HISTOLOGICAL PARASPINAL MUSCLE COMPARISON BETWEEN CURVE CONCAVITY AND CONVEXITY IN AIS

Marcelo Wajchenberg
Federal University of São Paulo - Brazil
Main causes: errors and postural muscle imbalance

First book of Orthopaedics: "L' Orthopedie "

Nicholas André 1658-1742
INTRODUCTION

IDIOPATHIC SCOLIOSIS

• Lateral deviation of the spine, with rotational shift, with no known cause in individuals with no neurological, muscular or other diseases.

• The onset / progression during the growth phase (Pubertal)
INTRODUCTION

Ethiology
Multifatorial Disease

– Connective tissue changes
– Central nervous system alterations
– Asymmetric growth
– Hormonal changes
– Decreased melatonin, growth hormone
– Heredity / Genetics
– Muscle imbalances

Lonstein JE. The Lancet; 1994 344: 1407-1412
OBJECTIVE

• Evaluate the muscle fibers of rotator muscles of the back at the apex of the curve of patients with adolescent idiopathic scoliosis, by histological and immunohistochemical analysis.
METHODS

• Muscles of collected 21 female patients operated.
• Signed informed consent form.
• Collected the multifidus muscles from concavity and convexity on the apex of the thoracic curve.
• Muscle followed by collection of the technique described by Schmidt et al.
• McNemar test (nonparametric variables) with significance level of 5%.

METHODS

• Muscle atrophy and hypertrophy.
• Fatty proliferation.
• Presence of endomysial and perimysial fibrosis, hyaline fibers.
• Mitochondrial proliferation
• muscle necrosis and nuclear centralization
• "Typegrouping" and inflammation
• classified as absent, sparse (up 25%), mild (50%), moderate (75%) and severe (above 75%)
RESULTS

Fibrosis and fatty proliferation

Hyaline fibers
RESULTS

Necrosis and nuclear centralization

Inflammation
RESULTS

Atrophy and mitochondrial proliferation "Core"!
## RESULTS

<table>
<thead>
<tr>
<th></th>
<th>concavity (%)</th>
<th>convexity (%)</th>
<th>“p” value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Necrosis</td>
<td>42.8</td>
<td>23.8</td>
<td>0.125</td>
</tr>
<tr>
<td>Muscular atrophy</td>
<td>52.3</td>
<td>38.0</td>
<td>0.453</td>
</tr>
<tr>
<td>Muscular hypertrophy</td>
<td>42.8</td>
<td>28.5</td>
<td>0.453</td>
</tr>
<tr>
<td>Fatty proliferation</td>
<td>85.7</td>
<td>47.6</td>
<td>0.039*</td>
</tr>
<tr>
<td>Endomysial fibrosis</td>
<td>81.0</td>
<td>47.6</td>
<td>0.016*</td>
</tr>
<tr>
<td>Periisial fibrosis</td>
<td>85.7</td>
<td>52.4</td>
<td>0.039*</td>
</tr>
<tr>
<td>Hyaline fibers</td>
<td>85.7</td>
<td>71.4</td>
<td>&gt; 0.999</td>
</tr>
<tr>
<td>Inflammatory tissue</td>
<td>14.3</td>
<td>9.5</td>
<td>&gt; 0.999</td>
</tr>
<tr>
<td>“core”</td>
<td>76.2</td>
<td>66.7</td>
<td>0.625</td>
</tr>
<tr>
<td>Mitochondrial proliferation</td>
<td>71.4</td>
<td>61.9</td>
<td>0.500</td>
</tr>
<tr>
<td>Type I fibers</td>
<td>89.5</td>
<td>88.1</td>
<td>0.110</td>
</tr>
<tr>
<td>Nerves</td>
<td>90.5</td>
<td>81.0</td>
<td>0.500</td>
</tr>
<tr>
<td>Nuclear centralization</td>
<td>61.9</td>
<td>47.6</td>
<td>0.375</td>
</tr>
</tbody>
</table>

* p < 0.05
MUSCULAR IMBALANCE

- Adams, XIX century – “Muscular debility”
CONCLUSION

The paraspinal muscles at the apex of the deformity had higher involvement in the concavity when analyzing the fatty involution and fibrosis. However, both sides showed signs of myopathy.