Systemic lupus erythematosus test questions essay

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A multisystem autoimmune disease whose etiology is unknown, systemic lupus erythematosus (SLE) involves a diverse and exaggerated autoantibody response as well as targets various autoantigens of nucleoprotein particles of ribosomes, DNA (nucleosomes), cytoplasmic RNPs (SS-A/Ro) and RNA-small nuclear ribonucleoproteins (snRNPs) (Lerner & Steitz, 1979).

Likewise, antibodies are directed in opposition to negatively-charged phospholipids (Lerner & Steitz, 1979). An outstanding characteristic of the various autoantigens in lupus is the deficiency in restriction to any subcellular location (Casciola-Rosen et al. 1994). Trademarks of SLE are the antibodies to nuclear components identified as macromolecular assemblies or macromolecules (Lerner & Steitz, 1979).

The general prevalence of SLE is estimated to occur in 100 for every 100, 000, is estimated to be three times more frequent among African-Americans than in Caucasians and happens 10 times more frequently in women (Smeltzer & Bare, 2003). A study found that SLE patients had a 4. 92-fold increased risk to die versus the general population with survival rates that continue to improve although causes of mortality vary at various stages (Abu-Shakra et al. 1995).

SLE Pathology SLE is a disease involving the development of an immune response to intact nuclear Ags (Arbuckle et al. , 2003). Apoptosis—or the abnormal programmed cell death—of lymphocytes in SLE may offer a source of extracellular nuclear Ag to force the immune response and to enable the development of immune complexes (Emlen et al. , 1994). Disturbed immune

regulation leads to an increased autoantibody production adds directly to the pathologic changes of SLE (Arbuckle et al.

, 2003). This immunoregulatory disturbance is due to a number of blending of hormonal, genetic and environmental factors (e. g. , thermal burns, sunlight), while procainamide, apresoline, isoniazid, antiseizures and chlorpromazine are the medications that have been implicated in drug-induced SLE (Smeltzer & Bare, 2003). The increase in autoantibody production is considered to be due to abnormal suppressor T-cell function resulting in tissue damage and immune complex deposition. Inflammation stimulates antigens, which then kindle additional antibodies and the cycle repeats (Smeltzer & Bare, 2003).

SLE can remain undiagnosed for a couple of years because the start of the disease is acute or insidious. Clinical attributes entail multiple body systems —as it is the circulatory system that transports the disease all over the body (Smeltzer & Bare, 2003). The inflammatory processes influence the integument which is observable by either the presence of plaques and scaling causes scarring and pigmentation changes (discoid lupus erythematosus), chronic rash with erythematous papules, or papulosquamous or annular polycyclic lesions (subacute cutaneous lupus erythematosus) (Smeltzer & Bare, 2003). The most well-known skin symptom is an acute cutaneous lesion, butterfly-shaped rash across the cheeks and bridge of the nose.

Lesions typically worsen during intensification of the systemic disease, which can be aggravated by artificial ultraviolet light or sunlight (Smeltzer & Bare,

2003). Oral ulcers at the hard palate and mucosa happen in crops. Involvement of the musculoskeletal system, like arthralgias, arthritis and synovitis, is marked by pain on movement accompanied by morning stiffness, tenderness and joint swelling (Smeltzer & Bare, 2003). Fibrinoid degeneration of valve cusps, verrucous vegetations, vasculitis, fibrotic scarring, the antiphospholipid antibodies, valvulitis and rupture of chordae tendineae cause the pathogenesis of valvular heart disease (Boumpas et al. , 1995). The range of lupus-related valvulopathies has been enlarged to incorporate valve leaflet thickening with or without lesions of Libman-Sacks endocarditis and valve dysfunction (Boumpas et al.

, 1995). Valvular abnormalities may lead to hemodynamically considerable lesions that need valve replacement (Boumpas et al. , 1995). Pericarditis is common (Smeltzer & Bare, 2003).

Atherosclerosis involves numerous risk factors, such as hyperlipidemia, hypertension and obesity. Pulmonary participation like acute lupus pneumonitis and alveolar hemorrhage is due to acute injury to the alveolar-capillary unit. Acute lupus pneumonitis features the patchy alveolar infiltrates on chest radiography without evidence of an underlying infection, dyspnea with hypoxemia and abrupt onset of fevers (Smeltzer & Bare, 2003). The alveolar hemorrhage syndrome exhibits analogous symptoms, with the exception of that it also involves decreased hemoglobin levels due to bleeding inside the lungs (Boumpas et al. 1995). It has been hypothesized that the elevated levels of complement split products detected in the plasma of patients may trigger circulating neutrophils which amass inside the

pulmonary vasculature—which explains for the observed decreased oxygenation capacity that cause acute reversible hypoxemia (Boumpas et al.

