Malnutrition induced Dermatomyositis

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Abstract

This is a case report of a 78 year-old woman whose diagnosis was dermatomyositis. Her illness responded well to nutrition correction.

INTRODUCTION

Dermatomyositis is an inflammatory myopathy with characteristic skin manifestations. The disorder is rare with a prevalence of one to 10 cases per million in adults. Early recognition and treatment are important ways to decrease the morbidity of systemic complications. So far the etiology of this disease is attributed to histocompatibility antigens, environmental agents like viruses and drugs, malignancy and autoimmunity ($_1$).

Muscle weakness and sometimes pain often but not always are symmetrical and proximal and are caused by muscle damage. This implies that muscle enzymes are liberated, electromyography is changed and lymphocytes gather around and in muscle cells and also around vessels. MRI has said to show characteristic series of patterns in inflamed muscles. The fat-suppressed T2 (STIR) image may show patchy bright signals characteristic of the edema that accompanies the inflammation myositis. Differential diagnoses of myositis are presented in table- 1($_2$).

Figure 1

Table 1: Differential Diagnosis of myositis

Neuromuscular disorders
Genetic muscular dystrophies
Spinal muscular atrophies
Neuropathies: the Guillain-Barré syndrome and other autoimmune polyneuropathies, diabetes mellitus, porphyria
Myasthenia gravis and the Eaton-Lambert syndrome
Amyotrophic lateral sclerosis
Myotonic dystrophy and other myotonias
Familial periodic paralysis
Endocrine and electrolyte disorders
Hypokalemia, hypercalcemia or hypocalcemia, hypomagnesemia
Hypothyroidism, hyperthyroidism
The Cushing syndrome, Addison disease
Metabolic myopathies
Familial periodic paralysis
Disorders of carbohydrate metabolism: McArdle disease, phosphofructokinase deficiency, adult acid maltase deficiency, and others
Disorders of lipid metabolism: carnitine deficiency, carnitine palmitoyl transferase deficiency
Disorders of purine metabolism: myoadenylate deaminase deficiency
Mitochondrial myopathies
Toxic myopathies
Alcohol
Chloroquine and hydroxychloroquine
Cocaine
Colchicine
Corticosteroids
Ipecac
Lovastatin and other lipid-lowering agents
Zidovudine
Infections
Viral: influenza, Epstein-Barr virus, human immunodeficiency virus, Coxsackievirus
Bacterial: staphylococcus, streptococcus, clostridia
Parasitic: toxoplasmosis, trichinosis, schistosomiasis, cysticercosis
Miscellaneous
Polymyalgia rheumatica
Vasculitis
The eosinophilia–myalgia syndrome
The paraneoplastic syndrome

Malnutrition which is said to be a predisposing factor can have overlap symptoms with dermatomyositis. Almost all organs are affected by malnutrition in the elderly who have a combination of poor appetite, difficulty in eating, and poor gastric absorption([3]).

THE CASE

A 78 year old woman with a history of two years of general itching had the chief complaint of muscle stiffness and pain. She developed skin erythema on the extensor and flexor aspect of her forearm and around her eyes after the month of Ramadan (fasting month of Muslims). She complained about pain in PIP joints, wrist, and shoulders. In physical examination these areas had 1+ tenderness with normal force (4/5+). She had no hepatosplenomegaly and no gross weight loss (her weight was between 35 - 38 kgs- in the past 10 years). She had 1-1.5 cm lymph node in her left arm pit palpable. She was anorexic, had dry cough and had pressure sores in her sacral area and sores in her mouth. Her life style analysis revealed that she was suffering from malnutrition after extracting her teeth. She had fatty liver in sonography and low level of albumin in her serum proteins panel. She was an obsessive-compulsive lady with an underlying depression.

She was hospitalized for further investigations to rule out malignancy-induced dermatomyositis.

She fulfilled DM criteria (table-2)([4]).

Figure 2

Table 2: Classification Criteria for Dermatomyositis*

1- Skin lesions
Heliotrope: red-purple edematous erythema on the upper palpebra
Gottron's sign: red-purple keratotic, atrophic erythema or macules on the extensor surface of finger joints
Erythema on the extensor surface of extremity joints, slight raised red-purple erythema over elbows or knees
2-Proximal muscle weakness (upper or lower extremity and trunk)
3-Elevated serum creatine kinase or aldolase level
4-Muscle pain on grasping or spontaneous pain
5-Myogenic changes on electromyography (short-duration, polyphasic motor unit potentials with spontaneous fibrillation potentials)
6-Positive anti-Jo-1 antibody test (histidyl-tRNA synthetase)
7-Nondestructive arthritis or arthralgias
8-Systemic inflammatory signs (temperature: more than 37°C [98.6°F] at axilla, Elevated serum C-reactive protein level or ESR of more than 20 mm per hour by Westergren)

9-Pathologic findings compatible with inflammatory myositis

*--Patients presenting with at least one finding from item 1 and four findings from items 2 through 9 are said to have dermatomyositis

Her EMG and NCV showed shoulder and pelvic girdle (paravertebral, pelvis and deltoid) acute myopathic process. Left thigh muscle biopsy also revealed myositis .

