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Medical and Psychiatric Features of Systemic Lupus Erythematosus

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Lupus erythematosus was first described by the French dermatologist Biett in 1828 and was so named because its classic skin eruptions resembled the mark of a wolf bite.[1] In 1875, Hebra and Kaposi[2] noted internal organ involvement and mental changes in conjunction with the traditional lupus skin lesions. Sir William Osler was the first to note that systemic lupus erythematosus (SLE) could cause internal organ involvement in the absence of skin eruptions. In 1900, he reported neurologic changes, hemiplegia, and aphasia as well.[3]

Since the description of the LE cell in 1948,[4] the number of cases of systemic lupus diagnosed each year has increased steadily. The American Lupus Society estimates that there may be as many as 200,000 people in the United States with SLE.[5] Blau[6] believes the true number of cases may be closer to one million.

SLE is currently classified as a rheumatic disease and is usually thought of as a chronic, multisystem, inflammatory disorder of connective tissue, whose course is characteristically punctuated by exacerbations and remissions. It occurs in both sexes and all age groups, with the most frequent victims being women between the ages of 20 and 40. It is also seen in all races and in all parts of the world.

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ETIOLOGY

The etiology of SLE is unknown; however, infection or exposure to sunlight or certain medications may precipitate its onset. Although it is not classified as a hereditary disorder, there is an increased incidence of connective tissue disorders and abnormal serologic factors among the relatives of SLE patients.[5,7-10]

The American Lupus Society estimates that as many as 10% of all cases of disseminated lupus are drug induced.[5] A review of the literature reveals considerable controversy regarding the role of medications in these cases, i.e., whether they are causative agents or merely precipitate the onset of a previously latent disease process.[11-15]

CLINICAL PRESENTATIONS

The mode of onset of SLE varies dramatically and provides a definite prognostic sign. Acute onset SLE represents approximately 20% of cases. Of these, more than 50% of patients die within 3 years.[11] An insidious onset, which may delay diagnosis for years, is the most common presentation of the disease. In fact, many mild cases of SLE may never be detected.

The early signs of systemic lupus are often vague and fleeting. Most frequently, the disease presents with fatigue, lassitude, fever (particularly low-grade afternoon elevation), weakness, anorexia, weight loss, and/or intermittent joint pain. Because of the vagueness and variability of these complaints, it is not uncommon for the lupus patient to be labeled "psychoneurotic." Following medical rebuffs and labeling, the patient may undertake a round of "doctor shopping" in an effort to obtain relief, only to be confronted with more frustrations and increasing self-doubt if the initial psychiatric diagnosis is upheld. One SLE patient summed it up well when she said, "I jumped up and down in the parking lot when I was finally diagnosed. It may sound funny that I was happy to discover that I was sick, but I could finally put a name on what was making me feel so lousy."

Because of both the multiplicity of the symptoms seen and the variability of the organ systems involved, the American Rheumatism Association has adopted specific diagnostic criteria for systemic lupus.[16] The diagnosis should be considered if 4 of the following 14 signs are present in a given patient:

1. Facial erythemia (butterfly rash)
2. Hemolytic anemia, leukopenia, or thrombocytopenia
3. Photosensitivity
4. Chronic false positive serologic test for syphilis
5. Alopecia

6. Psychosis or convulsions
7. Cellular casts in urine
8. Profuse proteinuria (> 3.5 mg/day)
9. Oral or nasopharyngeal ulceration
10. Discoid lupus
11. LE cells
12. Arthritis without deformity
13. Pleuritis or pericarditis
14. Raynaud's phenomenon

The clinical presentation of drug-induced lupus closely resembles that of the natural disease. In the drug-induced variety, however, symptoms normally disappear following discontinuation of the causative medication, and no further recurrences are noted unless the patient is again challenged by the drug. The drugs currently known to precipitate a lupus-like syndrome include penicillin, hydralazine, diphenylhydantoin and other anticonvulsants, penicillamine, quinidine, phenothiazines, reserpine, thiouracils, procainamide, sulfonamides, phenylbutazone, tetracyclines, isoniazid, and birth control pills.[11,14,15,17-20]

In their study of drug-induced lupus, Siegel, et al[19] found that females are more susceptible than males by a ratio of 2 to 1. Drug-induced lupus occurred more frequently in older age groups. The average age of patients with drug-induced lupus was 47.4 years, compared to 35.8 years for patients with naturally occurring SLE.

