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## MRI of acquired Brown syndrome: a report of two cases

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### ARTICLE INFO

#### Article history:

Received 24 July 2017  
Received in revised form 19 September 2017  
Accepted 25 September 2017  
Available online

#### Keywords:

Acquired Brown syndrome  
Magnetic resonance imaging  
Superior oblique tendon  
Strabismus

### ABSTRACT

Brown syndrome is characterized by upward gaze impairment while the eye is in adduction. It is caused by abnormalities involving the superior oblique tendon-trochlea complex. Imaging can help confirm the diagnosis, shed light on its etiology, and determine the best course of treatment. However, reports of magnetic resonance imaging findings of acquired Brown syndrome are scarce in the literature. Here, we describe magnetic resonance imaging features of 2 cases of acquired Brown syndrome.

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## Introduction

Brown syndrome was first described by Harold Whaley Brown in 1950 as impairment of upward gaze above the horizontal level while the eye is in adduction [1]. The condition is caused by pathologies involving the superior oblique (SO) tendon-trochlea complex that restricts the passage and movement of SO through the trochlea (Fig. 1; illustration of the extraocular muscle anatomy). Aside from congenital etiologies, the condition has numerous acquired causes, including trauma, sinus infection, inflammatory diseases e.g. (rheumatoid arthritis and systemic lupus erythematosus), and postoperative complications [2–8].

Computerized tomography (CT) has been used to evaluate SO tendon-trochlea complex abnormalities for Brown syndrome [9,10]. Although CT imaging can be valuable for the diagnosis of Brown syndrome, it has largely been replaced by

magnetic resonance imaging (MRI) because of the superior soft tissue resolution offered by the technique [11,12]. For this reason, MRI is superior to CT for most ophthalmologic conditions, especially pathologies that involve extraocular muscles [13–18].

Here, we describe the MRI features of 2 cases of acquired Brown syndrome, which have not been well reported in the literature.

## Case report

### Case 1

A 44-year-old female with past medical history of rheumatoid arthritis presented with a 2-week history of left retro-orbital pain and temporal headaches. She also developed transient diplopia with rightward or downward gaze. The left

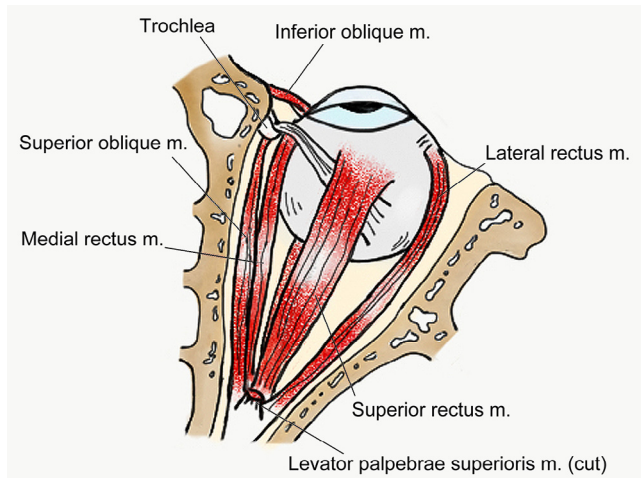
Competing Interests: All Authors report no conflict of interest.

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<https://doi.org/10.1016/j.radcr.2017.09.025>

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**Fig. 1 – Illustration of the extraocular muscle anatomy. Orientation: right eye, axial view, looking down from above.**

eye remained temporarily disconjugate with these gazes. Upon examination, there seemed to be a physical “catch” with adduction of the left eye. There were no associated visual field deficits, vertigo, facial weakness, or paresthesia.

A review of laboratory values showed positive antinuclear antibody, elevated sedimentation rate, and C-reactive protein levels. Rheumatoid factor was less than 10, and complete blood count, renal function panel, and liver function panel were all within normal limits. The patient’s long history of rheumatoid arthritis affected her knees and hands bilaterally, but she was not on any immunosuppression medication.

