DERMATOMYOSITIS[1]

A Discussion of the Recent Literature and Report of Two Cases

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WITH the advent of modern laboratory techniques, new light is thrown on the diseases of muscle. Prominent among muscular disorders is a large and important group which is closely allied to the collagen diseases. Dermatomyositis, formerly considered rare by many authors, has been reported in the literature with increasing frequency and has become an important member of this group of collagen disorders. This paper is a report and comment on two cases of dermatomyositis.

Case Reports

Case 1. This is a 27-year-old negro female who was well until two months prior to hospital admission. She was admitted to Chicago Wesley Memorial Hospital on 7/3/57. The first evidence of illness was a progressive bilateral and symmetrical weakness of the arms and legs and generalized muscle aching. The weakness was more noticeable in the proximal rather than in the distal portions of the extremities, although the latter were also involved. She complained of slight difficulty in swallowing and general malaise. There was no history of chills, fever, infection or skin disease. There was no family history of such illness.

Examination revealed a thin, febrile negro female who appeared chronically ill. She was unable to turn in bed and there was profound weakness of all extremities. She was unable to lift her head from the pillow. The muscles most profoundly involved were the sternocleidomastoids, the deltoids, pectorales, trapezii, supra- and infraspinati, the biceps, triceps, quadriceps femoris and the hamstrings. The involvement was symmetrical. There were no fasciculations. Respirations were adequate. Swallowing was done with slight subjective difficulty. Cranial nerves were otherwise intact. The deep tendon reflexes were absent throughout. The Hoffman sign was present bilaterally. Coordination was good except where limited by weakness. There was generalized muscle tenderness, especially in the deltoids. There were no skin lesions. Muscle tone was generally decreased and the muscles had a flabby, doughy consistency. Sensory examination was normal throughout.

There was a grade two rough systolic murmur over the entire precordium. The heart tones were distinct. The liver edge was barely palpable. No spleen or other organs or masses were palpable.

Laboratory Data: X-rays of the chest, esophagus, hands, knees, ankles, feet, colon, stomach, duodenum were all reported to be normal. No subcutaneous or muscular calcifications were seen on any of the films.

The white count was consistently elevated, ranging between 12,000 and 18,000 cells per cu. mm. The differential count was normal except for an original eosinophilia of 12% which fell to 5.5% after therapy. No sickling was noted. The sedimentation rate was consistently elevated. Urinalysis revealed proteinuria, a few red and white cells. Seven L. E. preps were negative. Serum transaminase is shown in Table I (normal values are

TABLE I

SERUM TRANSAMINASE

Date	7/11/57	7/29/57	8/11/57	9/27/57
Units	850	890	420	110

[1]From the Department of Neurology and Psychiatry, Northwestern University Medical School. Presented at the Chicago Neurological Society, May 13, 1958. Received for publication, December 29, 1958.
[2]The authors wish to thank Dr. Frederick Lestina and Dr. Samuel Fogelson for their cooperation in this study.

TABLE II

LIVER PROFILE STUDIES

Date	7/5/57	7/11/57	8/3/57	8/22/57	9/5/57	9/16/57	9/19/57
Test							
Thymol turbidity				20.3	25.5	20.2	
Cephalin Floculation				3+		4+	
Alkaline Phosphatase mg.%				16.1	8.69		
B. S. P. %					2.2%		4.8%
Total Protein gm.%	6.10	6.5	6.5			7.91	
Albumin gm.%	3.2	3.5	3.3			2.52	
Globulin gm.%	2.9	3.0	3.2			5.39	
Cholesterol mg.%		147					

up to 50 units). Spinal fluid transaminase was 13.5 units on 7/11/57. Urine porphyrins were all negative. ASO titer was negative. Spinal fluid examination revealed an opening pressure of 180 mm. of water and after removal of 10 cc. of cerebrospinal fluid the pressure fell to 146 mm. of water. Spinal fluid protein was normal; the gold curve was flat; there were no cells, and the serology was negative. Creatine clearance tests revealed an increased excretion of Creatine. B.M.R. studies were plus 54% and plus 60%. Protein bound iodine was 3.8 micrograms per 100 cc. Radioactive iodine uptake studies revealed a 24-hour uptake of 47.5, a conversion ratio of 16.1%. This is in the upper limits of normal. An electrocardiogram was normal. See Table II for liver profile studies. Serum electrophoresis studies done on 10/2/57 are given in Table III.