The differentiation of drug-induced lupus from the spontaneously occurring variety relies heavily on an accurate and complete history of both prescribed and over-the-counter medications consumed and the patient's reason for taking these medications.

LABORATORY FINDINGS

Although 80% of patients with disseminated lupus will have LE cells present at some point during the acute phase of their illness,[10,11,13] the cells may be present only intermittently, and their number cannot be correlated accurately with the degree of lupus activity.[10] It is important to remember that LE cells occur in a number of disease states, such as other types of connective tissue disease, hepatitis, ulcerative colitis, and drug reactions. Until recently, it was thought that the test for antinuclear antibodies (ANA) was positive in virtually all SLE patients tested by immunoflourescent methods.[15] However, Dubois (personal communication to American Lupus Society, April 1979) found that approximately 5% of his SLE patients had both negative ANA titers and LE cell tests, even though they exhibited overwhelming clinical evidence of SLE

and responded well to treatment.

A false positive serologic test for syphilis occurs in 20%[13] to 50%[11] of all SLE patients. However, *Treponema pallidum* immobilization and specific treponema antibody tests are negative in these patients. These false positive serologic tests for syphilis may confuse the diagnostic picture and may be present for years before other clinical signs of SLE appear.

Mild to moderate anemia is seen in 75%–80%[11,13] of SLE patients. Such anemias are usually unresponsive to traditional treatment. Leukopenia is quite common during all phases of the illness. A white count below 4,000/mm^3 is frequently found. Therefore, one must remember that an infection may be present when the SLE patient who normally runs a low white count (3,000–4,000) suddenly shifts to the normal range (8,000–9,000).

SLE may also present as "idiopathic" thrombocytopenic purpura. When this occurs in conjunction with splenomegaly, the physician should suspect disseminated lupus. The sedimentation rate remains high in nearly all SLE cases, even during periods of remission.[10]

Liver function tests are frequently normal. Serum globulin, particularly the gamma fraction, is often elevated. The BSP excretion test is recommended for evaluation of liver function, as alterations in serum proteins may cause cephalin flocculation and thymol turbidity tests to be positive, even in the absence of existing liver disease.

The presence and extent of protein, WBCs, RBCs, and casts in the urine reflect the type and degree of renal involvement.[14]

ORGAN SYSTEM INVOLVEMENT

Volumes have been written regarding the manifestations of systemic lupus erythematosus; the following is intended as a brief overview of the specific types of organ system involvement which occur.

Skin

Skin changes are seen in 78% to 85% of all SLE cases,[10,13] the most common changes being cutaneous lesions. Skin involvement may be minimal, confined to patchy erythema on the extremities, particularly the back of the fingers and toes, or it may be extensive, affecting all areas exposed to sunlight.[11] Photosensitivity is a frequent historical complaint; in such cases, exposure to sunlight results in pain, itching, and redness of the exposed areas. The butterfly mask, an erythematous rash over the cheeks and across the nose, which is often considered the sine qua non of lupus, appears in only one-third of cases.[13] Ten percent of patients who initially manifest classical discoid lupus develop SLE at some later point in time.[10,21]

Raynaud's phenomenon often precedes other signs of SLE by several years. Purpura may appear with or without thrombocytopenia.[22] SLE patients may also experience a thinning of scalp hair and coarsening of the hair that remains.

These skin changes and the resultant distortion of body image may contribute to the appearance of emotional symptoms. Distortions of body image and lethargy, coupled with marked fatigability and enforced limitation of activity, often produce episodes of depressed mood.

