the general population. Kidneys Painless passage of blood or protein in the urine may often be the only presenting sign of kidney involvement. Acute or chronic renal impairment may develop with lupus nephritis , leading to acute or end-stage kidney failure. The histological hallmark of SLE is membranous glomerulonephritis with "wire loop" abnormalities. Neuropsychiatric systemic lupus erythematosus Neuropsychiatric syndromes can result when SLE affects the central or peripheral nervous system. The American College of Rheumatology defines 19 neuropsychiatric syndromes in systemic lupus erythematosus. Neurological disorders contribute to a significant percentage of morbidity and mortality in people with lupus. Systemic lupus erythematosus and pregnancy SLE causes an increased rate of fetal death in utero and spontaneous abortion miscarriage. One of the factors associated with SLE is vitamin D deficiency. Research indicates SLE may have a genetic link. SLE does run in families, but no single causal gene has been identified. Part of the complexity of this disease is due to the effects of both environment and genetics factors that may contribute to its development. Candidate gene loci implicated with SLE include multiple alleles from the HLA region, Fc-gamma receptor , and complement component system. Since SLE is associated with so many genetic regions, it is likely an oligogenic trait, meaning that there are several genes that control susceptibility to the disease. Drug-induced lupus mimics SLE. However, symptoms of drug-induced lupus generally disappear once the medication that triggered the episode is stopped. More than 38 medications can cause this condition, the most common of which are procainamide , isoniazid , hydralazine , quinidine , and phenytoin. SLE is triggered by environmental factors that are unknown. During an immune reaction to a foreign stimulus, such as bacteria, virus, or allergen, immune cells that would normally be deactivated due to their affinity for self-tissues can be abnormally activated by signaling sequences of antigen-presenting cells. Thus triggers may include viruses, bacteria, allergens IgE and other hypersensitivity , and can be aggravated by environmental stimulants such as ultraviolet light and certain drug reactions. These stimuli begin a reaction that leads to destruction of other cells in the body and exposure of their DNA, histones , and other proteins, particularly parts of the cell nucleus. These antibodies clump into antibody-protein complexes which stick to surfaces and