Electrical Testing: Electrodiagnostic studies of the right biceps and deltoid muscles revealed a rheobase of 8 milliamperes and a chronaxie of 10 milliseconds in both muscles. The repetitive stimuli curve was ascending with a tetanus ratio of three. Electromyographic studies of both muscles showed no fibrillations or fasciculations. There were many action potentials present during voluntary motion.

Muscle Biopsy: Muscle biopsy revealed a moderate number of inflammatory cells composed of segmented neutrophils and occasional lymphocytes. Round cells were seen infiltrating the individual fibers. These were particularly prominent around the small vascular spaces. There were degenerative changes of the individual muscle fibers, swelling of the sarcoplasm and in some areas perforation of the sarcolemmal nuclei. The sarcoplasm of the cells was generally eosinophilic in staining quality. In other areas, where the nuclei were proliferating, the sarcoplasm was slightly basophilic.

Hospital Course: The patient was placed on 30 mg. of Meticorten daily. When the muscle tenderness subsided, physiotherapy was begun. She showed a gradual improvement in her general condition and regained much of her strength. The entire hospital stay was uneventful except for an attack of acute cholecystitis on 8/9/57. This was treated conservatively. Cholelithiasis was revealed on a subsequent cholecystogram. Throughout the hospital course she maintained a low-grade fever. The patient was discharged from the hospital on 9/5/57.

Nine days after discharge, she returned to the hospital with a six-day history of recurrence of generalized weakness, dysphagia, generalized muscle tenderness and malaise.

Physical findings were essentially the same as on the previous admission and the picture was that of an exacerbation of the former condition. Again, there were no cutaneous lesions. Laboratory findings at this time are incorporated in Table I.

TABLE III

SERUM ELECTROPHORESIS STUDIES

	Pt. (Gm.%)	Normal (Gm.%)
Albumin	1.40	3.2–3.7
Globulin		
Alpha 1	0.26	0.3–0.5
Alpha 2	0.97	0.7–1.0
Beta	0.69	0.0–1.1
Gamma	4.59	1.1–1.6

Treatment was continued with 30 mg. of Meticorten daily, and again physiotherapy was employed when the muscles were no longer tender. The patient made a good recovery. There was considerable return of strength. She was discharged on 10/13/57, able to walk and to use her upper extremities for most ordinary tasks.

Case 2. This patient is a 45-year-old white female who entered Chicago Wesley Memorial Hospital on 12/17/57 for an evaluation of generalized weakness present since 1952.

Six years prior to admission the patient experienced a rather sudden onset of fatigue, malaise, and pain in the thighs. There were no precipitating factors. Weakness, especially of the legs, followed a day later. She received a shot of penicillin which produced an allergic reaction and which was followed by more severe pain in her thigh muscles, accentuated by movement. She had a low-grade fever. The symptoms progressed so that in a few weeks she was unable to walk. These events were accompanied by a swelling of her ankles and thighs with redness of the overlying skin. These latter changes soon subsided.

The patient has remained weak, unable to walk without assistance. During the summer of 1957 she contracted an upper respiratory infection. Subsequently, the muscular weakness increased so that soon she was unable to sit up without assistance and could not get out of bed at all. Her weight has fluctuated. She lost 10 pounds of weight during the summer of 1957.

A positive serology was discovered in 1942 on routine examination. She was treated with Neosalversan and penicillin for three years. In 1945 her blood serology was negative. The spinal fluid serology was not determined. (Surgical history includes an appendectomy in 1949 and a hysterectomy for fibroids in 1953.)

A review of systems revealed that dysphagia had been present intermittently for six years and blurred vision had been noticed on occasion for one year prior to admission.

The family history was negative for a similar disease.

Physical Examination: The physical examination revealed a hirsute bronzed woman who appeared chronically ill. There was generalized wasting of the musculature. The temperature was 99.4F. orally. The skin around the fingernails was atrophic, shiny and fissured. There were non-pigmented striae in the skin over the lower thighs. The skin over the thighs was warm to touch; indeed, it was hot over the left knee.

Neurological Examination: There were no cranial nerve deficits. All modalities of sensation were intact to careful examination. There was some ataxia on the finger-to-nose test bilaterally, but it was felt that this was due to weakness.