Kidney

Significant renal impairment represents one of the least favorable prognostic signs. Once renal function is compromised, the patient usually begins a course of progressive deterioration.[13]

Significant histologic renal changes occur even in SLE patients who have relatively benign clinical findings.[10] Glomerulitis is the most common renal lesion of SLE. If episodes of glomerulitis occur before a diagnosis of systemic lupus is made, the patient may be thought to be suffering from a primary glomerulonephritis.

Hypertension, which frequently occurs in conjunction with renal impairment or the failure of the kidney to adequately clear its by-products, may cause or contribute to the development of an organic brain syndrome. The antihypertensives used to treat these patients, particularly reserpine, guanethidine, and alphamethyldopa, may themselves produce depression or acute confusional states.

Pulmonary

Lung involvement is frequent and may be directly related to the SLE or occur secondary to bacterial or fungal infection. Basilar pneumonitis and/or pleuritis is reported in up to 40% of SLE patients.[13] Regardless of type, the predominant characteristic of lupoid lung involvement is its persistence over long periods of time. Episodes of pleuritic pain may be transient and occur in the absence of other signs of an active disease process. The pain is often disproportionate to the findings and may result in an erroneous diagnosis of functional disorder.[10] This is an important point for the consultation-liaison psychiatrist to bear in mind. In the presence of underlying secondary infection, patients may experience toxic delirium. Panic or acute anxiety attacks often occur in conjunction with episodes of dyspnea which appear disproportionate to the patient's physical signs.

Cardiac

Pericarditis, myocarditis, or endocarditis may occur in association with SLE. Inflammatory pericarditis occurs with the greatest frequency; however, in such cases, bacterical pericarditis must be ruled out. Inflammatory myocarditis often presents with tachycardia, gallop rhythm, and cardiomegaly, and may thus mimic acute congestive heart failure or, if fever is present, acute rheumatic fever. Abacterial cardiac vegetations may occur.[23] When present, these most frequently involve the mitral valve; however, they usually do not cause significant valvular malfunction.

Organic brain syndromes commonly occur in those patients whose cardiac disease is severe enough to compromise cerebral blood flow. Cerebral anoxia may also produce what appears to be acute and/or chronic anxiety states. These are particularly common in those patients who develop paroxysmal atrial tachycardia. Clinically significant depressive episodes, which may reach psychotic proportions, are also frequently associated with the acute myocarditis of lupus.

Musculoskeletal

Joint involvement is the most frequently encountered symptom in patients with SLE. Dubois and Tuffanelli[24] found such involvement in 91.5% of the 520 patients they studied. Arthritis is often the earliest sign of lupus and may be the only abnormality noted for years. In such cases, patients are often diagnosed as having primary joint disease. The American Lupus Society has suggested that many cases of systemic lupus are initially misdiagnosed as rheumatoid arthritis.[5] The arthritis associated with SLE most commonly involves the joints of the hands, wrists, elbows, shoulders, knees, and ankles. Severe erosive arthritis is rare. The discomfort associated with arthritis is usually transient (i.e., 1 or 2 days' duration) and may be migratory in nature. Because of the variability of the arthritic complaints and their disproportionate pain, these patients are frequently suspected of exaggerating their complaints, malingering, or suffering from a functional disorder. Once the patient is labeled as histrionic or neurotic, other symptoms are frequently discounted and the correct diagnosis is further delayed.

Gastrointestinal

Any area of the gastrointestinal tract may become involved. Ulcerative lesions of the mucous membranes, particularly those of the mouth, are most common. Esophageal lesions which result in the development of dysphagia are easily confused with those of scleroderma. Other common GI complaints include anorexia, nausea and vomiting, diarrhea, abdominal pain, and bloody

stools. Mucosal alterations of the stomach and small intestine may ultimately result in ulceration or perforation, massive hemorrhage, or severe focal abdominal pain.[13] These widespread mucosal changes may also produce a malabsorption syndrome. This syndrome, with its resultant fluid and electrolyte loss, may be responsible for the development of an acute organic brain syndrome.

Liver, Spleen, and Lymph Nodes

Hepatomegaly, which is fairly common in SLE patients, usually occurs as the result of fatty infiltration of the liver. Unlike other patients with fatty liver infiltrates, however, SLE patients rarely go on to develop cirrhosis.