damage blood vessels in critical areas of the body, such as the glomeruli of the kidney; these antibody attacks are the cause of SLE. Researchers are now identifying the individual genes, the proteins they produce, and their role in the immune system. Each protein is a link on the autoimmune chain, and researchers are trying to find drugs to break each of those links. T cells, which regulate B-cell responses and infiltrate target tissues, have defects in signaling, adhesion, co-stimulation, gene transcription, and alternative splicing. Necrosis is increased in T lymphocytes. These cells normally engulf B cells that have undergone apoptosis after somatic hypermutation. Also, uningested apoptotic nuclei can be found outside of TBMs. This material may present a threat to the tolerization of B cells and T cells. Dendritic cells in the germinal center may endocytose such antigenic material and present it to T cells, activating them. Also, apoptotic chromatin and nuclei may attach to the surfaces of follicular dendritic cells and make this material available for activating other B cells that may have randomly acquired self-specificity through somatic hypermutation. This includes deficient phagocytic activity and scant serum components in addition to increased apoptosis. SLE is associated with defects in apoptotic clearance, and the damaging effects caused by apoptotic debris. When apoptotic material is not removed correctly by phagocytes, they are captured instead by antigen-presenting cells, which leads to development of antinuclear antibodies. Most of the monocytes and tingible body macrophages TBMs , which are found in the germinal centres of lymph nodes , even show a definitely different morphology; they are smaller or scarce and die earlier. Serum components like complement factors, CRP , and some glycoproteins are, furthermore, decisively important for an efficiently operating phagocytosis. With SLE, these components are often missing, diminished, or inefficient. Recent research has found an association between certain people with lupus especially those with lupus nephritis and an impairment in degrading neutrophil extracellular traps NETs. It leads to a progression of the apoptosis process and finally to secondary necrosis of the cells if this ability is disturbed. Increased appearance of apoptotic cells also stimulates inefficient clearance. That leads to maturation of DCs and also to the presentation of intracellular antigens of late apoptotic or secondary necrotic cells, via MHC molecules. Autoimmunity possibly results by the extended exposure to nuclear and intracellular autoantigens derived from late apoptotic and secondary necrotic cells. B and T cell tolerance for apoptotic cells is abrogated, and the lymphocytes get activated by these autoantigens; inflammation and the production of autoantibodies by plasma cells is initiated. A clearance deficiency in the skin for apoptotic cells has also been observed in people with cutaneous lupus erythematosus CLE. In some people with SLE, accumulation of apoptotic debris can be observed in GC because of an ineffective clearance of apoptotic cells. Autoreactive B cells can accidentally emerge during somatic hypermutation and migrate into the germinal center light zone. Autoreactive B cells, maturated coincidentally, normally do not receive survival signals by antigen planted on follicular dendritic cells and perish by apoptosis. In the case of clearance deficiency, apoptotic nuclear debris accumulates in the light zone of GC and gets attached to FDC. This serves as a germinal centre survival signal for autoreactive B-cells. After migration into the mantle zone, autoreactive B cells require further survival signals from autoreactive helper T cells, which promote the maturation of autoantibody-producing plasma cells and B memory cells. In the presence of autoreactive T cells, a chronic autoimmune disease may be the consequence. Antibody binding subsequently spread to other epitopes. The similarity and cross-reactivity between the initial targets of nRNP and Sm autoantibodies identifies a likely commonality in cause and a focal point for intermolecular epitope spreading. Recently, there is increasing evidence HMGB1 contributes to the pathogenesis of chronic inflammatory and autoimmune diseases due to its inflammatory and immune stimulating properties. Micrograph of a section of human skin prepared for direct immunofluorescence using an anti-IgG antibody. The skin is from a person with systemic lupus erythematosus and shows IgG deposits at two different places. The first is a bandlike deposit along the epidermal basement membrane "lupus band test" is positive ; the second is within the nuclei of the epidermal cells antinuclear antibodies are present. Several techniques are used to detect ANAs. Clinically the most widely used method is indirect immunofluorescence IF. When skin not exposed to the sun is tested, a positive direct IF the so-called lupus band test is an evidence of systemic lupus erythematosus. Subtypes of antinuclear antibodies include anti-Smith and anti-double stranded DNA dsDNA antibodies which are linked to SLE and anti-histone antibodies which are linked to drug-induced lupus. Because of this, the LE cell test is now performed only

rarely and is mostly of historical significance. The criteria, however, were established mainly for use in scientific research including use in randomized controlled trials which require higher confidence levels, so many people with SLE may not pass the full criteria. Criteria The American College of Rheumatology ACR established eleven criteria in , [73] which were revised in [74] as a classificatory instrument to operationalise the definition of SLE in clinical trials. They were not intended to be used to diagnose individuals and do not do well in that capacity. For the purpose of identifying people for clinical studies, a person has SLE if any 4 out of 11 symptoms are present simultaneously or serially on two separate occasions.

6: Lupus: Open Access- Open Access Journals

Systemic lupus erythematosus has many guises, but the unifying feature is the presence of antibodies against double-stranded DNA in almost all patients. This review provides data that show that.

