Myositis 101

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Myositis Association Annual Conference
Louisville, KY
Sept. 6 & 7, 2018
Disclosures

• Consulting:
  • Dynavax
  • Pfizer

• Off-label use:
  • Nothing is FDA approved other than steroids
I hope to answer some common questions:

- What is myositis?
- What causes myositis?
- What does myositis do to patients?
- How is myositis diagnosed?
- How is myositis treated?
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• How is myositis treated?
Myositis = muscle inflammation

a) Normal muscle
b) PM – endomysial inflammation
c) DM – perifascicular atrophy

Mammen, Nat Rev Neurol 2011; 7:343-54
Muscle

http://www.nooruse.ee/e-ope/opiobjektid/lihasfysioloogia/lihasfysioloogia_alused.html
Myositis = muscle inflammation

http://www.neuro.wustl.edu/neuromuscular/pathol/inflammation.htm
Myopathy = muscle abnormality

- Myopathy: general term for muscle abnormality
- Myositis: muscle inflammation
- Most of the muscle disorders discussed are caused by abnormality of the immune system
- Autoimmune: immune system directed toward self
- Sometimes immune system abnormality causes myopathy without inflammation
Idiopathic Inflammatory Myopathies

- Polymyositis (PM)
- Dermatomyositis (DM)

- Isolated, adult
- Juvenile
- Malignancy
- Overlap

Bohan et al., Medicine 56: 255, 1977
Idiopathic Inflammatory Myopathies

- Polymyositis (PM)
- Dermatomyositis (DM)

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- Juvenile
- Malignancy
- Overlap

- Inclusion body myositis (IBM)
- Antisynthetase syndrome
- Immune-mediated necrotizing myopathy (IMNM)
Idiopathic Inflammatory Myopathies
Immune-mediated

• Polymyositis (PM)
• Dermatomyositis (DM)

• Inclusion body myositis (IBM)
• Antisynthetase syndrome
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• **What causes myositis?**
• What does myositis do to patients?
• How is myositis diagnosed?
• How is myositis treated?
“Cause” is a difficult term

Correlation or association ≠ Causation!

Number of people who drowned by falling into a pool correlates with Films Nicolas Cage appeared in

“Cause” is a difficult term

**Disease triggers**
- Genetic factors
- Environmental factors
- Muscle fiber change

**Disease mechanisms**
- Immune system
- Inflammation
- Muscle damage
- Others?
Myositis is likely triggered by genetic and environmental factors
Genome-wide association study (GWAS) in DM/JDM

Genes related to the immune system’s recognition of foreign proteins are most highly associated with DM/JDM

Strongest association with MHC Non-MHC SNPs:
- PLCL1
- BLK
- CCL21

Miller et al., Arthritis Rheum 2013; 65: 3239-47
GWAS in PM and DM

Rothwell et al., Ann Rheum Dis 2016; 75: 1558-66
HLA-DRB1 in IBM

Rothwell et al., Arthritis Rheumatol 2017; 69: 1090-9
Classes of environmental exposures

- **Chemical factors**
  - Silica
  - Asbestos
  - Metals
  - Pesticides
  - Industrial chemicals and solvents
  - Air pollution
  - Smoking
  - Personal care products

- **Physical factors**
  - Ionizing radiation
  - UV radiation
  - Electric and magnetic fields

- **Biologic factors**
  - Infectious agents
  - Foods and dietary contaminants
  - Molds
  - Mycotoxins
  - Other toxins

# Pathogenesis of IIM

<table>
<thead>
<tr>
<th>Feature</th>
<th>DM</th>
<th>PM</th>
<th>IBM</th>
</tr>
</thead>
<tbody>
<tr>
<td>Increased MHC I</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>B-cell mechanisms</td>
<td>+</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>T-cell mechanisms</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Inclusions</td>
<td>-</td>
<td>-</td>
<td>+</td>
</tr>
<tr>
<td>Autoantibodies (Myositis specific antibodies)</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
</tbody>
</table>
MHC I expression in muscle

Normal

IBM

http://neuromuscular.wustl.edu/pathol/ibm.htm
Activated T cells (T lymphocytes) invade and damage muscle in IBM and PM

Arahata & Engel, Ann Neurol 1984; 16:193
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• How is myositis treated?
What does myositis do to patients?

