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Lupus Erythematosus

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I. LUPUS ERYTHEMATOSUS

A REVIEW OF SEVENTY-FIVE CASES

The earliest descriptions of lupus erythematosus were made by the eminent dermatologists, Biett, Cazenove and Kaposi. The skin lesions were first described in 1828, by Theodore Biett, under the name "erytheme centrifuge".[1] The condition had certainly been recognized prior to this time and had been known under a variety of names. In 1851, Biett's pupil, Cazenove[2] observed destructive forms of this disorder. He felt that the condition was related to lupus vulgaris so he applied the term "lupus erythematodes" to the malady. This was soon found to be more acceptable than previous terms.

One of the most important events in the evolution of the concept of this disorder occurred in 1872, when Kaposi[3] called attention to systemic disturbances in the disease. He observed patients who became acutely and fatally ill. These patients presented a high fever associated with skin lesions resembling erysipelas. He called the disease "erysipelas perstans faciei". These findings were soon confirmed by other observers. Jadassohn[4] was aware of the joint, mucous membrane, glandular, and renal involvement. Osler's[5] report on the visceral manifestations of the erythema group of skin diseases initiated the interest of general medicine in the problem. In 1924, Libman and Sacks[6] described an atypical form of verrucous endocarditis which was often associated with cutaneous manifestations; this was later recognized to be part of the lupus erythematosus picture. Important contributions to a better understanding of the disorder have been made by Klemperer and his group. They felt that lupus erythematosus might be classified with other diseases in which the collagen of the connective tissues was the seat of activity[7].

The discovery of the lupus erythematosus cell by Hargraves and associates[8] and the use of corticotropin and cortisone have given us new diagnostic and therapeutic weapons for our struggle against this disease.

Numerous attempts have been made to classify the types of this disorder[9,10,11,24]. Classification bases upon the morphology of the cutaneous lesions has been inadequate due to the variability of the cutaneous picture and because the lesions may be entirely lacking. Etiologic classification is not possible as long as the etiologic agent or agents are unknown. O'Leary[9] suggested classification into: (1) the chronic discoid type of the face and head, (2) the generalized discoid or chronic disseminated type with skin lesions beyond the face and head, (3) the subacute disseminated type, and (4) the more severe acute disseminated type. The first two types are usually considered to be primarily cutaneous conditions, while systemic manifestations became increasingly more important in the latter two types. Considerable confusion arose with the use of the term "disseminated"; to some it meant extension of the skin lesions, to others it connoted internal involvement. It is felt by some[12] that the term "disseminated" should be replaced by the term "systemic".

This division into systemic and apparently non-systemic types would appear to imply that the condition showing chronic discoid lesions is without systemic involvement. Indeed, there are those who feel that the chronic discoid and the systemic forms are two distinct clinical entities[13]. The recent trend has been to regard these conditions as being related[11]. This has been borne out by clinical and experimental observations. Huff and co-workers[14] were able to demonstrate similar blood flow changes in chronic discoid and systemic forms of the disease.

Another classification has been proposed by Michelson[10]. It includes the types already described with two additions: (1) the acute, localized, edematous type, and (2) the protracted systemic type. The lesions of the acute,

localized, edematous type are circumscribed discs which may disappear completely, or they may become transformed into the discoid form. The patient with the protracted systemic type presents a prolonged prodromal period with weight loss, low grade fever and other indefinite symptoms of undermined health. A positive L. E. cell test is the only definite means of diagnosing this type of lupus erythematosus. However, the same clinical findings may exist in a patient who has a negative L. E. cell test; the diagnosis is then a presumptive one.

Whatever classification is employed, it must be an arbitrary one based for the most part on the clinical course of the disease.

Various sized plaques characterize the chronic discoid form, these begin as papules accompanied by varying amounts of edema and erythema. The lesions become dry and scaly; this scaling extends into follicular and sweat duct openings. The lesions progress by peripheral extension with the central portion showing atrophic scarring. These changes are usually accompanied to some degree by telangiectasis and pigmentation. The usual sites involved are the face, ears, scalp and lips. The chronic generalized lesions are morphologically identical with those of the discoid type. The affected areas extend beyond those of the chronic discoid form. The sides and "V" of the neck, the chest, the arms and the hands, are frequently involved. Systemic manifestations are usually mild in both chronic forms.

These sites of involvement are areas which are usually exposed to the sun and to trauma. With the exception of the scalp, the covered areas are infrequently involved. Since the earliest pathologic changes occur in the cutaneous vessels, this predilection for certain areas might be due to some vascular peculiarity. Lewis[15] felt that the vessels in these areas were "atonic" because they did not respond in the usual manner to vasoconstrictor substances.

The clinical picture of the acute type of disease is variable. Most of the patients are women of childbearing age. The patient presents an initial ill-defined prodromal period with complaints of malaise, fatigue, weight loss, arthralgia and a low grade fever. Various types of skin lesions may appear early or late in the disease, their appearance usually indicates an increased severity. The widespread pathologic involvement of the connective tissue in various body organs and systems results in polymorphic symptoms and signs. The low grade fever may become septic in type. Arthralgia involving the small joints may be the prominent complaint; this often leads to an incorrect diagnosis of rheumatic fever or rheumatoid arthritis. Serosal involvement results in pleuritic, pericardial and peritoneal irritation. Involvement of the gastrointestinal, respiratory, cardiovascular, renal and central nervous systems will present symptoms and signs referable to the involved areas. Peritoneal and gastrointestinal involvement may simulate an acute surgical condition. Enlargement of the liver and spleen are common. Lymphadenopathy may be a prominent feature. Conjunctivitis and retinal exudates are found in some patients.

