Clinical medicine historically differentiated polymyositis from dermatomyositis only by the presence of skin findings, although these two inflammatory myopathies are now known to be fundamentally different conditions with varying pathology and clinical consequences. An accurate methodology to differentiate between these two conditions is therefore critical.

Antisynthetase syndrome is a collection of symptoms that include myositis, interstitial lung disease, arthritis, constitutional symptoms, Raynaud’s phenomenon, and Gottron’s papules. Most commonly, antisynthetase syndrome presents with anti-Jo1 antibodies. In many patients, the symptoms of interstitial lung disease such as dyspnea can be the presenting symptom.

We present the case of a 38 year old Caucasian female that presented with a chief complaint of worsening cough productive of white phlegm. She also reported diffuse muscle weakness and edema in the lower extremities bilaterally. Her past medical history was significant for ARDS and multiple episodes of pneumonia. Her prednisone dose was currently being tapered.

Initial chest x-ray is shown in Figure 1. Positive ANA testing prompted further evaluation with the following results:

<table>
<thead>
<tr>
<th>ANA</th>
<th>AntiJo-1</th>
<th>ESR</th>
<th>CRP</th>
<th>CK</th>
</tr>
</thead>
<tbody>
<tr>
<td>Positive</td>
<td>Positive</td>
<td>75mm/hr</td>
<td>6mg/dl</td>
<td>6677U/L</td>
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</tbody>
</table>

Muscle biopsy was then performed. After bronchoscopy showed negative cultures, the patient was then started on solumedrol 125 mg and tacrolimus 1 mg daily. After an initial steroid dose, she began to show modest clinical improvement in muscle strength and significant improvement in pain control. CK levels decreased to 3913 U/L one day after steroid use.

These lab values, muscle biopsy, and the patient’s gradual clinical improvement with continued steroid use suggested a diagnosis of antisynthetase syndrome.

The patient showed increased strength of upper and lower extremities bilaterally, and was then discharged to acute rehabilitation therapy for continued care.

This case illustrates that the presence or absence of skin findings does not accurately differentiate between polymyositis and dermatomyositis. Cases of dermatomyositis with absent skin findings, or dermatomyositis sine dermatitis, can be mistakenly diagnosed as polymyositis.

Accurate differentiation between the inflammatory myopathies is crucial as dermatomyositis has a significantly increased risk of associated malignancy. Muscle biopsy results are essential to diagnosis. While dermatomyositis shows perifascicular atrophy (Figure 2), polymyositis exhibits the lymphocytic invasion of healthy appearing, individual muscle fibers.

The clinician may also face challenges in the diagnosis of antisynthetase syndrome because of its diverse presentation. Although there are multiple symptoms, not all of them are required or frequently seen. There can also be extreme variability in the timing between the presentation of myositis and interstitial lung disease.

Prognosis of antisynthetase syndrome greatly depends on the type, severity, and progression of the interstitial lung disease. Systemic glucocorticoids are started for patients with respiratory impairment. A second agent is often necessary because of the rapidly progressive nature of interstitial lung disease. Systemic glucocorticoids are started for patients with respiratory impairment.