- It depends
  - On the specific disorder
  - On the individual patient
- Muscle weakness is the hallmark
- Many other things can occur
Assessment of Muscle Weakness

• Patient report of effect on activities

• Manual muscle strength testing

• Physiologic testing

• Functional tests
  • Timed stands
  • 6 minute walk
  • Functional Index-2
Evaluation of Muscle Disease: Laboratory Tests

• CK – Creatine kinase (CPK)
• Aldolase
• LDH – Lactate dehydrogenase
• AST – aspartate aminotransferase
• ALT – alanine aminotransferase
Muscle MRI can show inflammation or scarring
Resting EMG

Normal muscle - no resting activity

Positive waves

Fibrillation potentials and occasional positive waves

Complex repetitive discharges

Myotonic discharge
Motor Unit Action Potentials (MUAPs)

A: Normal MUAPs

B: Myopathy: Low amplitude, short, polyphasic MUAPs

C: Neuropathy: Large, long duration, polyphasic MUAPs
Dermatomyositis: characterized by rash

• Heliotrope
• Gottron’s papules
• Shawl sign & others
• Calcinosis cutis
Heliotrope
Gottron’s papules
Gottron’s sign
Shawl sign
Periungual erythema
Calcinosis cutis
Amyopathic Dermatomyositis (Dermatomyositis siné myositis)

• Cutaneous features of DM
• No muscle weakness
  • Maybe elevated muscle enzymes or EMG, but not weak

Gerami et al., J Am Acad Dermatol 2006;54:597-613
Other organ system problems – mostly PM and DM

- **Lung** – interstitial lung disease
- **Gastrointestinal** – dysphagia, anorexia, reflux
- **Heart** – inflammation, fibrosis, rhythm
- **Joints** – arthritis, arthralgia, morning stiffness
- **Raynaud’s phenomenon**
- **Constitutional** – fatigue, fever
Autoimmune Connective Tissue Diseases

- Rheumatoid arthritis
- Sjogren’s
- Scleroderma
- Lupus
- PM/DM

Other terms:
- Overlap CTD
- Undifferentiated CTD
- Mixed CTD
Inclusion body myositis

• Muscle biopsy findings define the disorder

Myxovirus-Like Structures in a Case of Human Chronic Polymyositis

Abstract. Intranuclear and intracytoplasmic aggregates of filaments with tubular structures and transverse striations occurred in muscle tissues biopsied from a patient with chronic polymyositis. The filamentous tubules bear a close resemblance to the incomplete form of myxovirus in which the envelope is missing. Three biopsies from the same patient, taken during a period of 1½ years, all revealed these structures. This finding provides presumptive evidence that a chronic persistent viral infection may be involved in the pathogenesis of chronic polymyositis.

SHI-MING CHOU

Department of Pathology and Regional Primate Research Center, University of Wisconsin Medical School, Madison

15 December 1967
Inclusion Body Myositis

- First description 1967
- “IBM” term coined 1971
- Sporadic form (s-IBM)
- Several hereditary forms (h-IBM)
- Clinically similar:
  - Weakness: insidious, distal, atrophy
  - CK minimally to moderately elevated
  - EMG: myopathic +/- neurogenic
- Hereditary: younger; no inflammation
Clinical Features of sIBM

- Insidious onset
- ~6 years to diagnosis
- Weakness generalized or localized to limbs; may be asymmetric
- Reflexes normal initially, eventually diminished in 40%
- Dysphagia in 2/3 – late
- Myalgia uncommon but aching in thighs and knees in some