Laboratory examinations usually reveal renal disturbances with albumin, red cells, white cells and casts in the urine. The blood picture may show evidence of suppression of hematopoetic function with anemia, leucopenia and thrombocytopenia. The erythrocyte sedimentation rate is elevated and the albumin-globulin ratio may be reversed.

It is usually possible to demonstrate L. E. cells in the venous blood or bone marrow in the systemic cases, but not in the chronic or mild subacute types.

The patients with the subacute form of the disease occupy an intermediate position between the two extremes already described. The cutaneous lesions are usually symmetrically distributed on the upper portions of the body. They tend to be edematous and erythematous, with little scarring when healed. Sys-

temic involvement is less severe than that in the acute form, but more extensive than in the chronic type.

There have been other series of lupus erythematosus cases reported in the literature[11,16,17,18,19,20,21,22] Most of these have concerned themselves with the disseminated systemic type, only a few have included the internal findings in chronic cases.

Our present study included all types of lupus erythematosus occurring in patients who were admitted to the University of Minnesota Hospitals during the ten year period, 1944 through 1953. The classification used in compiling our data was the same as that reported on the patients' hospital record. Wherever a classification had not been made, the case was classified according to O'Leary's descriptions. Uncertain and suspected cases were not included. In instances where similar findings had been recorded previously, similar criteria were employed by us.

There were 75 patients with lupus erythematosus admitted during the 10 years. Twenty patients had the chronic type, 16 the chronic discoid and four the chronic disseminated. There were 35 with the subacute disseminated type and 20 with acute disseminated form admitted during the period. A total of 124 separate admissions were made to various hospital services. The 94 admissions to the dermatology service comprised 7.2% of this service's total admissions for the 10 years. There was a notable increase in admissions since 1949. This increase might be accounted for by any one or all of the following: (1) growing medical interest in the disorder, (2) improved diagnostic methods, (3) increased incidence, and (4) prolonged survival of the more severe cases as a result of improved therapy. Dubois[21] reported the recent incidence at the Los Angeles County General Hospital to be one half that of rheumatic fever.

Seventy of our patients were American-born, with nationality backgrounds similar to those of the general hospital population. Two of the patients were American Indians (a male had chronic discoid lesions and a girl expired from the systemic form of the disease). Five patients were foreigh-born, two in Sweden, one in Germany, one in Austria and one in the Philippines. There were no Negroes in our series. Their occupations were varied; most of the female patients were housewives. Our group included the following hospital personnel: a nurse, two hospital maids, a bacteriologist, a laboratory technician and a nurses' aide. The last three had fatal outcomes. Lupus erythematosus has been reported as the second most common cause of death in 750 nurses who had been followed for 15 years[23].

The sex incidence in our study is comparable to that in other series. The chronic group consisted of nine males and 11 females whose average age at onset was 37 years, with age extremes of 4 and 69 years. There were 30 females and 5 males in the subacute group; the average age at onset was 33 years, with age extremes of 6 and 74 years. The acute group was composed of 16 females and four males whose average age at onset was 30 years, the age extremes were 12 and 63 years. Twenty patients with subacute and nine with acute forms had the onset of their complaints during the second and third decades of life.

The transition of the chronic discoid form into the more severe systemic form has been reported[17,25] as having occurred in approximately 25% of subacute and 20% acute cases, but in our series this transition was less frequent. In five patients (14%) with the subacute form and one patient (5%) with the acute form, the disease began as the chronic discoid form.

The symptoms and signs presented in our series are summarized in tables I and II. The clinical picture was polymorphic in most instances. There did not appear to be any specific clinical finding or combination of clinical findings which might be essential for a diagnosis of lupus erythematosus. It was necessary to appraise carefully the

TABLE I

FREQUENCY OF SYMPTOMS

75 CASES OF LUPUS ERYTHEMATOSUS

Symptoms	Chronic 20 Cases		Subacute 35 Cases		Acute 20 cases	
	No.	%	No.	%	No.	%
Malaise	6	30	31	89	20	100
Arthralgia	5	25	20	57	17	85
Gastrointestinal	1	5	17	49	13	65
Weight loss	4	20	20	57	12	60
Light sensitivity	8	40	17	49	6	30
Ocular disturbances	0	0	6	17	6	30
Central Nervous System	5	25	16	46	5	25
Respiratory	0	0	13	37	5	25
Skin Lesion Pruritus	5	25	17	49	4	20
Cardiovascular	0	0	10	29	4	20
Menstrual disorders	0	0	5	14	4	20

TABLE II

FREQUENCY OF SIGNS

75 CASES OF LUPUS ERYTHEMATOSUS

Signs	Chronic 20 Cases		Subacute 35 Cases		Acute 20 Cases	
	No.	%	No.	%	No.	%
Fever, Septic	0	0	18	54	20	100
Fever, low grade	5	25	12	34	0	0
Skin lesions	20	100	31	89	18	90
Lymphadenopathy	1	5	16	46	18	90
Tachycardia	0	0	10	29	13	65
Mouth lesions	0	0	6	17	12	60
Hair loss	2	10	9	27	11	55
Mental Changes	1	5	12	34	7	35
Systolic Murmur	0	0	10	29	7	35
Hepatomegaly	1	5	7	20	6	30
Nasal Lesions	0	0	2	6	5	25
Raynaud's Phenomenon	1	5	5	14	4	20
Joint Swelling	0	0	9	27	3	15
Fundoscopic Abnormalities	0	0	2	6	3	15
Pleural effusion	0	0	1	3	3	15
Splenomegaly	1	5	3	9	2	10

patient's whole clinical and laboratory picture before making this diagnosis.

