Superior oblique tendon sheath syndrome

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The superior oblique tendon sheath syndrome, originally described by Brown (1950), was thought to be caused by a congenital fascial anomaly of the sheath of the superior oblique tendon.

However, several other theories have since been proposed, and a critical assessment of these was made in a previous article (Sandford-Smith, 1973). It was concluded that the superior oblique tendon sheath syndrome was most likely to be a type of stenosing tenosynovitis, and a few individual cases showing these changes were described.

A similar conclusion was reached by Mein (1971) from an analysis of a much larger series of 37 patients, but the evidence in this study was circumstantial, being derived from the natural history of the disease rather than from any direct observation of the clinical signs of stenosing tenosynovitis.

Stenosing tenosynovitis produces clinically demonstrable changes. There is a dilatation of the tendon where it changes direction (the trochlea), hypertrophy of the sheath, and localized tenderness. These changes should be present in patients with the tendon sheath syndrome, and in this study patients were examined specifically for these signs.

Clinical material and methods of examination

A clinical diagnosis of the superior oblique tendon sheath syndrome was made in 19 patients seen during the last 3 years at the Bristol Eye Hospital. When possible a Hess screen chart was plotted to confirm the diagnosis, and in a few cases a forced duction test under anaesthesia was also performed. Four of these 19 patients were excluded from this series, three because they had undergone previous surgical treatment to the superior oblique tendon or its sheath, and one because the patient had moved.

The examination for the signs of stenosing tenosynovitis was done by gentle palpation over the trochlea and just posterior to it while the patient was actively elevating and depressing the eye in adduction. A swelling, if present, could be felt just posterior to the trochlea in depression which moved forwards to abut against the trochlea in attempted elevation. A similar examination of the other eye made a good control.

The assessment of tenderness was sometimes more difficult, partly because of its subjective nature and partly because vigorous palpation even of a normal trochlea would cause discomfort to a young child. By simultaneously gently palpating both trochleas with the ball of the thumb and asking the child to point to one side if it was tender, consistent results on repeated examinations were obtained.

To try to eliminate any bias in these observations the patients were also examined by an independent observer when possible and positive results were only recorded if both were in agreement.

Results

<table>
<thead>
<tr>
<th>Clinical findings</th>
<th>No. of patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>*Palpable swelling with localized tenderness</td>
<td>7</td>
</tr>
<tr>
<td>*Palpable swelling but no tenderness</td>
<td>1</td>
</tr>
<tr>
<td>No palpable swelling but localized tenderness</td>
<td>4</td>
</tr>
<tr>
<td>No swelling or tenderness</td>
<td>3</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>15</strong></td>
</tr>
</tbody>
</table>

* The size of the swelling was variable. In one case it was large enough to be easily visible but usually it was about the size and shape of a melon seed.

Comment

In this series seven of 15 patients with the tendon sheath syndrome showed the typical clinical changes of stenosing tenosynovitis and a further four showed some of these changes. The tendency to spontaneous remission in the tendon sheath syndrome is now well known, and in the one case in this series in which there was a swelling without tenderness the patient remarked that it had been tender some months previously.

Stenosing tenosynovitis is a chronic inflammatory condition associated with 'wear and tear' changes in a tendon passing around a pulley. As the inflammatory changes subside some organic adhesions may persist. Thus the absence of any tenderness or swelling in three cases does not positively exclude stenosing tenosynovitis as a possible cause.
One additional interesting feature observed from this series was the threat to binocular vision from the tendon sheath syndrome especially in younger children, as six of the original 19 patients developed secondary convergent squints.

Summary

Clinical examination of 15 patients with the superior oblique tendon sheath syndrome showed that 12 of them had some evidence of stenosing tenosynovitis.

References