The clinician should not be confused by the term "lupoid hepatitis," which was so named because it most commonly occurs in young women in association with facial eruptions, arthralgia, myalgia, signs of renal impairment, plural effusion, hemolytic anemia, or thombocytopenia, and thereby mimics disseminated lupus.[14] At autopsy, however, these patients do not demonstrate the characteristic histologic alterations of SLE.

The fever, lymphadenopathy, splenomegaly, and anemia which occur with lupus may suggest a diagnosis of lymphoma.[12] Lymph node enlargement, when present, is most frequently found in the neck and axilla.

Eye

Cytoid bodies, associated with vasculitis of the central nervous system, occur in 10% to 20% of patients with SLE.[10] Their presence, in the absence of essential hypertension or diabetes mellitus, is virtually diagnostic of systemic lupus.[13] The associated vasculitis often causes a rapidly developing dementia or schizophreniform psychosis.

Salivary, Thyroid, and Adrenal Glands

Approximately one-third of SLE patients develop Hashimoto's thyroiditis,[13] which by itself may produce psychiatric manifestations ranging from mild depression to frank psychosis.[25,26] Characteristically, these patients report lability of mood and periods of extreme fatigue which alternate with episodes of tension and "pressured behavior." Paranoid delusions and hallucinations frequently occur when thyroid levels change suddenly.

Sjögren's syndrome also occurs in conjunction with SLE. Characteristically, these patients complain of severe dryness of all mucous membranes. Hypofunction of the lacrimal and parotid glands is associated with weakness, fatigue, muscular aches, and chronic polyarthritis. The presence of DNA antibodies differentiates the SLE patient with a Sjögren's-like syndrome from

those with Sjögren's syndrome associated with some other disorder.

Histologic changes of the adrenal glands (vasculitis and necrosis) resulting in adrenal insufficiency have been reported in lupus patients, but are rare. When present, adrenal insufficiency may produce a psychiatric picture of depression and lethargy preceded by a period of profound fatigue and muscular weakness.

CENTRAL NERVOUS SYSTEM INVOLVEMENT

Central nervous system abnormalities are the result of arteritic lesions of varying extent.[10] Therefore, there is no typical pattern, and many presentations, such as psychosis, organic brain syndrome, seizures, cerebrovascular accidents, transverse myelitis, and peripheral neuropathies, may be seen.[6] In their study of 520 SLE patients, Dubois and Tuffanelli[24] reported CNS involvement in 25.5%, seizures in 13.8%, psychosis in 12.1%, peripheral neuritis in 11.7%, and cerebral spinal fluid pleocytosis in 8.5%.

Numerous other neurologic signs and symptoms occur. Clark and Bailey[27] reported the following CNS manifestations: convulsions, hemiplegia, diplopia, choked discs, polyneuritis, subarachnoid hemorrhage, nystagmus, vertigo, choreiform movements, monoplegia, paraplegia, quadraplegia, aphasia, intention tremor, Bell's palsy, cortical blindness, and decerebrate states. Peripheral neuropathies associated with myositis and myopathy may present with paresthesias, weakness, pain, tenderness, or inability to use the hands.[11]

Central nervous system involvement ranks second only to nephritis as a cause of death among SLE patients. Mental disturbances and frank psychoses are reported as one of the most common manifestations of such impairment.[13]

Richardson[11] has suggested that seizures are the most common symptoms of CNS lupus. Such seizures may be either focal or generalized[6] and can precede other signs of SLE by years. In these cases, the seizures are often misdiagnosed as primary manifestations of idiopathic epilepsy. The electroencephalogram has not proven to be a clinically useful tool for predicting the long-term CNS effects of lupus. EEG abnormalities occur in 50%–60% of all SLE patients who experience seizures;[6] however, similar EEGs also occur in SLE patients with no history of seizures. To further complicate the picture, Stern and Robbins[28] have described SLE patients with clinically manifest brain damage whose EEGs were normal.