Cutaneous lesions were present on hospital admission in all the patients with the chronic form and in 89% of those with the subacute form. Fifteen of those with the acute disease presented skin lesions on admission, and three others developed lesions while in hospital. Only two patients with the acute form failed to show any skin lesions. Cutaneous lesions preceded systemic symptoms and signs in 57% of the subacute and 45% of the acute group. Localized hair loss at the sites of scalp involvement was noted in two patients with the chronic variety. A diffuse thinning and loss of hair was noted on all the body hairy areas in nine patients with the subacute and in 11 with the acute form. Skin lesions were localized to the "butterfly" area of the face in only two with acute and three with subacute forms. The skin eruptions were most extensive in those with the more severe disease.

The patients in the chronic group were not lacking in constitutional symptoms; however these were not as frequently found as in the subacute and acute forms of the disease. Malaise, weight loss and arthralgia assumed greater significance as the severity of the condition increased. Joint symptoms were more frequent than grossly detectable joint alterations. Gastrointestinal symptoms in order of decreasing frequency were: anorexia, nausea, vomiting, diarrhea and constipation.

Ocular manifestations were most common in the acute types, less common in the subacute and absent in the chronic types. Five of the patients with the acute type presented edema and crusting of the eyelids. The eyelid lesions were usually associated with cutaneous lesions elsewhere[26]. Retinal exudates were found in two patients with subacute and three with acute forms. It is felt by some[27] that this "toxic retinitis" cannot be regarded as pathognomonic of lupus erythematosus. Blurred vision, photophobia and diplopia occurred with equal frequency in the acute and subacute groups in this series.

Scattered, shifting neurological findings have been demonstrated throughout the course of the disease[28]. Peripheral neuritis is not an uncommon finding[29]. Epilepsy, preceding or associated with the disorder, was felt by Russell and co-workers[30] to be one of the most frequent central nervous system manifestations. Our patients with chronic lupus manifested headache most commonly. Two of them complained of numbness and tingling of the hands and feet. One was admitted to the Psychiatric service with a "depressive reaction". Another experienced grand and petit mal seizures seven months prior to the appearance of his skin lesions. His electroencephalographic pattern indicated "diffuse cortical damage".

Nine of those with the subacute form complained of headache of no constant pattern, four complained of numbness and tingling in the limbs and three complained of occasional dizziness. One of them had a history of grand and petit mal seizures for 14 years prior to the onset of her symptoms. Her EEG tracing was of the petit mal type. Six others had normal EEG patterns. Two of them with fatal outcomes and one patient with the acute form who underwent a prolonged remission, had grand mal convulsions during the course of their disease.

Three patients with the acute form complained of headaches and one presented a diffuse polyneuritis. Electroencephalographic findings were normal in three patients in this group. One of these was a female patient who showed normal EEG tracings before and during a severe psychotic episode which followed cortisone therapy. Various mental and personality changes, consisting of nervousness, depression and irritability were found in a total of 19 of the patients with subacute and acute forms.

Respiratory system involvement may be: (1) primary, due to the disease process,

or (2) secondary, due to an infective pneumonitis. Israel[33] felt that lupus erythematosus should be considered in all instances of pulmonary or pleural diseases of obscure etiology. Thorell[31] felt that the chest X-ray findings of pleural effusion, irregular plueral thickening and mottled or streaky parenchymal infiltrations were suggestive of lupus erythematosus. Rapaport and associates[32] reviewed the literature concerning pulmonary manifestations. They considered that the commonest findings consisted of patchy, shifting areas of pneumonitis usually found in the lung bases. This was often associated with a pleural effusion.

Our series of patients presented symptoms of pleuritic chest pains and cough more frequently than positive physical findings. Patients with chronic form did not exhibit any respiratory symptoms or chest X-ray findings which might be associated with the disorder. Radiological findings in the subacute group included pleural effusion in seven, increased bronchovascular markings in four, pleural adhesions in three, basal areas of pneumonitis in two, and bilateral mottled densities in one. Seventeen patients of the acute type exhibited radiological abnormalities; nine presented a pneumonitis, five with basal involvement and four with scattered mottled involvement throughout both lung fields. Increased bronchovascular markings were present in seven, pleural effusion was found in four. One patient who died of the disease had atalectesis of a lung lobe.

Dyspnea was the most prominent cardiovascular complaint in a total of 14 patients in the subacute and acute groups. Systolic murmurs were present in 10 patients with the subacute and seven with the acute form. Two patients with fatal acute lupus developed systolic murmurs while under observation in hospital. Tachycardia was a prominent feature in the more severe instances.

The prodromal low grade fever became septic in type in all those with acute and in over half of those with subacute forms.

Menstrual irregularities were recorded in the subacute and acute forms of the disease. These irregularities did not display any uniform pattern; they included dysmenorrhea, amenorrhea and menorrhagia. One patient with subacute and two with acute lupus had exacerbations during their menstrual periods. Abortions and miscarriages were recorded in two patients in each of the subacute and acute groups. Two others with the subacute form noted marked improvement while pregnant; they did not experience any difficulties at the time of delivery.