Typical involvement:
- Finger flexors
- Wrist flexors
- Knee extensors
- Ankle dorsiflexors

*Needham & Mastaglia, Lancet Neurol 6: 620-31, 2007*
Anti-synthetase Syndrome

- Anti-synthetase antibody – Jo-1, others
- PM/DM
- Interstitial lung disease
- Inflammatory arthritis
- Raynaud’s phenomenon
- Mechanic’s hands
- Fever
Mechanic’s hands
Immune-mediated necrotizing myopathy

- Characterized by muscle biopsy with necrotic muscle fibers without inflammation
- Specific autoantibodies
  - Anti-SRP
  - Anti-HMGCR
    - Often associated with statin use

# IIM: Epidemiology

### Incidence
- 0.5 to 8/million

<table>
<thead>
<tr>
<th>Condition</th>
<th>Age</th>
<th>Female:male</th>
</tr>
</thead>
<tbody>
<tr>
<td>Polymyositis/Dermatomyositis</td>
<td>Bimodal, 10-15 in kids, 45-60 in adults</td>
<td>2:1</td>
</tr>
<tr>
<td>Inclusion Body Myositis</td>
<td>&gt;50</td>
<td>1:2</td>
</tr>
</tbody>
</table>
## Epidemiology of IBM and PM
Olmsted County, 1981-2000*

<table>
<thead>
<tr>
<th></th>
<th>IBM</th>
<th>PM</th>
</tr>
</thead>
<tbody>
<tr>
<td>Incidence</td>
<td>0.79 (0.24-1.35)</td>
<td>0.41 (0.08-0.73)</td>
</tr>
<tr>
<td>Prevalence</td>
<td>7.06 (0.87-13.24)</td>
<td>3.45 (0.00-7.35)</td>
</tr>
</tbody>
</table>

*Age- and sex-adjusted rates per 100,000 population; (95% CI)

Wilson et al., J Rheumatol 2008; 35:445-7
Epidemiology of DM
Olmsted County, 1976-2007*

<table>
<thead>
<tr>
<th></th>
<th>DM</th>
<th>CADM</th>
</tr>
</thead>
<tbody>
<tr>
<td>Incidence</td>
<td>0.96</td>
<td>0.21</td>
</tr>
<tr>
<td></td>
<td>(0.61-1.32)</td>
<td>(0.04-0.38)</td>
</tr>
<tr>
<td>Prevalence</td>
<td>2.14</td>
<td></td>
</tr>
<tr>
<td></td>
<td>(1.31-2.98)</td>
<td></td>
</tr>
</tbody>
</table>

*Age- and sex-adjusted rates per 100,000 population; (95% CI)

Bendewald et al., Arch Dermatol 2010; 146: 26-30
Systematic Review: Adult IIM

Meyer et al., Rheumatology 2015; 54: 50-63
Systematic Review: sIBM

Meyer et al., Rheumatology 2015; 54: 50-63
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PM/DM Classification Criteria

• Proximal muscle weakness
• Elevated serum levels of skeletal muscle enzymes
• Myopathic changes on EMG
• Muscle biopsy evidence of inflammation
• Skin rash

*Definite PM or DM: 4 criteria*
*Probable PM or DM: 3 criteria*
*Possible PM or DM: 2 criteria*

Bohan et al., Medicine 56: 255, 1977
Muscle histopathology

a) Normal muscle
b) PM – endomysial inflammation
c) DM – perifascicular atrophy
d) Necrotizing myopathy

Mammen, Nat Rev Neurol 2011; 7:343-54
Myositis = muscle inflammation

http://www.neuro.wustl.edu/neuromuscular/pathol/inflammation.htm
IBM: Vacuoles
IBM Congo red staining

Askansas & Engel, J Neuropathol Exp Neurol 60:1, 2001
Antibodies

- Immunoglobulin
- Produced by plasma cells in the immune system
- Identify and neutralize viruses and bacteria
- Each recognizes a unique protein (antigen)
Autoantibodies