Can be segmental or global. NPSLE is often a difficult diagnosis to make. In many cases, a brain biopsy would be the only definitive test, and this is rarely performed. The clinical features vary from central nervous system disease causing headache and seizures, or psychiatric diagnoses including depression and psychosis, to peripheral nervous system involvement causing neuropathy. The investigations of choice will vary with the presentation. Central nervous system disease usually warrants magnetic resonance imaging MRI of brain or spinal cord, and examination of the cerebrospinal fluid where appropriate. This included patients with focal disease clinically. Interestingly, only one of the 85 patients included in this study proceeded to brain biopsy, which is probably indicative of generally accepted practice. The frequency of musculoskeletal disease in SLE means that rheumatologists often make the initial diagnosis. Arthralgia and myalgia occur in most patients. A rheumatoid-like arthritis is seen more rarely, sometimes associated with a positive rheumatoid factor. Similarly, an overlap with myositis also occurs. Skin involvement in lupus is also very common. In addition to the classic malar and discoid rashes, more generalized photosensitivity is often present, and furthermore sun exposure is known to trigger systemic disease flares. Alopecia can be scarring when associated with discoid lesions, or more diffuse, often fluctuating with disease activity. Recurrent crops of mouth ulcers are also a feature of active disease. Haematological features include normocytic normochromic anaemia, thrombocytopaenia sometimes, but not always associated with antiphospholipid antibodies and leukopaenia. Severe haematological disease can occur, but this is relatively rare [13]. Pleuritis , causing chest pain, cough and breathlessness, is the most common pulmonary manifestation of SLE [14]. Although pleuritic symptoms may relate directly to active lupus, pulmonary embolism must always be considered, particularly in those who have antiphospholipid antibodies. Pleural effusions are usually exudates, have low levels of complement, and test positive for anti-nuclear antibodies ANA. Infections are common, and any parenchymal lesion must be treated as infectious until proven otherwise. Rarer complications include interstitial lung disease and pulmonary hypertension both more common in systemic sclerosis and pulmonary haemorrhage. Gastrointestinal involvement [15] most commonly results in non-specific abdominal pain and dyspepsia though it can be unclear whether such pain results from the illness itself or from drug side-effects. Hepatosplenomegaly can come and go with disease activity. Mesenteric vasculitis is very rare, but can be life-threatening, especially if it leads to perforation, and may only be diagnosed at laparotomy. SLE is associated with a variety of vascular manifestations. Abnormalities in the micro vasculature are also thought to account for the association with livedo reticularis. In the last decade, it has become clear that patients with SLE are at increased risk of atherosclerosis. Chronic inflammation and the use of corticosteroids contribute to this risk, and have led rheumatologists to treat SLE as an independent risk factor for stroke and myocardial infarction, much as an endocrinologist might regard the risk associated with diabetes. Ward [16] showed that in women between 18 and 44 years of age, those with SLE were twice as likely to develop a myocardial infarction or stroke, and nearly 4 times as likely to present with heart failure. Screening for cardiac disease with echocardiography ECHO has established that asymptomatic valvular lesions are common. In addition, pericarditis and pericardial effusions are common though myocardial disease is relatively rare. Significant titres are accepted to be of 1: A positive ANA is also seen in many other illnesses including systemic sclerosis and polymyositis, as well as some chronic infections. All patients should be screened for extractable nuclear antigens ENA. Antibodies to double-stranded DNA dsDNA , and more recently to nucleosomes though this test is not commonly available in most routine labs are more specific for SLE, and anti-dsDNA titres are also predictive of renal involvement. Moreover the titres of these antibodies fluctuate with disease activity and therefore serial testing is a useful monitoring tool. Typically, a disease flare is accompanied by a rising titre of dsDNA antibodies and erythrocyte sedimentation rate ESR , and falling complement and lymphocyte count. Treatment SLE is a relapsing and remitting disease, and treatment aims are threefold: Our limited