PSYCHIATRIC MANIFESTATIONS

The psychiatric presentations of SLE are variable and include such symptoms as anxiety (mild to severe), memory impairment, lability of mood, personality change, depression (mild to severe), mental deterioration, hallucina-

tions, paranoia, obsessive reactions, and frank psychosis. The theories explaining these symptoms are as numerous as the symptoms themselves. Marked differences exist for even such straightforward information as the incidence of SLE-related psychoses, variously reported to range from 9%[29] to 52%.[30] There is overall agreement among researchers, however, that two basic forms of SLE psychosis exist: those which are related to the disease itself and those which occur secondary to its treatment.

Guze[31] found a higher incidence of psychiatric disorders among SLE patients than would be expected in a general medical population. Of the 101 patients he studied, 12% required psychiatric intervention; 8% required psychiatric hospitalization. Although the etiology of these psychiatric disorders was uncertain, Guze hypothesized that psychiatric symptoms resulted from lupus-induced changes in brain structure or chemistry. He also suggested that lupus might exacerbate and make manifest a latent psychiatric disorder. The psychiatric syndromes he encountered varied but, in general, could be classified as organic brain syndromes, affective illnesses, or schizophreniform disorders.

In his study of 40 SLE patients, O'Connor[32] rated only 14 as psychologically healthy. Psychotic episodes, classified as either acute brain syndromes or functional psychoses (schizophrenia or psychotic depression), were seen in 21 patients; 5 patients were considered neurotic and were diagnosed as environmentally anxious or dissociated. O'Connor felt that the development of psychiatric problems in these patients could not be related to a single factor, but was influenced by diffuse organic brain damage, high-dose steroid therapy, and psychogenic issues such as a past history of personality disorder or acute emotional trauma.

Stern and Robbins[23] found 26 of 53 lupus patients to be psychotic. Patients experiencing psychotic episodes were categorized as suffering from organic mental syndromes (OMS), schizophrenia, unclassified psychoses, psychotic depression, or steroid-induced psychoses. The organic mental syndromes were further subcategorized into pure and mixed types. The pure OMS presented with memory loss, confusion, difficulty with orientation and calculation, and vague delusional/behavioral disturbances. These patients responded well to aggressive steroid treatment. A direct correlation existed between the remission of physical indicators of lupus activity and the clearing of psychiatric symptoms. The mixed organic mental syndrome presented with florid hallucinations and bizarre delusions, in addition to the previously mentioned signs of OMS. These patients did not respond uniformly to steroid therapy. Their mental impairment persisted, even when other indicators of lupus activity suggested that their disease had remitted. In the "schizophrenic" group, psychosis persisted in the absence of other signs of active SLE. Major tranquilizers were useful in treating psychotic, agitated patients. Patients with psychotic depressions responded best to supportive psychotherapy in conjunc-

tion with steroid treatment.

The steroid psychosis also frequently presented as an organic mental syndrome. Its differentiation from the lupus-induced OMS was based on the relationship between the onset of psychiatric symptoms and the institution of steroid therapy. The steroid psychosis most characteristically occurred in patients who had received high doses (i.e., exceeding 40 mg/day of prednisone or the equivalent). These patients showed no consistent pattern of response to treatment.

It is noteworthy that 8 of the 27 nonpsychotic patients in the Stern and Robbins study were felt to be significantly depressed; 3 required long-term psychiatric treatment.

No definite "lupus susceptible" personality has been established. However, Stern and Robbins[28] found that their organic patients with mixed features and their schizophrenic patients had certain similar premorbid personality constellations. They tended to be rigid and obsessive, with a history of extensive social impairment. Their premorbid personalities were characterized by suspiciousness, shyness, and chronic anxiety. Hall et al[33] were unable to replicate these findings in their study of 56 lupus patients.

