Title

Clinical and imaging features of congenital and acquired isolated inferior rectus muscle hypofunction.

Journal

Journal of American Association for Pediatric Ophthalmology and Strabismus, 25(1)

Authors

Solanes, Federica
Demer, Joseph

Publication Date

2021-02-01

DOI

10.1016/j.jaapos.2020.09.007

Peer reviewed

HHS Public Access

Author manuscript

J AAPOS. Author manuscript; available in PMC 2022 February 16.

Published in final edited form as:

J AAPOS. 2021 February ; 25(1): 11.e1–11.e9. doi:10.1016/j.jaapos.2020.09.007.

Clinical and imaging features of congenital and acquired isolated inferior rectus muscle hypofunction

Federica Solanes, MD[a,b,c], **Joseph L. Demer, MD, PhD**[a,b,d,e]

[a]Department of Ophthalmology, David Geffen Medical School at the University of California, Los Angeles;

[b]Stein Eye Institute, David Geffen Medical School at the University of California, Los Angeles;

[c]Pontificia Universidad Católica de Chile, Santiago, Chile;

[d]Bioengineering Department, David Geffen Medical School at the University of California, Los Angeles

[e]Department of Neurology, David Geffen Medical School at the University of California, Los Angeles

Abstract

Background—Inferior rectus (IR) underaction may arise from various causes that are distinguishable through imaging. We investigated clinical and imaging characteristics of congenital and acquired causes of IR underaction.

Methods—Cases of IR underaction were selected from data prospectively collected in a study of orbital imaging in strabismic patients.

Results—Review identified 3 cases of congenital IR underaction (2 with bilateral IR aplasia and 1 with unilateral IR hypoplasia), 12 acquired cases, including 4 due to denervation (2 idiopathic, 1 after multiple strabismus surgeries, 1 after head trauma), and 8 cases of direct IR damage (5 with orbital trauma and 3 with previous surgery, including 2 sinus surgery and 1 laser blepharoplasty). Of the 23 cases, 11 adults had high-resolution magnetic resonance imaging, and 2 children had computed tomography. Imaging identified the anatomic diagnosis in congenital cases; in acquired cases, imaging helped to identify atrophy and exclude alternative orbital causes; and in direct mechanical damage, imaging clarified the mechanism of underaction, extent of IR damaged, and the degree of retained contractility. Patients with congenital IR absence or hypoplasia exhibited A pattern exotropia that was typically absent in isolated acquired denervation or direct IR damage.

Conclusions—Orbital imaging demonstrates a variety of abnormalities in patients with congenital or acquired IR hypofunction, helping to clarify the underlying mechanism and guide management.

Correspondence: Joseph L. Demer, MD, PhD, Stein Eye Institute, 100 Stein Plaza, UCLA, Los Angeles, California, 90095-7002 (jld@jsei.ucla.edu).

Isolated inferior rectus (IR) underaction can be caused by disorders affecting the oculomotor nucleus, nerve, myoneural junction, or the muscle itself.[1,2] The most frequent cause is accidental or surgical trauma.[3,4] Congenital abnormalities of extraocular muscles include supernumerary muscles, anomalous bands, and muscle hypoplasia or aplasia.[5] Although clinical examination can suggest IR hypofunction, findings are so highly variable[2,6,7] that they may not define the pathophysiology accurately. Force generation testing can demonstrate absence of IR rotational force on the eye but not the reason for the deficit, and the test is not feasible in many clinical situations.

Magnetic resonance imaging (MRI) can directly demonstrate extraocular muscle locations, sizes, contractility, and innervation, as well as the presence of supernumerary bands. Khitri and Demer[5] demonstrated the utility of high-resolution MRI in detecting hypoplasia and misdirection of cranial nerves as well as neurogenic extraocular muscle atrophy in strabismus caused by cranial nerve palsies. Moreover, MRI can define muscle function to inform treatment.[8] A hypoplastic or atrophic IR exhibits a reduced cross section that fails to increase in attempted infraduction. Although an IR paralyzed by contusion also lacks contractile increase in cross section, the acutely paralyzed IR usually exhibits irregularly increased cross section due to intramuscular edema. This contrasts with a transected IR, which exhibits posterior shift in maximum cross section that further increases in attempted infraduction.[9]

The purpose of the current study was to compare the clinical, imaging, and intraoperative presentation of acquired and congenital IR hypofunction to identify distinguishing features of each. We also illustrate an innovative surgical technique helpful to enhance infraduction and treat incyclotorsion in cases of severe IR underaction.

Subjects and Methods

We analyzed data prospectively collected by an ongoing study of strabismus approved continuously since 1990 by the Institutional Review Board of the University of California, Los Angeles, and compliant with requirements of the US Health Insurance Portability and Accountability Act of 1996 and the tenets of the Declaration of Helsinki. We selected for analysis all adequately imaged cases categorized as representing IR absence and IR palsy, defined to be deficient infraduction in abduction, and hypertropia maximal in depression and abduction. Results reported are cumulative over the study. Subjects reported previously are briefly described with references.

Subjects underwent complete ophthalmic evaluation, including visual acuity assessment, stereopsis, slit-lamp examination, ophthalmoscopy, and refraction. Binocular alignment was measured by placing prism before the paretic eye during alternate cover testing for a distant target in cardinal gazes, and for a central target at near, including during head tilt as indicated. Ductions were quantified by a 9-point scale, with 0 indicating normal, −4 indicating inability to pass midline, and +4 indicating maximal over-rotation. Torsion was measured using double Maddox rods.

