Malnutrition induced Dermatomyositis

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Citation

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 paralyses

Endocrine and electrolyte disorders
- Hypokalemia, hypercalcaemia or hypocalcaemia, hypomagnesemia
- Hypothyroidism, hyperthyroidism
- The Cushing syndrome, Addison disease
- Metabolic myopathies
- Familial periodic paralyses
- Disorders of carbohydrate metabolism: McArdle disease, phosphofructokinase deficiency, adult acid maltase deficiency, and others
- Disorders of lipid metabolism: carnitine deficiency, carnitine palmitoyltransferase deficiency
- Disorders of purine metabolism: xanthine 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 induced Dermatomyositis

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.

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).

Figure 2

Table 2: Classification Criteria for 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, Sonography, 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 J01, 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

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

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

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

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

References
Author Information

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