- Antibodies directed toward an individual’s normal proteins
- Autoantibodies may:
  - Cause disease
  - Simply be markers of disease

<table>
<thead>
<tr>
<th>Autoantibody</th>
<th>Target</th>
<th>Disorder</th>
</tr>
</thead>
<tbody>
<tr>
<td>Antinuclear antibodies (ANA)</td>
<td>Contents of cell nuclei</td>
<td>Lupus and related conditions</td>
</tr>
<tr>
<td>Rheumatoid factor (RF)</td>
<td>IgG</td>
<td>Rheumatoid arthritis</td>
</tr>
<tr>
<td>Anti-Jo-1</td>
<td>Histidyl tRNA synthetase</td>
<td>Polymyositis with ILD</td>
</tr>
<tr>
<td>Anti-PR-3 (c-ANCA)</td>
<td>Neutrophil proteinase-3</td>
<td>Granulomatosis with polyangiitis</td>
</tr>
<tr>
<td>Anti-thyroid antibodies</td>
<td>TPO, Thyroglobulin</td>
<td>Hashimoto’s thyroiditis</td>
</tr>
<tr>
<td>Anti-AChR</td>
<td>Acetylcholine receptor on muscle</td>
<td>Myasthenia gravis</td>
</tr>
<tr>
<td>Anti-TTG</td>
<td>Tissue transglutaminase</td>
<td>Celiac disease</td>
</tr>
</tbody>
</table>
### Non-specific Autoantibodies in Myositis

#### Percent of Patients with Various Autoantibodies

<table>
<thead>
<tr>
<th>Antibody</th>
<th>All (n=212)</th>
<th>PM (n=58)</th>
<th>DM (n=79)</th>
<th>CTM (n=36)</th>
<th>CAM (n=13)</th>
<th>IBM (n=26)</th>
</tr>
</thead>
<tbody>
<tr>
<td>ANA</td>
<td>52</td>
<td>40</td>
<td>62</td>
<td>77</td>
<td>31</td>
<td>23</td>
</tr>
<tr>
<td>ds-DNA</td>
<td>5</td>
<td>3</td>
<td>3</td>
<td>11</td>
<td>8</td>
<td>4</td>
</tr>
<tr>
<td>SSA/Ro</td>
<td>12</td>
<td>12</td>
<td>11</td>
<td>17</td>
<td>0</td>
<td>12</td>
</tr>
<tr>
<td>SSB/La</td>
<td>8</td>
<td>5</td>
<td>6</td>
<td>19</td>
<td>8</td>
<td>8</td>
</tr>
<tr>
<td>Sm</td>
<td>3</td>
<td>0</td>
<td>1</td>
<td>17</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>U1RNP</td>
<td>11</td>
<td>7</td>
<td>13</td>
<td>25</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>PM/Scl</td>
<td>2</td>
<td>0</td>
<td>4</td>
<td>3</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>RF</td>
<td>6</td>
<td>5</td>
<td>8</td>
<td>8</td>
<td>0</td>
<td>4</td>
</tr>
</tbody>
</table>

Love et al, Medicine 1991; 70: 360-74
# Myositis-Specific Antibodies

<table>
<thead>
<tr>
<th>Feature</th>
<th>Synthetase</th>
<th>SRP</th>
<th>Mi-2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clinical</td>
<td>Arthritis, ILD fever, Raynaud’s</td>
<td>Cardiac myalgias; black women</td>
<td>Classic DM</td>
</tr>
<tr>
<td>Rate</td>
<td>Acute</td>
<td>Very acute</td>
<td>Acute</td>
</tr>
<tr>
<td>Severity</td>
<td>Severe</td>
<td>Very severe</td>
<td>Mild</td>
</tr>
<tr>
<td>Season</td>
<td>Spring</td>
<td>Fall</td>
<td>Unknown</td>
</tr>
<tr>
<td>Response</td>
<td>Moderate</td>
<td>Poor</td>
<td>Good</td>
</tr>
<tr>
<td>Prognosis</td>
<td>Poor (70%)</td>
<td>Terrible (25%)</td>
<td>Good (~100%)</td>
</tr>
<tr>
<td>Frequency</td>
<td>20-25%</td>
<td>&lt;5%</td>
<td>5-10%</td>
</tr>
</tbody>
</table>
### Antisynthetase antibodies

