Inflammatory myopathies are characterised by proximal skeletal muscle weakness and evidence of muscle inflammation. These disorders are rare (Dermatomyositis/Polymyositis combined incidence of 2 per 100,000 annually). Myositis is generally not painful, with mild discomfort only if present but patients will have weakness on neurologic examination. Patients can also have systemic features like skin rash or interstitial lung disease.

Classification
Inflammatory myopathies form several groups according to clinical and pathologic features – all share immune-mediated muscle injury.

- Dermatomyositis
- Overlap syndromes (with another systemic rheumatic disease)
- Inclusion body myositis
- Immune mediated necrotizing myopathy
- Polymyositis
- Other rarer subtypes

Some patients cannot be subtyped into a specific category and are classified as having ‘non-specific’ myositis.

Pathology testing
General laboratory testing:
Inflammatory myopathy can increase muscle enzymes including lactate dehydrogenase (LD), creatine kinase (CK), aspartate aminotransferase (AST), and alanine aminotransferase (ALT).

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Statin associated myositis: important points
Statin lowers serum cholesterol for primary and secondary prevention of cardiovascular disease and dyslipidemia. A common side effect is muscle pain and weakness, typically seen as proximal, symmetric muscle weakness or soreness with difficulty raising arms above the head, rising from sitting and climbing stairs.

Symptoms usually start within weeks to months after statin initiation but can occur at any time.

In most cases, statin related myopathy resolves after medication is stopped. However, immune-mediated necrotising myopathy can persist after cessation and may respond to immunosuppression.

Potential effects can be divided into
- Myalgia - muscle discomfort with normal CK
- Myopathy - muscle weakness with or without increased CK
- Myositis - muscle inflammation and myonecrosis with increased muscle enzymes either from base line or compared to ULN
- Severe myonecrosis is uncommon (affects <0.5% of patients).

Necrotizing myopathy – reported to be histologically distinct, non-inflammatory statin related myopathy with macrophagocytic engulfment of necrotic muscle fibres. Patients respond to immune therapy and this is presumed to be autoimmune in origin.

- Antibodies to HMG CoA reductase have been seen in these patients.
- HMG CoA reductase antibodies however, can be seen in statin-naive patients (37-62% of patients with positive antibodies and myopathy were statin-naive in some papers, another paper reported 94% of patients with antibodies had statin exposure however).
- Adverse effects are not necessarily seen as a progression from less to more severe however necrotising myopathy and inflammatory myopathy may be a spectrum.

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