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Superior Oblique Tendon Sheath Syndrome (Brown Syndrome): MR Findings

Multiplanar capability and improved tissue characterization have made MR an important technique for study of the orbit. We report a case of a patient with juvenile rheumatoid arthritis and vertical diplopia in whom Brown syndrome was diagnosed clinically [1]. MR clearly showed the fibrotic nature of the affected superior oblique tendon caused by chronic inflammation. To our knowledge, this is the first report of MR findings of orbital Brown syndrome.

Case Report

The patient was a 37-year-old woman who had had juvenile rheumatoid arthritis since she was 3 years old. She had had Sjogren syndrome with dry eyes for 6 years and was being treated with artificial tears. Vertical diplopia and restriction of upward gaze of the left eye had been present for 5 years. The strabismologist considered a clinical diagnosis of left-sided Brown syndrome. MR study of the orbits was performed on a 1.5-T unit. T1-weighted, proton-density, and T2-weighted images showed a nodular left superior oblique tendon sheath of low signal intensity (Fig. 1), representing fibrosis from chronic inflammation. No strabismus surgery was planned because of the lack of fusion, the concomitant hypertropia, and the variable nature of the diplopia.

Discussion

In 1950, Brown [1] reported a stenosing tenosynovitis of the superior oblique tendon and its sheath, referred to as superior oblique tendon sheath syndrome or Brown syndrome. The clinical features include (1) vertical diplopia, (2) intermittent symptoms, (3) a clicking sensation sometimes noted with prolonged upward gaze, (4) widened palpebral fissure on adduction, (5) inability to raise the affected eye above the midpoint, and (6) a resulting backward tilt of the head in some patients [2]. Normally, as the inferior oblique muscle moves the eye upward and inward, the superior oblique muscle relaxes, and the tendon lengthens and slides smoothly in the trochlea. If the superior oblique muscle cannot relax or its tendon cannot lengthen, the eye cannot be elevated. This restriction of movement occurs most strikingly in the adducted position, mimicking inferior oblique muscle palsy [3]. Clinically, vertical diplopia occurs. In Brown syndrome, the thickened tendon cannot lengthen and creates resistance to motion. As the resistance is overcome, a clicking occurs with the sudden release of the tendon [2].

In 1974, Brown [3] divided this syndrome into congenital and acquired forms. The congenital form occurs only in those patients

who have a congenital short anterior sheath of the superior oblique muscle; this form may be bilateral, is rarely familial, and has been reported in monozygotic twins [4]. The acquired form can be a rare complication of either the adult or the juvenile form of rheumatoid arthritis [5] or may occur in association with sinusitis, orbital trauma, scleral buckles, blepharoplasty [6], superior oblique tuck procedures, and even isolated metastases in the superior oblique muscle [7].

Juvenile rheumatoid arthritis is a disease or group of diseases characterized by the presence of chronic inflammation of synovium, tendons, tendon sheaths, and bursae. Acquired Brown syndrome associated with this type of arthritis is due to inflammation of the tendon and tendon sheath in the region of the trochlea. During the acute inflammatory stage, local administration of steroids may be beneficial [5]. Various surgical procedures designed to ameliorate the signs and symptoms of Brown syndrome by weakening the tendon have been reported [8], although most patients who have the congenital form do not need surgery.

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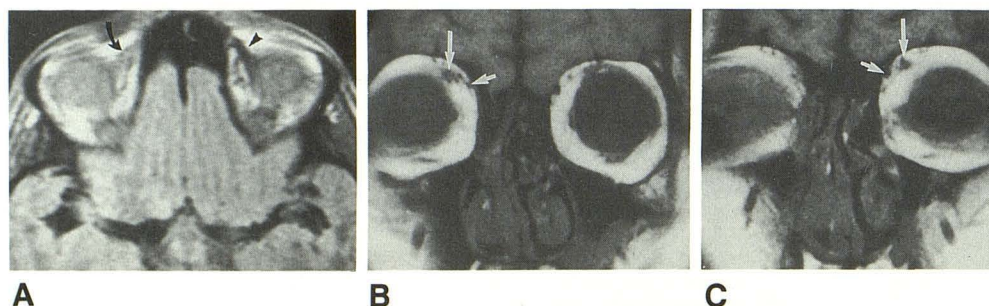


Fig. 1.—Brown syndrome.

A, Axial mixed-weighted MR image, 2000/30/2, of upper orbits shows slightly nodular left superior oblique tendon sheath (arrowhead) of low signal intensity compared with normal right tendon (arrow), which is relatively isointense to brain.

B and C, Because of patient's obliquity in imager, composite T1-weighted MR images, 600/20/2, were used to show both superior oblique tendons. B is 5 mm posterior to C. In B, right trochlea (short arrow) and superior oblique tendon (long arrow) are isointense to brain. In C, diseased left superior oblique tendon (long arrow) and trochlea (short arrow) are of low signal intensity.