The marked sex incidence and involvement during the active sexual part of life have led some authors to suggest that therapeutic benefit might be achieved by oophorectomy[34]. This procedure has resulted in only questionable success. One of our patients with the subacute form had oophorectomy performed three years prior to the onset of her lupus erythematosus symptoms. Another received irradiation to the ovaries but without any sustained improvement in her general condition.

Lymphadenopathy was more prominent in the acute group; half of the acute patients presenting enlarged glands had simultaneous involvement of the cervical, inguinal and axillary nodes. One presented cervical adenopathy as the first sign of her lupus erythematosus.

Hepatomegaly was a more frequent finding than splenomegaly.

Ulcerating mouth lesions were found in six patients in the subacute and 12 in the acute groups. The nasal mucosa was involved to a lesser extent. Two patients who later died of the disease presented vaginal involvement which was grossly similar to their cutaneous lesions.

The association of lupus erythematosus with Raynaud's phenomenon had been noted as early as 1908.[35] We encountered this phenomenon in one chronic, fine subacute

TABLE III

LABORATORY FINDINGS IN 75 CASES OF LUPUS ERYTHEMATOSUS

	CHRONIC		SUBACUTE		ACUTE	
	No. Cases Examined	% with Abnormality	No. Cases Examined	% with Abnormality	No. Cases Examined	% with Abnormality
Urine						
White Blood Cells	20	40%	35	62%	20	90%
Albumin	20	10%	35	46%	20	90%
Red Blood Cells	20	10%	35	31%	20	85%
Casts	20	5%	35	8%	20	80%
Blood						
Leucopenia (less than 5,000 WBC/Cu.mm.)	20	45%	35	77%	20	75%
Anemia (less than 4 million RBC, less than 12 Gm. Hem.)	20	20%	35	43%	20	70%
Sedimentation Rate	18		35		20	
0-2- mm. per hour		22%		8%		0
21-40		22%		23%		5%
41-60		34%		23%		0
over 61		22%		46%		95%
Serum Protein	16		33		18	
6-8 Gm.%		87%		78%		83%
less than 6 Gm. %		0%		6%		17%
more than 8 Gm. %		13%		16%		0
Reversed A. G. Ratio	16	62%	33	64%	15	100%
Increased Gamma Globulin	0		16	56%	8	100%
L.E. Cell	7	0	29	27%	14	78%

and four acute cases.

The laboratory findings of the patients in our series are tabulated in Table III. These findings are similar to those reported elsewhere. Some of the examinations in our series were performed in such small numbers as to detract from their value. It is of interest to note the frequency of abnormal laboratory findings in the chronic group which has usually been considered to be free of any systemic manifestations.

As the systemic involvement became more extensive, the laboratory tests revealed evidence of increased renal irritation, increased erythrocyte sedimentation rate and a more uniform reversal of the albumin globulin ratio.

Various abnormal urinary findings were found in 80 to 90% of our patients with the acute type. Seventy-five per cent of this group also exhibited leucopenia on admission. The lowest recorded white cell count was 800 per cubic millimeter, the usual range was from 2500 to 4000 leucocytes per cubic millimeter. The neutrophilic leucopenia was accompanied by a relative lymphocytosis in 55% of those with acute and 61% of those with subacute lupus. Sixty-five per cent of those in the acute and 40% of those in the subacute group had been subject to a variety of intercurrent infections; these patients were able to respond with a relative or absolute leucocytosis, the white cells showing a marked shift to the left. Lupus erythematosus patients admitted with a superimposed infection present a leucocytosis rather than a leucopenia; the leucopenia becomes apparent after the infection has subsided.

A moderate degree of thrombocytopenia is not an uncommon finding in lupus erythematosus. Platelet counts of less than 150,000 per cubic millimeter were recorded in 41% of 17 patients with subacute, in 50% of 12 with acute, and two of five with chronic forms.

Anemia was found in all forms of the disorder, most commonly in the acute. It was normocytic and normochromic in most instances. The anemia decreased slowly as the patients' general condition improved.

The sedimentation rate paralleled the clinical course of the disease. Sedimentation rates greater than 61 mm. per hour were recorded in 95% of patients with acute, 46% of those with subacute and 22% of those with chronic forms. The sedimentation rate decreases only slowly as the condition improves. It tends to remain somewhat elevated, even in patients who have shown a favorable response to steroid therapy.

Plasma protein changes have been accepted as a common finding in lupus erythematosus. During the course of the disease reversed albumin globulin ratios were found in 62% of 16 patients with the chronic type, 64% of 33 with the subacute, and in 100% of 15 with the acute. The gamma globulin fraction was measured in eight patients with acute and in 16 with subacute lupus. It was elevated in all of the former and in 56% of the latter. This increase in gamma globulin has also been reported in the chronic discoid form[36] Gamma globulin levels have not been studied in any of our patients with the chronic type. The factor responsible for the L. E. cell phenomenon has been shown to be in the gamma globulin fraction[37]. Three of the patients in the subacute group and two in the acute who exhibited increased gamma globulin fractions failed to exhibit the L. E. cell phenomenon. The level of gamma globulin in these patients was as high as in the other patients who had positive L. E. cell tests.

Seven patients with chronic discoid lupus were examined for L. E. cells; all had negative tests. The L. E. cell phenomenon was found in 27% of 29 patients with subacute and in 78% of 14 with acute types. The three patients with the acute form who had negative L. E. cell tests presented typical clinical pictures of lupus erythematosus; one of these patients expired with the

disease. It is evident that the L. E. cell test is a useful diagnostic procedure but it is not essential that it be positive before a diagnosis of acute lupus erythematosus is made.