Malignancy was yet to be proved as all tests (lymph Biopsy, GI Endoscopy, Pap Smear, Sonograhy, Chest X-ray , HRCT and tumor markers) were not indicative of any malignancy. The only tumor marker which showed high level was CA 15-3 (breast cancer indicator) that was 72 u/ml and remained 72 by the next test after one month and 30.7 u/ml after 7 months from the initiation of therapy. No immunologic antigen(Anti Scl, Anti Rnp, Anti Ssa, Anti Jo1, Anti Sm) was positive. She had scant muscle mass that made CPK not reliable on its absolute value. Aldolase was within normal range.

Her lab results changed according to her progress. Her CPK level was used to taper steroid level (table 3).

Figure 3

Table 3: Lab results based on dates and events

date	3/31	7/26	9/8	10/9	12/4	1/8
event	Before fasting	In the hospital before treatment	30 mg/d prednisolone dizziness	20 mg/d pred. diarrhea	15mg/d Pred. Sore inside the mouth,cystitis	7.5 mg/d pred. Left Knee pain due to trauma and 100 mg celebrex consumption
WBC /µl	6570	6400	8100	8600	18000	7800
RBCx 10*6/µl	4.36	4.36	4.17	4.15	3.83	5.02
HB g/dl	13.2	10	11.9	12.9	11.9	14.8
HCT %	37.7	37.7	35.4	37.7	35.5	41.1
MCV fl	86.5	80	84.9	90.8	92.7	82
ESR 1st hr	23	25	20	5		72
FBS mg/dl	76		80	68		75
TG mg/dl	88		152	124		115
HDL mg/dl	52		75	75		36
LDL mg/dl	96		152	209		130
RATIO	1.9		2	3		3.6
SGOT IU/L	34	62	38	39		17
SGPT IU/L	20	34	54	59		7
ALP IU/L	153					150
LDH IU/L	260	653	241	613		336
CPK U/L	135	81	35	30		28
Na meq/l	140			137		144
K meq/l	4.1			4.6		4.2
Cal mg/dl	10.3	8.7	9.1	8.8		9.1
Aldolase		4.1	2.7	6		2.7
P mg/dl		3.4	2.9			3
RF	negative	negative	positive	positive		positive
BUN mg/dl		28	28	28		22
Cr mg/dl		1	0.6	0.8		0.6
CA 15-3 U/ml		72	72			30.7
S/E (OB)	NL	NL	NL	NL	NL	NL
CBC differential	NL	NL	NL	NL	NL	NL

She was prescribed prednisolone 1mg/kg/d. Within some days of nutritional intervention, her pain subsided and she found appetite to ingest enough food blended into liquid form for better absorption. She was also given Ranitidine and Calcium+vit D and Osteofos to prevent Corticosteroid

side effects.

The Mismanagements, or "over managements " prevented:

1- She was administered MTX despite rapid improvement in her condition after taking prednisolone.

It is written in rheumatologic texts that MTX should be started when the patient is not responsive to corticosteroid.

2- On high dose prednisolone, the patient developed dizziness (due to middle ear electrolyte changes) which caused her to fall down twice severe enough to warrant emergency department admission to rule out head trauma and hip fracture. Tapering Corticosteroids to the lowest level of effect was wrongly hesitated.

3-The next mismanagement was for her cholesterol level which rose to borderline levels because of her nutritionist order of taking omega 3 pearls (which are needed during steroid therapy). But her LDL/HDL ratio was normal. Statin (a cause of drug –induced myositis ($_4$) was administered to lower the borderline level cholesterol . While the first step in lowering cholesterol is by diet, prescribing Statins seems to be a mismanagement.

4- According to her lifestyle, the main focus of therapy became correcting nutrition and providing enough rest to the muscles. She was given VM protein (table 4), Cal+vit D, and vit B1 (for left foot neuropathy developed after quadriceps biopsy or as a process of the disease).

Figure 4

Table 4: VM protein ingredients in one 15-gr sachet prescribed per day:

Vit A acetate	1250 I U
Vit D3	125 IU
Vit E	2.5 I U
Vit B1	1.25 mg
Vit B2	1.25 mg
Vit B6	0.125 mg
Vit B12	0.5 mcg
Vit C	12.5 mg
Nicotinamide	3.75 mg
Calcium Pantothenate	1.25 mg
Cacium(as Phosphate and Caseinate)	207 mg
phosphorous	30.5 mg
Calcium Caseinate	10.5 gr
Iron	2.5 mg
L-Lysine	0.65 g
Choline bitartrate	25 mg
Inositol	12.5 mg
Copper	0.25mg
Iodine	0.025 mg
potassium	7.5 mg
Manganese	0.25 mg
Zinc	0.125mg
Magnesium	0.25mg
Sucrose	4 gr
Total protein	60%
Sodium	0.15%
Fat	2%

Prednisolone was tapered to 5 mg per day after 7 months. She started to gain weight and recovered pressure sores, mouth sores, anorexia, dizziness and depression. The skin rashes disappeared. There was no positive finding for myositis in the previous site of muscle biopsy in MRI done for left knee trauma. Prednisolone could not be discontinued as further follow-up seem to be necessary and none of these changes seem conclusive to treat this case like a case of malignancy induced DM.

CONCLUSION

If we believe there is another subgroup of Dermatomyositis: a malnutrition-induced-dermatomyositis, can it be cured by correcting malnutrition? This deduction is based on the fact that DM caused by malignancy can be cured by eradication of the malignancy and the fluctuating nature of immunologically- induced- dermatomyositis can not be considered for malignancy-induced dermatomyositis once the malignancy is treated.

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