If psychiatric symptoms emerge during a course of steroid therapy, these drugs must be considered as a possible cause. In attempting to differentiate between lupus-related and steroid-induced psychoses, the specific presenting symptoms may be of little help, as both conditions frequently present as spectrum psychoses.[34] Signs and symptoms of many diverse primary psychiatric disorders (schizophrenia, panic states, depression, OMS, etc.) routinely appear. Characteristic of the steroid psychoses are marked and rapid fluctuations of symptoms, affective state, and degree of cognitive impairment. Glaser[35] suggests that lupus patients who develop psychiatric problems while receiving steroids may actually have a more severe form of the disease and thus a poorer prognosis. Other investigators[33,34] have been unable to substantiate this claim and have suggested that an episode of steroid psychosis does not seem to affect either outcome or clinical state.

A psychotic episode due to SLE is most likely to occur during an acute exacerbation of the illness, while the steroid psychoses are most likely to occur shortly after steroids are instituted or the dose is increased. This distinction has value in suggesting the correct diagnosis.

In cases of lupus-induced psychoses, *insufficient* doses of steroids may in fact contribute to the appearance of psychiatric symptoms as a result of inadequate control of the disease. A history of previous steroid psychosis does not predispose patients to an increased incidence of such reactions when steroids are readministered at a later date.[28,31,34] It is noteworthy that, due to the selectivity of steroid prescription and the increased awareness of the potential side effects of the drugs, the incidence of steroid psychoses in the general hospital

population has declined. In contrast, little or no decrease in the frequency of lupus-related emotional disturbances has been noted.[32]

Treatment-related psychiatric symptoms must also be considered if patients are being given antimalarials or large doses of aspirin. Although rare, the side effects of the 4-aminoquinolines, such as chloroquine, include lassitude, nervousness, irritability, and psychosis, while CNS stimulation, depression, and incoherent speech may result from salicylate toxicity.

TREATMENT

At the present time, there is no definitive treatment for SLE. Mild cases may respond to simple supportive treatment, while the fulminant variety may remain unresponsive to the most vigorous treatment methods. To date, steroids remain the most efficacious treatment for SLE. Although not curative, they have proven highly effective for controlling the acute inflammatory manifestations of the disease.

Opinions regarding when to use steroids and at what dosages vary markedly. Richardson[11] states that the optimal smallest dosage is the amount capable of achieving control. Other authors[10,13-15] recommend doses ranging from 7 to 40 mg/day of prednisone for the management of the early phases of the disease. Once control is achieved, the dose may be tapered *very* gradually to a low maintenance dose of 20 mg/day or less.[10] In some cases, alternate-day therapy has been sufficient to maintain remission. The uninterrupted administration of steroids during the early years following diagnosis seems particularly useful, as discontinuation during this time may produce more exacerbations of the illness. Doses of prednisone in excess of 80 mg/day may be required to control the nephritis, hemolytic anemia, or central nervous system manifestations of the disease. It should be remembered that steroid psychoses are most likely to appear when doses exceed 40 mg/day of prednisone or the equivalent. Most steroid-induced psychoses develop within 5 days of exceeding this dose or adjusting the dose upward.[34]

Antimalarial agents are effective for the control of the cutaneous manifestations of the disease. It has been suggested that antimalarials may also be useful for controlling the disease when steroids are being tapered or discontinued, or when the dosage of steroids must be reduced.[5] Some authors believe, however, that the risk of producing a toxic retinopathy limits the use of antimalarials.[11,13,14]

Salicylates are useful for the management of musculoskeletal discomfort. In some SLE cases, adequate control has been achieved by the use of aspirin alone (40 to 80 grains q.d.).

Because of increased drug sensitivity, careful consideration must be given

before prescribing any medication to SLE patients. Patients must also be cautioned regarding the use of over-the-counter preparations.

Patients should be instructed to avoid direct sunlight except for brief periods (no more than a few minutes) and to use sun screens if exposure cannot be avoided. Adequate rest is also an important factor in avoiding exacerbations of the disease. Therefore, it is recommended that patients get a minimum of 8 to 10 hours sleep per night and not pursue any activity to the point of fatigue.

TREATMENT OF PSYCHIATRIC MANIFESTATIONS

Thioridazine is considered the drug of choice for treatment of the SLE psychosis because it is unlikely to lower the seizure threshold appreciably or to produce extrapyramidal symptoms or chemical hepatitis, and has a mild sedative and antidepressant effect. For these patients, behavior is best controlled with initial doses of 200–400 mg/day, which may be titrated upward as needed to a maximum dose of 800 mg/day.