Liver function studies were carried out in 10 patients with the chronic form, 20 with the subacute and 13 with the acute. Those in the chronic group failed to reveal any abnormalities; three in the subacute and six in the acute presented positive thymol turbidity and cephalin cholesterol flocculation tests. These findings may have been due to plasma protein alterations rather than to hepatic damage. Bromosulphalein retention was found in one patient in the subacute and two in the acute groups.

Biologic false positive serologic tests for syphilis have been reported in all types of lupus erythematosus[17,38,39]. There have been reports[40] of false positive tests preceding the clinical symptoms by as long as seven years. Moore and Mohr[41] felt that chronic biologic false positive tests were often related to some serious underlying condition, often to a proven or suspected case of lupus erythematosus.

Serologic tests for syphilis were done in all of our patients with the acute form; two of these (10%) had false positive findings. Twenty-nine in the subacute group were studied in a similar manner; three (10%) had false positive serologic tests. One of these three patients had received luetic treatment before a diagnosis of lupus erythematosus had been made. There were no false positive tests in the 14 patients with chronic lupus examined. Two of these had residual positive serologies from past syphilitic infections.

There have been reports of increased porphyrins in the urine of patients with all types of lupus erythematosus[42,43,44]. Some of the observers[42] felt that the increased "hematoporphyrin" acted as a photodynamic sensitizer of the skin. Ten of our patients in the chronic group underwent urinary porphyrin studies. One of these had increased urinary coproporphyrin. Fifteen in the subacute group were also studied, and three of these patients had increased urinary coproporphyrins. Of five in the acute group similarly studied, two who had normal urinary porphyrins on admission excreted increased amounts shortly before death, a third had normal levels on admission but exhibited an increase in the urinary coproporhyrin during a relapse in his condition. The levels in the other two were within the normal range.

There is a divergence of opinion concerning the effect of surgical procedures on lupus erythematosus patients. This problem has been reviewed by Greenhouse[45], who also reported 24 patients who had undergone various minor and major surgical procedures without any ill effects.

In our series, five patients in the chronic group underwent a thyroidectomy, a cystocele repair and other minor procedures without any difficulty. Various minor procedures were carried out on 15 patients in the subacute group without any deleterious effect upon their general condition. One patient felt improved after extraction of some bad teeth. Ten patients with the acute type underwent minor procedures without mishap. The condition of one acute patient deteriorated after multiple skin biopsies and another patient flared after having some teeth extracted. Two others showed improvement after incision and drainage of multiple abscesses.

There have been numerous methods of treatment advocated. Recent reports indicate that the drugs of choice in the treatment of the chronic discoid form are quinacrine[46,47,48,49], and chloroquine[50]. Quinacrine has also been used in certain patients with the subacute form[51]. The treatment of the latter requires individual appraisal. Steroid therapy may be required in the more severe case. The severe acute form usually requires corticotropin or cortisone therapy. These hormones suppress the fever, arthritis and sero-

sal symptoms, but they have less effect upon the associated laboratory findings. They do not cure the disease. They are palliative and prolong life in these patients.

The course of any form of lupus erythematosus is unpredictable. It is therefore very difficult to evaluate any form of therapy. Remissions and relapses may occur frequently. Ben-Asher[52] was able to observe a patient through several remissions and relapses during a 23 year period.

There have been 11 deaths in our group of 20 patients with the acute form. Four who died had received steroid therapy. The average duration of the disease until death was 20 months, with extremes of one month and four years. All three children in the acute group died. Four patients in the acute group are under observation, four others have not been followed since their hospital release. One has now had a three year remission. She did not receive any steroid therapy during the course of her disease. Her general condition is quite satisfactory, her former positive L. E. cell test is now negative, and there has been an improvement in her anemia. She still presents abnormal urinary findings, an elevated sedimentation rate and a slight leucopenia.

The recent years have witnessed an increased interest in the problem of lupus erythematosus. This disease has become the common meeting place of the various branches of medicine. Many important advances have been made during the past ten years; however, there are many problems that have yet to be solved.

SUMMARY

The clinical and laboratory features of seventy-five patients with lupus erythematosus have been reviewed. This study included all types of lupus erythematosus occurring in patients admitted to the University of Minnesota Hospitals during the ten year period 1944 through 1953.

Clinical, laboratory and experimental observations indicate that the chronic discoid form of lupus erythematosus frequently presents systemic manifestations similar to those of the other types. It will be necessary to evaluate further this relationship.

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II. MEDICAL SCHOOL NEWS

Coming Events

April 1	Clarence M. Jackson Lecture; "Management of Massive Upper Gastro-Intestinal Bleeding;" Dr. J. Garrott Allen, Professor of Surgery, University of Chicago Medical School; Owre Amphitheater; 8:00 p.m.
April 1 - 3	Continuation Course in Emergency Surgery for General Physicians
April 5	Seminar on History of Medicine; "One Thousand Years of Medicine and Surgery 600-1600 A.D.;" Dr. August C. Krey, Professor of History, University of Minnesota; Todd Amphitheater, University Hospitals; 7:30 p.m.
April 5 - 7	Continuation Course in Eye, Ear, Nose, and Throat for General Physicians
April 8	Duluth Clinic Lecture; "The Role of the Ionic Environment in Carbohydrate Metabolism;" Dr. A. Baird Hastings; Owre Amphitheater; 8:00 p.m.
April 8 - 10	Continuation Course in Urology for General Physicians

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Continuation Course

The University of Minnesota will present a continuation course in Urology for General Physicians next April 8 to 10, 1954, at the Center for Continuation Study. Common urological problems encountered by the physician in general practice will be discussed in detail. Guest speaker will be Dr. Wyland F. Leadbetter, Professor of Urology, Tufts College Medical School, and Chief, Department of Urology, New England Center Hospital, Boston. The course will be presented under the direction of Dr. C. D. Creevy, Professor and Director, Division of Urology, who will be joined by members of the faculty of the University of Minnesota Medical School and the Mayo Foundation.