understanding of the precise pathogenesis of SLE means that the majority of treatments are still broadly immunosuppressive in action, and hence carry a significant risk of adverse effects. At the milder end of the spectrum, hydroxychloroquine is commonly used. This is effective for skin disease, joint pain and fatigue. Non-steroidal anti-inflammatory drugs are also useful for arthralgia and arthritis, though more aggressive treatment with methotrexate may be required. Low dose oral steroids or intramuscular injections of depot steroid preparations are sometimes used for mild disease, but immunosuppressive therapies and high dose steroids are generally reserved for major organ involvement. Lupus nephritis remains the complication which carries with it the biggest risk of death or long-term morbidity. Treatment of renal disease Cochrane review [17] was standardized by the National Institute of Health guidelines [18] published in Combining high dose corticosteroids with cyclophosphamide was the gold standard in the management of proliferative lupus nephritis for many years. Although efficacious, this regimen is limited by significant toxicity. Both agents are immunosuppressive. In addition, corticosteroids are associated with a whole host of adverse effects including osteoporosis and weight gain, and cyclophosphamide can cause haemorrhagic cystitis and infertility. More recently, the classic regimen of monthly boluses of 1g cyclophosphamide for 6 months, followed by once every three months for the next 2 years, has been modified by some groups, who instead advocate the use of "low-dose" cyclophosphamide 6 fortnightly pulses of mg. The so-called Euro-lupus trial, published in , showed that the use of this lower dose regimen has better outcomes in terms of infertility risk, with no deleterious impact on renal disease [19]. Following remission induction, azathioprine is commonly used for maintenance therapy. Mycophenolate mofetil [20] has been added to the repertoire of drugs used for the treatment of lupus nephritis. This is now used commonly as maintenance therapy following cyclophosphamide, and its use in the induction phase has been adopted in some centres. Similarly, immunosuppressive treatments, such as cyclophosphamide and azathioprine, are also used for central nervous system involvement and rarely, serositis and haematological disease. Furthermore, persistent autoimmune thrombocytopenia sometimes requires immunoglobulin. In an attempt to improve management, biological therapies are being developed, which target specific cells or molecules within the abnormally functioning immune system. For example, the depletion of B cells using rituximab, an anti-CD20 monoclonal antibody previously used in the treatment of B cell lymphomas, is now being used in patients with severe disease which has not responded to conventional treatments [21]. Prognosis Despite significant advances in treatment over the last decade, SLE still caries a significant risk of mortality and long term morbidity. Mean age at death was 44, but varied widely from 18â€”81 years. Cause of death varies with disease duration. In one cohort [22] , renal lupus accounted for the biggest number of deaths in those with less than 5 years of disease, whereas vascular disease was the most important factor in the group who died later in the disease course. As mentioned previously, we are becoming increasingly aware of the impact that premature atherosclerosis is having on the long term prognosis of lupus patients who survive the early years of illness. As we develop better immune targeted therapies, optimizing the management of these longer term complications will become increasingly important. The revised criteria for the classification of systemic lupus erythematosus. Updating the American College of Rheumatology revised criteria for the classification of systemic lupus erythematosus. The prevalence and incidence of systemic lupus erythematosus in Birmingham, England. Relationship to ethnicity and country of birth. Prevalence of physician-diagnosed systemic lupus erythematosus in the United States: J Womens Health Larchmt ; The pathogenesis of systemic lupus erythematosus. Genetics of human systemic lupus erythematosus: The prevalence and associations of fatigue in systemic lupus erythematosus. The classification of glomerulonephritis in systemic lupus erythematosus revisited. J Am Soc Nephrol. Value of MRI of the brain in patients with systemic lupus erythematosus and neurologic disturbance. Prevalence, patterns of disease and outcome in patients with systemic lupus erythematosus who develop severe haematological problems. Pulmonary disease in systemic lupus erythematosus and the antiphospholpid syndrome. A review of gastrointestinal manifestations of systemic lupus erythematosus. Premature morbidity from cardiovascular and cerebrovascular diseases in women with systemic lupus erythematosus. Treatment for lupus nephritis. Cochrane Database Syst Rev. Controlled trial of pulse methylprednisolone versus two regimens of pulse cyclophosphamide in severe lupus nephritis. Immunosuppressive therapy in lupus nephritis:

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N Engl J Med. An open study of B lymphocyte depletion in systemic lupus erythematosus. Outcome of a cohort of patients with systemic lupus erythematosus attending a dedicated clinic for over two decades.

7: Diagnosis of Systemic Lupus Erythematossus - - American Family Physician

Systemic lupus erythematosus (SLE) is a prototypic autoimmune disease characterised by the production of antibodies to components of the cell nucleus in association with a diverse array of clinical manifestations.

8: Systemic lupus erythematosus - Wikipedia

Systemic lupus erythematosus (SLE) is an autoimmune disease with a prevalence of / in Germany and a female/male ratio of The clinical course is variable, with a broad spectrum of organ manifestations; lupus nephritis develops in about half of all patients.

SYSTEMIC LUPUS ERYTHEMATOSUS JOURNAL pdf

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