Detailed muscle evaluation revealed profound weakness and atrophy of the following muscles: triceps, biceps, forearm muscles, including the brachioradialis and pronators of the wrist, and the small muscles of the hands. Similar findings were obtained in the examination of the abductors, adductors, internal rotators and external rotators of the thighs, in the quadriceps, hamstring muscles, triceps surae and the invertors and evertors of the feet. The pectoralis major muscles were similarly affected. The supinators of the wrists and the deltoid muscles were only a little stronger. The rectus abdominus, psoas complexes, dorsiflexors of the feet and the right extensor hallucis longus were moderately weak. The only muscle tested that was even moderately strong was the left extensor hallucis longus. The patient was unable to walk or even to sit up alone. She was able to comb her hair and feed herself with difficulty. All muscle deficits mentioned above were bilateral and quite symmetrical. All limbs were hypotonic. No fasciculations were observed and no clonus was elicited. The deep tendon reflexes were absent, even with reinforcement, except for a trace of the left triceps and both knee jerks. There were no pathological reflexes.

Laboratory Studies:

Electrodiagnostic Studies are shown in Table IV.

Electromyographic studies: In all three muscles studied in Table IV, there was no activity at rest. On motion, action

TABLE IV

	Right Biceps	Right Ext. Dig. Comm.	Right Tibialis Ant.
Rheobase (ma.)	8	10	10
Chronaxie (msec.)	20	less than 1	5
Tetanus Ratio	3	3	3
Repetitive Stimuli	Flat	Flat	Flat

potentials were present with a duration of 4 msec. and an amplitude of 150 mv. No fibrillation potentials were seen.

A lumbar puncture was done. The opening pressure was equivalent to 165 mm. of water. After 10 cc. of fluid had been removed, the pressure fell to 90 mm. of water. The spinal fluid protein was 37.5 and 30.5 mg.% in tubes I and III respectively. The colloidal gold curve was flat. The serology was negative. There was 1 monocyte in the spinal fluid.

An electrocardiogram showed premature ventricular beats, but was otherwise normal.

The chest x-ray was normal.

Blood chemistries: FBS 80 mg.%; NPN 16.1 mg.%; calcium 5.0 mg.%; phosphorus 1.6 mg.%; total protein 7.7 gm.%; albumin 3.6 gm.%; globulin 4.1 gm.%; sodium 138 meq./L.; potassium 4.48 meq./L.; chloride 104.1 meq./L.; BSP 5% retention at 45 min.; transaminase 94 units; electrophoresis of the serum proteins: albumin 2.6 gm.%; alpha$_1$ .3 gm.%; alpha$_2$ .9 gm.%; beta .8 gm.%; gamma 3.1 gm.%; creatinine: 0.8 mg.%.

Hematology: WBC were always normal. On two determinations there was a 3% and a 6% eosinophilia. Three LE preparations were negative. Sedimentation rate was 38 mm. per hr.

Urine studies: Three porphyrin determinations were negative. Urinalysis revealed 20 to 30 WBC/hpf. A urine culture grew out *E. coli communis*. Creatine tolerance tests: In 650 cc. of urine excreted in 24 hours there was 253 mg. of creatinine and 423 mg. of creatine.

BMR was −5%.

Hospital Course: A muscle biopsy of the left biceps was performed on 1/4/58. Microscopic sections of the tissue revealed dilated vascular channels surrounded by infiltrations of round cells and proliferating fibroblasts. These infiltrations extended among the muscle fibers.

The patient was placed on Meticorten 2.5 mg. b.i.d., Chloromycetin 250 mg. q.i.d. and one capsule of Surbex with C daily. Physiotherapy was begun. The improvement in muscle strength was slight.

Discussion

Pathology: The pathology of dermatomyositis was first described by Wagner in 1863 (25). He stated that this disease was a "rare disease" and reported a case originally diagnosed as periostitis of the upper extremities. Grossly there was patchy atrophy of the muscles with linear hemorrhages. Microscopically the muscle fibers were undergoing waxy degeneration. There was proliferation of the sarcolemmal nuclei and infiltration of the interstitial tissues with round cells. Wagner believed this to be a primary disease of muscle. The pathology of dermatomyositis has been described more recently by Aird (1). Grossly the muscles are pale with small hemorrhagic spots. Early the muscles are swollen; later they are shrunken, firm, fibrotic, and may be friable. Microscopically, there is first a patchy, non-specific inflammatory reaction. There follows a proliferation of the sarcolemmal nuclei with loss of the transverse striations and separation of the myofilbrils. Following this there are degenerative changes consisting of fragmentation, vacuolation, capillary thickening, serous exudates, phagocytosis, cellular infiltrates, hemorrhages and vascular occlusions. Finally, there is fibrous replacement with involvement of neighboring nerve endings, subcutaneous tissue and skin.