<table>
<thead>
<tr>
<th>Antigen</th>
<th>tRNA synthetase</th>
<th>JDM*</th>
<th>ADM*</th>
<th>Non-white</th>
</tr>
</thead>
<tbody>
<tr>
<td>Any</td>
<td></td>
<td>1-5</td>
<td>30</td>
<td>AA 29</td>
</tr>
<tr>
<td>Jo1</td>
<td>Histidyl-</td>
<td>2-5</td>
<td>25-30</td>
<td>AA13</td>
</tr>
<tr>
<td>PL12</td>
<td>Alanyl-</td>
<td>1-3</td>
<td>&lt;5</td>
<td></td>
</tr>
<tr>
<td>PL7</td>
<td>Threonyl-</td>
<td>&lt;1</td>
<td>&lt;5</td>
<td>Japanese 17</td>
</tr>
<tr>
<td>EJ</td>
<td>Glycyl-</td>
<td>&lt;1</td>
<td>&lt;5</td>
<td></td>
</tr>
<tr>
<td>OJ</td>
<td>Isoleucyl-</td>
<td>&lt;1</td>
<td>&lt;5</td>
<td></td>
</tr>
<tr>
<td>KS</td>
<td>Asparagynyl-</td>
<td>NA</td>
<td>&lt;1</td>
<td></td>
</tr>
<tr>
<td>HA</td>
<td>Tyrosyl-</td>
<td>NA</td>
<td>&lt;1</td>
<td></td>
</tr>
<tr>
<td>ZA</td>
<td>Phenylalanyl-</td>
<td>NA</td>
<td>&lt;1</td>
<td></td>
</tr>
</tbody>
</table>

*Caucasian

Robinson & Reed, Nat Rev Rheumatol 2011; 7: 664-75
Serologic Subgroups in IIM 2

- **Anti-TIF-1γ (anti-transcription intermediary factor 1γ; anti-p155):** DM, including JDM, malignancy
  
  Targoff et al., Arthritis Rheum 2006; 54: 3682-3689
  Trallero-Araguas et al., Medicine 2010; 89: 47-52

- **Anti-MDA-5 (anti-melanoma differentiation-associated protein 5; anti-CADM):** CADM, rapidly progressive ILD
  
  Sato et al., Arthritis Rheum 2005; 52:1571-6

- **Anti-NXP2 (anti-nuclear matrix protein 2; anti-MJ):** JDM, especially with calcinosis, malignancy
  
  Gunawardena et al., Arthritis Rheum 2009; 60: 1807-14
Autoantibodies in IBM

Anti-cytoplasmic 5’-nucleotidase 1A

- Initially detected as 43 kd autoantibody
  - 13/25 (52%) IBM + vs 0/40 controls
  - Antigen identified as cN1A
  - Most abundant in skeletal muscle
  - Catalyzes nucleotide hydrolysis to nucleosides
  - Perinuclear and vacuole accumulation of cN1A