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Faculty News

Dr. William T. Peyton, Professor and Director, Division of Neurosurgery, presented the lecture entitled "Some Observations on the Blood Brain Barrier and the Use of Radioactive Isotopes in Neurosurgery" to the medical faculty at the University of Toronto and to the Concinnati Society of Neurology and Psychiatry last month.

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Publications of the Medical School Faculty

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III. UNIVERSITY OF MINNESOTA MEDICAL SCHOOL

WEEKLY CALENDAR OF EVENTS

Physicians Welcome

March 29 - April 3, 1954

Monday, March 29

Medical School and University Hospitals

9:00 - 9:50 Roentgenology-Medicine Conference, L. G. Rigler, C. J. Watson and Staff; Todd Amphitheater, U. H.

9:00 - 10:50 Obstetrics and Gynecology Conference; J. L. McKelvey and Staff; W-612, U. H.

10:00 - 12:00 Neurology Rounds; A. B. Baker and Staff; Station 50, U. H.

11:30 - Tumor Conference; Doctors Hitchcock, Moore, and Stenstrom; Todd Amphitheater, U. H.

12:15 - Obstetrics and Gynecology Journal Club; Staff Dining Room, U. H.

1:30 - 2:30 Pediatric-Neurological Rounds; R. Jensen, A. B. Baker and Staff; U. H.

1:30 - 3:30 Dermatology Hospital Rounds; H. E. Michelson and Staff; Dermatology Histopathology Room, M-434, U. H.

4:30 - Public Health Seminar; Recent Trends in Group Practice; Dr. Goldman; 15 Owre Hall.

4:30 - Infectious Disease Rounds; Station 43, U. H.

5:00 - 6:00 Physiology-Surgery Conference; Todd Amphitheater, U. H.

5:00 - 6:00 Urology-Roentgenology Conference; C. D. Creevy, O. J. Baggenstoss, and Staff; Eustis Amphitheater.

Ancker Hospital

8:30 - 10:00 Tuberculosis and Chest Conference; Auditorium.

2:00 - 3:00 Surgery Journal Club; Classroom.

Minneapolis General Hospital

8:30 - Pediatric Rounds; L. Arey; Stations I and J.

10:30 - 12:00 Medicine Rounds; Thomas Lowry; Station F.

11:00 - Orthopedic and Fracture Rounds; Drs. John Moe and Arthur Zierold; Station A.

11:00 - Pediatric Rounds; Erling Platou; Station K.

12:30 - Surgery Grand Rounds; Dr. Zierold; Station E.

1:30 - 2:30 Tuberculosis Conference; J. A. Myers; Station M.

2:00 - Pediatric Rounds; Stations I and J.

Monday, March 29, (Cont.)

Veterans Administration Hospital

9:30 - Infectious Disease Rounds; Drs. Hall, Zinneman, Lubin and Sherman.

1:30 - Cardiac Conference; Drs. Berman, Smith, Hoseth, and Wexler; Conference Room, Bldg. I.; Rounds immediately following conference.

Tuesday, March 30

Medical School and University Hospitals

9:00 - 9:50 Roentgenology-Pediatric Conference; L. G. Rigler, I. McQuarrie and Staff; Eustis Amphitheater, U. H.

12:30 - 1:20 Pathology Conference; Autopsies; J. R. Dawson and Staff; 102 I. A.

3:30 - Pediatric Seminar; Infections of the Newborn; Florence Char; Sixth Floor, U. H.

3:30 - Biophysics-General Physiology Seminar; Protein Structure and Information Content; C. P. Barnum; 323 Zoology Building.

4:00 - 5:00 Pediatric Rounds on Wards; I. McQuarrie and Staff; U. H.

4:30 - 5:30 Clinical-Medical-Pathological Conference; Todd Amphitheater, U. H.

Ancker Hospital

8:00 - 9:00 Fracture Conference; Auditorium.

9:00 - 10:00 Medical X-ray Conference; Auditorium.

Minneapolis General Hospital

9:30 - Pediatric Contagion Rounds; Elizabeth Lowry; Station K.

10:00 - Psychiatry Grand Rounds; R. W. Anderson; Station H.

11:00 - 12:00 Medicine-Surgery Conference; Classroom, Station M.

12:30 - 2:30 Dermatology Rounds on Clinic; Carl W. Laymon and Staff.

12:30 - ECG Conference; Boyd Thomes and Staff; 302 Harrington Hall.

1:00 - Tumor Clinic; Drs. Eder, Coe, and Lipschultz; Classroom.

3:00 - 5:00 Pediatric Psychiatry Conference; Jack Wallinga; Classroom, Station I.

Veterans Administration Hospital

7:30 - Anesthesiology Conference; Conference Room, Bldg. I.

8:30 - Surgery Staff Seminar; Conference Room, Bldg. I.