The pathology of the skin is grossly one of subcutaneous edema. Microscopically there are perivascular round cell infiltrations in the cutis (18). The interstitial lymphorrhages are thought to be a secondary phenomena, the purpose of which is the phagocytosis of degenerating muscle fibers (2).

Etiology: The etiology of dermatomyositis has been discussed for almost a hundred years and still has not been determined. Many etiologic factors have been suspected. Among these are viruses, allergic phenomena, reactions to various drugs such as penicillin (13), malignancy (21, 24, 5), infection, reticuloendothelial disturbance (23), and protozoa (11), to mention a few. In recent years, evidence has accumulated which places dermatomyositis among the so-called collagen disorders (12). Nevertheless, the etiology of dermatomyositis, and indeed the other collagen diseases, remains obscure.

Symptomatology: The clinical picture of dermatomyositis is frequently that of an acute febrile illness, accompanied by malaise, weakness and myalgia. However, the illness may be subacute with an insidious onset of muscular weakness and pain. The course is usually progressive and muscular involvement may vary widely in location and severity. The proximal muscle groups are more often involved than the distal. Muscular involvement may involve the pharyngeal, palatal, laryngeal, diaphragmatic intercostal, ocular, and cardiac muscles, in order of frequency (18). This involvement gives rise to the varied symptomatology of dysphagia, dysphonia, dyspnea and diplopia. There also may be paresthesias, hyperesthesias, numbness and pain. The deep tendon reflexes are usually decreased or absent. As the disease progresses, there is generalized muscle wasting, weight loss and inanition. The course of the disease is characterized by remissions and exacerbations which may be as much as two years apart. Approximately 50% of the patients die within the first two years. If the patient lives six years, it is unlikely that he will succumb to this malady. Death is usually due to respiratory failure, pneumonia, pericarditis, or myocardial dilitation. The disease must be differentiated from progressive muscular dystrophy, trichinosis, scleroderma, lupus erythematosis, polyneuritis, myasthenia gravis and acute rheumatic fever, to mention a few.

Diagnosis: Although the clinical picture is distinctive, the physician depends heavily on laboratory studies for confirmation of the diagnosis.

The white count is commonly elevated, rarely above 20,000. The differential count reveals an eosinophilia in about 25% of the cases (12). The sedimentation rate is elevated and, as the disease progresses, anemia develops.

The urine frequently shows evidence of a systemic disease, and contains protein, white cells, red cells and occasionally casts. Urine creatine is elevated as shown by the creatine tolerance tests.

The serum glutanic oxalacetic transaminase has been shown to be elevated to exceedingly high levels (19). However, spinal fluid transaminase has been shown to be unrelated to the serum transaminase level and does not rise in dermatomyositis (14). The total serum protein is elevated, with the albumin-globulin ratio reversed. Electrophoresis of the serum proteins reveals an increased gamma globulin fraction. It is the elevation of this fraction which accounts for the high serum globulin and elevated total protein. There are frequently alterations in the liver profile indicating parenchymal damage (i.e., elevated thymol turbidity, cephalin flocculation, etc.).

In an acute episode it is not uncommon to find the basal metabolism rate elevated in the face of normal radioactive iodine uptake studies and normal protein-bound iodine determinations (9).

Spinal fluid studies are generally normal. Roentgenograms in the acute phase are normal. In the chronic phase one may see calcium deposits in the muscles and subcutaneous tissues, with or without an associated osteoporosis.

The electrocardiogram may show varying degrees of myocardial damage.

Electrodiagnostic and electromyographic studies while not diagnostic are frequently helpful. The electromyographic studies show an increased irritability of muscle to insertion of the needle electrode and fibrillation potentials at rest. In voluntary motion there is an increased number of motor unit potentials, relative to the strength of contractions, than would obtain in normal muscle. Finally, there is an increased proportion of sharp spikes and polyphasic potentials of low amplitude (9). Electro-

diagnostic studies will reveal evidence of peripheral nerve involvement, usually incomplete, if the process has involved the nerve endings in the muscle.