Based on the clinical features, the patient was sent for MRI evaluation. Within the left orbit, there was diffuse asymmetric thickening and enhancement of the left superior oblique muscle, best seen on coronal Short tau inversion recovery (STIR) and postcontrast fat-saturated T1 images (Fig. 2A, B). The left SO muscle appeared enlarged in the craniocaudal dimension on axial postcontrast fat-saturated T1 images (Fig. 2C). The enlarged left SO muscle mildly abutted the medial rectus muscle, which otherwise appeared unremarkable. There was also minimal fat stranding within the adjacent left intraconal space.

The diagnosis of Brown syndrome was made based on clinical presentation and MRI findings. The patient is to be followed-up in the clinic in a month.

#### Case 2

A 35-year-old female presented with a 2-year history of diplopia. She complained of wandering left eye since January 2014 and compensated her diplopia with a mild backward head tilt. On examination, she had upward gaze impairment of the left eye, especially on adduction. Optic nerve, macula, and peripheral retina all appeared normal on fundoscopic examination.

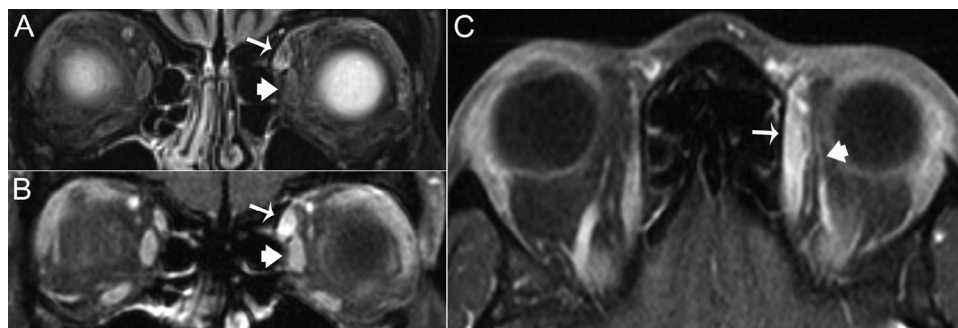
Laboratory results were unrevealing as rheumatoid factor, erythrocyte sedimentation rate, complete blood count, renal functional panel, and antinuclear antibody were all normal. The patient had no history of ophthalmologic surgery or other pertinent medical history.

The patient was subsequently sent for imaging. Coronal and axial postcontrast fat-saturated T1 images demonstrated mild asymmetric thickening and enhancement in the expected region of the left SO tendon sheath better than STIR images (Fig. 3). Specifically, there was prominent enhancement of the SO tendon-trochlea complex on postcontrast fat-saturated T1 images (Fig. 3). Findings were indicative of a nonspecific inflammatory process of the SO tendon-trochlea complex.

After the diagnosis of Brown syndrome was made, the patient was treated with a month-long course of 50 mg ibuprofen, 3 times a day. Her diplopia had since improved significantly and no surgical intervention was required.

#### Discussion

When the condition was first described as “SO tendon sheath syndrome” by Dr. Harold W. Brown in 1950, he postulated that congenitally short anterior SO tendon sheath and inferior oblique palsy were the cause of the condition [1]. However, anatomic and clinical examinations revealed that the described anterior sheath did not exist, and the condition was inconsistent with inferior oblique palsy [19]. Further studies showed that the condition was the result of SO tendon-trochlea complex abnormalities that can be attributed to a number of causes [20,21].



**Fig. 2 – Patient 1. Coronal STIR (A) and postcontrast fat-saturated T1 (B) images; axial postcontrast fat-saturated T1 (C). There is diffuse asymmetric thickening and enhancement of the left superior oblique muscle (arrows). The enlarged left-sided superior oblique muscle mildly abutts the medial rectus muscle, which otherwise is unremarkable (arrow head).**

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