, 1995). Pulmonary hypertension is considered to be due to vascular occlusion caused by parenchymal lung disease, vasoconstriction, platelet aggregation or thrombosis, and vasculopathy or vasculitis (Smeltzer & Bare, 2003). Hematologic disease—such as autoimmune thrombocytopenia—is due to antiplatelet autoantibodies' binding to one or more surface glycoproteins (Boumpas et al. , 1995). The antibody-coated platelets are eaten by macrophages of the bone marrow, liver, spleen, and lymph nodes, which carry receptors for the Fc region of immunoglobulin.

Localization of immune complexes in the kidney seems to be the stimulating occurrence for the growth of lupus nephritis (Smeltzer & Bare, 2003). Cross-reactivity of anti-DNA autoantibodies with glomerular cell surface antigens, with normal components of basement membrane and mesangial matrix, supports glomerular immune complex formation and causes the location of these deposits within the glomerulus (Boumpas et al. , 1995). Therefore, factors that cause the settling of various proinflammatory immune complexes within the subendothelial region of the glomerular capillary wall, next to the circulation, can possibly stimulate cellular proliferation, an inflammatory response, necrosis, and finally fibrosis. Moreover, a subset of autoantibodies may infiltrate glomerular cells, combine to nuclei, and participate in glomerular proliferation and proteinuria (Smeltzer & Bare, 2003). Multifocal cerebral cortical microinfarctions related with microvascular injury are the principal histopathologic abnormalities attributed to neuropsychiatric lupus that incites vascular occlusion due to vasculitis,

thrombosis, vasculopathy, antibody-mediated neuronal cell injury or dysfunction, and leukoagglutination (Boumpas et al.

, 1995). Antibodies may gain entry to the CNS from a blood-brain barrier disturbed by the vascular injury or may be produced intrathecally—which shows several degrees of apathy, memory impairment and behavioral change; loss of intellect or judgment, orientation, agitation, stupor, delirium, or coma are seen in extreme instances (Smeltzer & Bare, 2003). Dementia may be brought about by active ongoing neuropsychiatric lupus, which may result from multiple infarctions due to antiphospholipid antibodies (Boumpas et al. , 1995). Assessment and Diagnostic FindingsSLE diagnosis is founded on blood tests, physical examination and a complete medical history (Mills, 1994). Inspection of the skin is made for hyperpigmentation or depigmentation, erythematous rashes and plaques, and sunlight sensitivity (Smeltzer & Bare, 2003).

The scalp must be checked for alopecia, while the mouth and throat must be checked for ulcerations (Mills, 1994). Cardiovascular assessment includes auscultation for pericardial friction rub, likely related with pleural effusions and myocarditis (Smeltzer & Bare, 2003). Pleural infiltrations and effusions—which suggest respiratory insufficiency—are manifested by abnormal lung sounds and are visible on CXR (Mills, 1994). Purpuric lesions, erythematous and necrotic papular indicate vascular involvement. Physical examination can detect edema, tenderness, joint swelling, warmth, stiffness, and pain on movement. The classic symptoms include fatigue, weight loss and fever, and even pericarditis, arthritis, and pleurisy (Smeltzer & Bare, 2003).

Neurologic assessment is made to determine changes in behavior and CNS (Mills, 1994). Blood testing can determine positive antinuclear antibodies, leukocytosis or leucopenia, anemia, and thrombocytopenia, while urinalysis may detect creatinine and hematuria (Smeltzer & Bare, 2003). Medical Management The aims of treatment include preventing therapy-related complications, minimizing the risk of acute disease, avoiding progressive loss of organ function, and reducing disease-related disabilities (Boumpas et al, 1995). SLE management entails routine monitoring to evaluate effectiveness of the therapeutic as well as disease activity; medication therapy for SLE is founded on the idea that local tissue inflammation is interceded exaggerated immune responses (Smeltzer & Bare, 2003).

The NSAIDs employed for minor clinical manifestations are typically used together with corticosteroids—the single most valuable available drug for SLE (Boumpas et al, 1995). To effectively manage mild systemic, musculoskeletal and cutaneous features of SLE, antimalarial drug is used (Smeltzer & Bare, 2003). Immunosuppressive agents (e. g. purine analogs and alkylating agents) are also utilized due to their effect on immune function (Boumpas et al, 1995; Smeltzer & Bare, 2003). Nursing Management Generally, individuals with SLE experience problems on body image disturbance, comfort, impaired skin integrity, fatigue, and lack of knowledge to make self-management decisions (Doenges & Moorhouse, 1991).

The disease or its management may cause significant distress and considerable changes in appearance in the patient. Support groups can help patients through the various information, management tips and social

support that they provide (Smeltzer & Bare, 2003). Inform patients to protect themselves with clothing and sunscreen and keep away from exposure (Doenges & Moorhouse, 1991). With CVD risk, a dietary consultation is indicated for dietary recommendations. The nurse educates the patient regarding the value of continuing prescribed medications and deals with the side effects and changes (Doenges & Moorhouse, 1991). Due to the increased risk for systemic involvement, patients must recognize the need for regular periodic screenings, monitoring as well as health promotion exercises (Doenges & Moorhouse, 1991; Smeltzer & Bare, 2003).