Phenothiazines are also recommended for control of the symptoms of steroid psychosis. However, these patients respond well to lower doses than those needed to control the lupus psychosis. In a study conducted by the authors,[34] excellent response was obtained with an average daily dose of about 200 mg of chlorpromazine or thioridazine. We prefer thioridazine for the reasons stated above.

In those patients who develop an organic mental syndrome, symptoms are usually well controlled on lower doses of phenothiazine, i.e., 50–200 mg/day. High-dose phenothiazines in these patients may result in further deterioration.

When evaluating the drug regimens of these patients, it is worthwhile to remember that most phenothiazines, with the probable exception of thioridazine, tend to lower the seizure threshold. Since seizures are one of the most frequent CNS signs of lupus, the use of thioridazine may provide neurologic as well as psychiatric benefit. It should be noted, however, that both thioridazine and antidepressants, when used in conjunction with corticosteroids, will raise intraocular pressure. Tricyclic antidepressants may produce an acute worsening of a steroid-induced psychosis and should be avoided during the acute treatment of these conditions, regardless of whether depressive symptoms exist.[36]

EMOTIONAL SEQUELAE

In previous sections, we have discussed the psychiatric symptoms produced by specific organ involvement. Other types of psychiatric problems also occur. In a previous report,[33] we discussed several insights into lupus patients'

emotional state offered by patients themselves and by their families. Many of these patients reported acute exacerbations of their illness following emotional stress. Worry, followed by sleeplessness and increased fatigue, were viewed by a majority of patients as stressors associated with relapse. Sleeplessness and increased fatigue seemed to be specific prodromal symptoms which preceded the development of such psychiatric symptoms as anxiety, depression, or increased emotional lability.

Overall, those patients who maintained membership in specific support groups fared better than those who did not. We were particularly struck by the subjective differences in feeling state between these two groups. Patients with major support systems also appeared better adjusted. This observation may indicate that social activity, which reduces isolation and stress while serving to enhance self-concept, has a salutary effect on the course or impact of symptoms produced by the illness, or it may simply reflect the fact that sicker patients are less able to function socially.

Profound lethargy was reported by half of these patients as their most striking symptom. The lethargy was of such magnitude that they were unable to perform simple chores and was associated with episodes of spontaneous tearfulness and intermittent feelings of depression. Emotional lability was experienced by over 80% of the patients; many reported that they were "losing their minds" or becoming "erratic and irrational" because they were unable to control their sudden mood swings.

Many patients also commented on the emotional pain associated with misdiagnosis, which resulted in increased feelings of worthlessness. Following the diagnosis of lupus, patients reported fears of death, incapacitation, financial ruin, inability to care for their families, inability to maintain a job, body disfigurement, chronic invalidism, insanity, and an extreme sense of frustration related to looking well while feeling so weak that they were unable to function.

Those patients interviewed who had sought psychiatric treatment following the diagnosis of lupus found such involvement beneficial. Psychotherapy provided emotional support during periods of stress, consensual validation for decision-making, and an opportunity to ventilate fears and frustrations. In selected cases, couples therapy was reported as useful. Most patients reported that it was reassuring to know that they were not "going crazy" and that their psychiatrist would be available to manage any severe emotional or psychotic symptoms with medications or hospitalization if necessary. Both patients and family members felt more secure knowing psychiatric support was available should problems arise.

CONCLUSIONS

SLE is associated with many psychiatric problems. Some stem from physiologic changes in various organ systems, while others develop as side effects of treatment. The disease disrupts the person—his or her present and future,

family, mind, and body. The effects of stress precipitate relapse and interfere with coping abilities. Perhaps the most damaging effect of the disease is its ability to temporarily render the patient emotionally nonfunctional or psychotic, making it difficult for the patient or the family to plan ahead.

Psychiatric management is directed at "supporting the patient" during the course of the illness by providing information, consistent follow-up, medical direction, and effective psychopharmacologic management.

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