9:30 - Surgery-Pathology Conference; Conference Room, Bldg. I.

10:30 - Surgery-Tumor Conference; L. J. Ray, J. Jorgens and Donn Mosser; Conference Room, Bldg. I.

1:00 - Review of Pathology, Pulmonary Tuberculosis; Conference Room, Bldg. I.

Tuesday, March 30, (Cont.)

Veterans Administration Hospital (Cont.)

1:30 - Combined Medical-Surgical Chest Conference; Conference Room, Bldg. I.

2:00 - 2:50 Dermatology and Syphilology Conference; H. E. Michelson and Staff; Bldg. III.

4:00 - Thoracic Surgery Problems; Conference Room, Bldg. I.

Wednesday, March 31

Medical School and University Hospitals

8:00 - 9:00 Roetgenology-Surgical-Pathological Conference; Paul Lober and L. G. Rigler; Todd Amphitheater, U. H.

11:00 - 12:00 Pathology-Medicine-Surgery-Pediatrics Conference; Todd Amphitheater, U. H.

12:30 - 1:30 Physiology 114B -- Transport Seminar; Nathan Lifson and M. B. Visscher; 214 Millard Hall.

1:00 - 2:00 Dermatology Clinical Seminar; F. W. Lynch; 300 North Clinic.

1:30 - 3:00 Pediatric Allergy Clinic; Albert V. Stoesser and Lloyd Nelson; W-211, U. H.

3:30 - 4:30 Dermatology Pharmacology Seminar; J. D. Krafchuk; 3rd Floor Conference Room, Heart Hospital.

4:30 - 5:50 Dermatology Infectious Disease Seminar; J. D. Krafchuk; 3rd Floor Conference Room, Heart Hospital.

5:00 - 5:50 Urology-Pathological Conference; C. D. Creevy and Staff; Eustis Amphitheater, U. H.

5:30 - 7:30 Dermatology Journal Club and Discussion Group; Hospital Dining Room.

7:30 - 9:30 Dermatology Pathology Seminar; Review of Interesting Slides of the Week; Robert W. Goltz; Todd Amphitheater, U. H.

Ancker Hospital

8:30 - 9:30 Clinico-Pathological Conference; Auditorium.

12:30 - 1:30 Medical Journal Club; Library.

Minneapolis General Hospital

9:30 - Pediatric Rounds; Richard Raile; Station J.

10:30 - 12:00 Medicine Rounds; Thomas Lowry and Staff; Station D.

10:30 - Pediatric Seminar; Arnold Anderson; Classroom, Station I.

12:30 - Pediatric Staff Meeting; Classroom, Station I.

1:30 - Pediatric Rounds; Erling Platou; Classroom, Station I.

2:00 - 5:00 Infectious Disease Rounds and Conference; Wesley W. Spink; Sta. 100.

Wednesday, March 31,(Cont.)

Veterans Administration Hospital

8:30 - 10:00 Orthopedic X-ray Conference; E. T. Evans and Staff; Surgical Conference Room, Bldg. 43.

8:30 - 12:00 Neurology Rehabilitation and Case Conference; A. B. Baker.

9:00 - Gastro-Intestinal Rounds; Drs. Wilson, Zieve, Hay, Brakel, Nesbitt and O'Leary.

11:00 - Gastrenterology Conference; Conference Room, Bldg. I.

12:30 - Medical Journal Club; Doctors' Dining Room.

12:30 - X-ray Conference; J. Jorgens; Conference Room, Bldg. I.

1:30 - 3:00 Metabolic Disease Conference; Drs. Flink, Schultz and Brown.

7:00 - Lectures in Basic Science of Orthopedics, Conference Room, Bldg. I.

Thursday, April 1

Medical School and University Hospitals

9:00 - 11:50 Medicine Ward Rounds; C. J. Watson and Staff; E-221, U. H.

11:00 - 12:00 Cancer Clinic, K. Stenstrom, A. Kremen and F. Zimmermann; Todd Amphitheater, U. H.

12:00 - 1:00 Medical Journal Club; Intra-Abdominal Sensations; John Jenne; 116 Millard Hall.

12:30 - Physiological Chemistry Seminar; The Nature of the Effects of Ions on Hydrolytic Enzymes; F. Bollum; 214 Millard Hall.

12:30 - 1:30 Electrocardiography Conference; Ernst Simonson; Cardiac Clinic; Staff Room, Heart Hospital.

1:30 - 4:00 Cardiology X-ray Conference; Heart Hospital Theatre.

*8:00 - Clarence M. Jackson Lecture; "Management of Upper Gastro-Intestinal Bleeding;" Dr. J. Garrott Allen, Professor of Surgery, University of Chicago Medical School; Owre Amphitheater.

Ancker Hospital

8:00 - 10:00 Medical Grand Rounds; Auditorium.

Minneapolis General Hospital

9:30 - Neurology Rounds; Heinz Bruhl; Station I.

9:30 - Pediatric Contagion Rounds; Elizabeth Lowry; Station K.

10:00 - Psychiatry Grand Rounds; R. W. Anderson and Staff; Station H.

11:30 - 12:30 Clinical Pathological Conference; John I. Coe; Classroom.

* Indicates special meeting. All other meetings occur regularly each week at the same time on the same day. Meeting place may vary from week to week for some conferences.

Thursday, April 1, (Cont.)

Minneapolis General Hospital (Cont.)

12:30 - 2:30 Dermatology Rounds and Clinic; Carl W. Laymon and Staff.