### Table 1: Sensitivity and specificity of anti-cN-1A autoantibodies

<table>
<thead>
<tr>
<th>Sera</th>
<th>Number</th>
<th>Anti-cN-1A reactivity*</th>
<th>Anti-cN-1A reactivity*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Inclusion body myositis</td>
<td>238</td>
<td>88</td>
<td>37</td>
</tr>
<tr>
<td>Polymyositis/dermatomyositis</td>
<td>185</td>
<td>8</td>
<td>4</td>
</tr>
<tr>
<td>Polymyositis/scleroderma overlap</td>
<td>12</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Neuromuscular diseases</td>
<td>93</td>
<td>4</td>
<td>4</td>
</tr>
<tr>
<td>Sjögren’s syndrome</td>
<td>22</td>
<td>8</td>
<td>36</td>
</tr>
<tr>
<td>Systemic lupus erythematosus</td>
<td>44</td>
<td>9</td>
<td>20</td>
</tr>
<tr>
<td>Scleroderma</td>
<td>44</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>Rheumatoid arthritis</td>
<td>44</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>Multiple sclerosis</td>
<td>40</td>
<td>2</td>
<td>5</td>
</tr>
<tr>
<td>Type 1 diabetes</td>
<td>40</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Disease controls†</td>
<td>458</td>
<td>16</td>
<td>3</td>
</tr>
</tbody>
</table>

*Reactivity with at least one of the three cN-1A peptides higher than cut-off.
†Disease controls: total of all disease control groups except IBM, SLE and SjS.
cN-1A, cytosolic 5’-nucleotidase 1A; IBM, inclusion body myositis; SjS, Sjögren’s syndrome; SLE, systemic lupus erythematosus.

Myositis Specific Autoantibodies

Betteridge & McHugh, J Intern Med 2015; Epub
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General Approach to Treatment of Myositis
PM, DM, ASS, Overlap myositis

Autoimmune process → Inflammation → Symptoms
General Approach to Treatment of Myositis
PM, DM, ASS, Overlap myositis

Autoimmune process → Inflammation → Symptoms

Damage

Inflammation → Damage
General Approach to Treatment of Myositis
PM, DM, ASS, Overlap myositis

Autoimmune process

Immune suppressing agents

Steroids

Inflammation

Symptoms

Damage
General Approach to Treatment of Myositis Immune-mediated necrotizing myopathy

Auto-immune process

Immune suppressing agents

Mechanism?

Damage

Symptoms

Steroids
General Approach to Treatment of Myositis Cancer-associated myositis

Cancer → Autoimmune process → Inflammation → Symptoms → Damage

Immune suppressing agents? → Steroids?
General Approach to Treatment of Myositis
Inclusion body myositis

Autoimmune process

Inclusions

Damage

Inflammation

Symptoms

Immune suppressing agents?

Steroids?
Approach to Management

- Start with high-dose prednisone (e.g., 1 mg/kg/day)
  - Consider IV to begin
  - Consider split daily dose
- Continue about 1 month with slow taper
- Use an immunosuppressive agent
- Attention to side effects of therapy (e.g., osteoporosis, infection)
My Approach to Prednisone

- Begin 1 mg/kg/d (usually max 80 mg/d)
- Continue 1 month
- 2 weeks each:
  - 40 mg/d
  - 30 mg/d
  - 25 mg/d
  - 20 mg/d
  - 17.5 mg/d
  - 15 mg/d
  - 12.5 mg/d
- 10 mg/d and then decide what next
# My Approach to Immunosuppressives

### First-line agents
- Methotrexate
- Azathioprine (Imuran)
- Mycophenolate mofetil (CellCept)

### Second-line agents
- IVIg
- Rituximab (Rituxan)
- Tacrolimus (Prograf)
- Cyclosporine A (Neoral, Sandimmune)
- Leflunomide (Arava)

### Severe disease
- Cyclophosphamide (Cytoxan)

### Studies
- Abatacept (Orencia)
- Tocilizumab (Actemra)
- Belimumab (Benlysta)

### Never used
- ACTHAR gel
Also remember

- Pneumocystis pneumonia prophylaxis when on high dose prednisone
- Influenza, pneumococcal, and other immunizations
- Osteoporosis attention: calcium and vitamin D; bone density
- Mobility and assistive devices; fall prevention
- Dysphagia
- Exercise
I hope to answer some common questions:

• What is myositis?
• What causes myositis?
• What does myositis do to patients?
• How is myositis diagnosed?
• How is myositis treated?
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Questions?