Muscle biopsy is an invaluable adjunct in the diagnosis. Changes will be found as described above, compatible with the phase of the disease.

Treatment: There is no specific treatment for dermatomyositis. Therapy must be symptomatic, supportive and rehabilitative. An important cornerstone in therapy is the adrenal cortical and corticotrophic hormones. ACTH may be given intramuscularly in divided doses of 80 to 120 mg. daily. In acute dermatomyositis, 20 to 40 mg. may be given intravenously over a 12-hour period (12). Cortisone may be used. More recently Meticorten has been employed in doses of 20 to 40 mg. daily in divided doses. The best results with steroids are obtained in acute fulminating cases. Results in chronic cases are less dramatic, though steroids are still of value (16). Testosterone proprionate in oil may be given intramuscularly twice weekly in doses of 50 mg. each. Aspirin is used freely for pain and fever.

On empiric grounds, many therapeutic agents have been reported to be of value. These include penicillin (1). There are reports of penicillin precipitating, exacerbating, and alleviating the disease. At the present time, penicillin cannot be considered an important chemotherapeutic agent in the treatment of dermatomyositis.

Another cornerstone of treatment is physiotherapy and rehabilitation, to prevent contractures of fibrosed muscles and to make best possible use of the remaining, healthy muscle fibers.

Finally, the patient must be continually encouraged and supported through the course of this chronic illness. The physician must continually maintain an attitude of optimism to provide hope to the patient who has a potentially crippling disease.

Comment: The two cases herein presented are the antipodes of this disease. The first case is one of acute dermatomyositis in which the skin changes were minimal. The presence of multisystem involvement, as evidenced by the hepatic, renal, and hematologic abnormalities, together with generalized muscular pathology, places this case in the group of collagen diseases. Clinically, however, it falls into the nosologic group of the polymyositidies. More modern thinking is that the basic pathology of acute dermatomyositis and acute polymyositis is the same: a disorder of collagen (1, 2). If the clinical involvement is chiefly muscular, the term polymyositis may be used. When there is additional involvement of the skin, the term dermatomyositis is more applicable (3). Likewise, there are associated conditions of neuromyositis (22) in which there is involvement of both muscle and nerve endings, and, finally, fibromyositis in which the involvement includes muscle and connective tissue hyperplasia.

The second case represents the end stage of this collagen disorder. It may be correctly termed a chronic dermatomyositis or a chronic polymyositis. Some authors prefer to use the term chronic polymyositis for similar changes in muscle, secondary to a variety of systemic disease including neoplasm (2).

Although not present in either of our cases, calcinosis universalis is frequently associated with chronic dermatomyositis (5, 10).

Many authors have reported the coexistence of malignant disease with dermatomyositis (21, 24, 5). These two entities may appear one before the other, or simultaneously. This association was present in 6.7% of a large series of cases (5, 21).

In acute fulminating diseases of the muscle, the serum transaminase rises to great heights and is proportional to the activity of the destructive process. Levels may be low or normal in chronic, though disabling, muscle disease (19). This is well illustrated by our two cases. The serum transaminase was eight times normal in the first case, falling to a two-fold elevation after treatment with Meticorten. In the second case, though the patient was severely disabled, the level of transaminase was not even twice normal. Thus, the serum transaminase may be used both in the differential diagnosis of muscular and neural diseases,

and to follow the course of the disease and the results of therapy (19).

The elevation of the basal metabolism rate in acute polymyositis has been considered an important diagnostic finding. This occurs without concomitant alteration in other tests of thyroid function (9). The reason for the increased basal metabolism rate in this disease is yet to be elucidated. However, it is likely that this increase in basal metabolism rate is a reflection of increased oxygen consumption not related to thyroid abnormality.

The elevation of the serum gamma globulin is present in both of our cases and is a manifestation of reticuloendothelial activation. It bears further study. The eosinophilia is a well-known finding, occurring in the collagen disorders and allergic phenomena.

Summary

1. Two cases of dermatomyositis have been reported.

2. The basic pathology of dermatomyositis, polymyositis, neuromyositis and fibromyositis has been described and discussed. They are allied diseases and are considered part of a large group of collagen disorders.

3. A discussion of the nosology, laboratory findings and treatment of dermatomyositis has been presented.

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