1:00 - Fracture - X-ray Conference; Drs. Zierold and Moe; Classroom.

1:00 - House Staff Conference; Station I.

Veterans Administration Hospital

8:00 - Surgery Grand Rounds; Conference Room, Bldg. I.

8:00 - Surgery Ward Rounds; Lyle Hay and Staff; Ward 11.

8:30 - Hematology Rounds; Drs. Hagen and Fifer.

11:00 - Surgery-Roentgen Conference; J. Jorgens; Conference Room, Bldg. I.

1:00 - 3:00 Bacteriology Conference; Pneumococcus; Dr. Hall; Conference Room, Bldg. I.

4:00 - Medical-Surgical Conference; Conference Room, Bldg. I.

Friday, April 2

Medical School and University Hospitals

8:00 - 10:00 Neurology Grand Rounds; A. B. Baker and Staff; Station 50, U. H.

9:00 - 9:50 Medicine Grand Rounds; C. J. Watson and Staff; Todd Amphitheater, U.H.

10:30 - 11:50 Medicine Rounds; C. J. Watson and Staff; Todd Amphitheater, U. H.

10:30 - 1:50 Otolarynogology Case Studies; L. R. Boies and Staff; Out-Patient Department, U. H.

11:00 - 12:00 Vascular Rounds; Davitt Felder and Staff Members from the Departments of Medicine, Surgery, Physical Medicine, and Dermatology; Eustis Amphitheater, U. H.

11:45 - 12:50 University of Minnesota Hospitals Staff Meeting; Some Studies in Nursing; Rena E. Boyle and Ruth V. Johnston; Powell Hall Amphitheater.

1:00 - 2:50 Neurosurgery-Roentgenology Conference; W. T. Peyton, Harold O. Peterson and Staff; Todd Amphitheater, U. H.

1:30 - 2:30 Dermatology Grand Rounds; Presentation of Cases from Grouped Hospitals (University, Ancker, General and Veterans) and Private Offices; H. E. Michelson and Staff; Eustis Amphitheater, U. H.

2:30 - 4:00 Dermatology Hospital Rounds; H. E. Michelson and Staff; Begin at Dermatology Histopathology Room, M-434, U. H.

3:00 - 4:00 Neuropathological Conference; F. Tichy; Todd Amphitheater, U. H.

3:30 - 4:30 Dermatology-Physiology Seminar; J. D. Krafchuk; 3rd Floor Conference Room, Heart Hospital.

4:00 - 5:00 124 Advanced Neurophysiology Lecture; Werner Koella and Ernst Gellhorn; 111 Owre Hall.

Friday, April 2, (Cont.)

Medical School and University Hospitals (Cont.)

4:30 - 5:20 Ophthalmology Ward Rounds; Erling W. Hanson and Staff; E-534, U. H.

5:00 - Urology Seminar and X-ray Conference; Eustis Amphitheater, U. H.

Ancker Hospital

1:00 - 3:00 Pathology-Surgery Conference; Auditorium.

Minneapolis General Hospital

9:30 - Pediatric Rounds; Richard Raile; Station J.

10:30 - Pediatric Surgical Conference; Oswald Wyatt, Tague Chisholm, and B. Spencer; Classroom, Station I.

12:00 - Surgery-Pathology Conference; Dr. Zierold, Dr. Coe; Classroom.

1:00 - 3:00 Clinical-Medical Conference; Thomas Lowry; Classroom, Station M.

1:15 - Pediatric X-ray Conference; Oscar Lipschultz; Classroom, Main Bldg.

2:00 - Pediatric Rounds; Station I and J.

Veterans Administration Hospital

10:30 - 11:20 Medicine Grand Rounds; Conference Room, Bldg. I.

1:00 - Chest Pathology Follow-Up Conference; E. T. Bell; Conference Room, Bldg. I.

2:00 - Autopsy Conference; E. T. Bell; Conference Room, Bldg. I.

Saturday, April 3

Medical School and University Hospitals

7:45 - 8:50 Orthopedic X-ray Conference; W. H. Cole and Staff; M-109, U. H.

9:00 - 10:30 Pediatric Grand Rounds; Eustis Amphitheater, U. H.

9:00 - 11:50 Medicine Ward Rounds; C. J. Watson and Staff; Heart Hospital Amphitheater.

9:15 - 10:00 Surgery-Roentgenology Conference; L. G. Rigler, J. Friedman, Owen H. Wangensteen and Staff; Todd Amphitheater, U. H.

10:00 - 11:30 Surgery Conference; Todd Amphitheater, U. H.

10:00 - 12:50 Obstetrics and Gynecology Grand Rounds; J. L. McKelvey and Staff, Station 44, U. H.

11:30 - Anatomy Seminar; The Influence of Growth Hormone upon Fetal Development; Carl Heggestad; 226 Institute of Anatomy.

Ancker Hospital

8:30 - 9:30 Surgery Conference; Auditorium.

Minneapolis General Hospital

8:00 - Urology Staff Conference; T. H. Sweetser; Main Classroom.

9:00 - Psychiatry Grand Rounds; R. W. Anderson; Station H.

11:00 - 12:00 Medical - X-ray Conference; O. Lipschulz, Thomas Lowry and Staff; Main Classroom.

Veterans Administration Hospital

8:00 - Proctology Rounds; W. C. Bernstein and Staff; Bldg. III.

8:30 - Medical X-ray Conference; Conference Room, Bldg. I.