After written informed consent, MRI was performed with an array containing two surface coils for each orbit (Medical Advances, Milwaukee, WI), using a 1.5-T General Electric Signa scanner (Milwaukee, WI). High-resolution T1 or T2 fast spin-echo MRI was performed using a fiberoptic fixation target, as described elsewhere.[10–12] Two children underwent x-ray computed tomography (CT) of the orbits for clinical diagnosis and planning; these cases were included by retrospective review.

Muscle contractility was determined by the change in maximal cross-sectional area from relaxation to contraction, for example, for the IR from supraduction to infraduction with fixation controlled by the normal eye to ensure infraduction effort. This measure demonstrates normal contractility comparably to the alternative posterior partial volume, but it is better interpreted when the IR is abnormal.[13] A normally contracting IR exhibits a conspicuously larger cross section in attempted downward gaze than in upward gaze, whereas a weak or paralyzed IR does not. This increase requires neither scleral IR attachment nor oculorotary force on the globe, so this technique can identify contractility of an IR that appears clinically inactive.

Results

A total of 15 subjects (9 female) were included (eSupplement 1, available at jaapos.org). According to imaging findings, three mechanisms of inferior rectus underaction were identified: (1) congenital underdevelopment (cases 1–3), (2) acquired denervation (cases 4–7), (3) direct mechanical damage (cases 8–15).

Case 1

A 52-year-old woman presented with progressive congenital strabismus. She had lowered her chin lifelong because she was otherwise unable to look downward. There was 35^{Δ} alternating A-pattern exotropia in central gaze, decreasing to 18^{Δ} in sursumversion and increasing to $>100^{\Delta}$ in attempted infraversion. There was 25^{Δ} left hypertropia in levoversion, reversing to 5^{Δ} right hypertropia in dextroversion. She had −5 infraduction limitation bilaterally, more in abduction (Figure 1). Orbital MRI showed bilateral IR absence, superior rectus (SR) and levator palpebrae superioris hypoplasia, incyclorotation of rectus pulley array, bilateral hypoplasia of infraorbital motor nerves that were too small to visualize using careful technique, and a band from the right SR to the inferior oblique muscle (IO). See Figure 2.

Surgical exploration confirmed bilateral absence of IR muscles, tendons, and ciliary vessels (Figure 3A). The left superior rectus (SR) tendon was fibrotic. The superior oblique (SO) tendons were attenuated and adherent to the SR tendons (Figure 3B). Surgical repair consisted of bilateral IO transposition to the site of the normal IR insertion, and lateral rectus (LR) recession with inferior transposition of one tendon width. Also bilaterally, suture was placed in the sclera posterior and lateral to the posterior SO insertion and then through the anterior portion of the SO tendon at the insertion. The anterior three-quarters of the SO tendon were detached (Figure 3C–E) and the suture tied to rotate the anterior SO tendon posteriorly (Figure 3D–F). The left SR tendon was recessed 3 mm. After surgery the ocular

torticollis improved, and the infraduction in adduction was improved to −3 bilaterally, with 12^{Δ} residual exotropia in central gaze. The A pattern collapsed.

Case 2

The 38-year-old mother of case 3 had undergone childhood strabismus surgery. She had 15° left head turn. In central gaze there was 17^{Δ} right A-pattern exotropia that increased to 25^{Δ} in levoversion and disappeared in dextroversion, with 12^{Δ} right hypotropia that increased in sursumversion but decreased in infraversion. There was −4 supraduction and −2 infraduction in the right eye. MRI demonstrated a hypoplastic right IR (Figure 5), and an abnormal band connecting the medial rectus to the LR muscle.[5]

Case 3

This 8-year-old daughter of case 2 had congenital left exotropia and hypertropia, and chin-down position. There was 40^{Δ} A-pattern exotropia and 15^{Δ} left hypertropia in central gaze that increased in infra- and levoversion but diminished in sursum- and dextroversion. Left hypertropia increased in levoversion and in right tilt and decreased in dextroversion and with left tilt. Both eyes had limited infraduction in abduction, −4 in the right and −2 in the left eye. CT imaging revealed bilateral IR absence with SR hypertrophy (Figure 4).

Case 4

This 35-year-old woman had diplopia and chin-down torticollis for 18 months. She had 7^{Δ} left hypertropia that increased in infraversion, levoversion, and contralateral head tilt but decreased in dextroversion and was absent in sursumversion and ipsilateral tilt. MRI showed left IR atrophy and diminished contractility in infraduction (Figure 6). Recession of the left SR, resection of the left IR and posterior fixation of the right IR relieved diplopia.

Case 5

This 40-year-old man had progressive left hypertropia for the past 20 years. He demonstrated 30^{Δ} left hypertropia that decreased in sursumversion and increased in infraversion, limitation of infraduction in the left eye more in abduction (−4) than in adduction (−3), and normal vertical saccades. Orbital MRI showed left IR atrophy without contractile changes in attempted infraduction (Figure 7).

Case 6

This 62-year-old woman had recurrent diplopia despite multiple strabismus surgeries for presumed thyroid ophthalmopathy. There was 16^{Δ} esotropia in central gaze, 4^{Δ} left hypertropia in infraversion and −2 limitation of left eye infraduction, but normal saccades. MRI revealed a small left IR with subnormal contractility in infraduction.

Case 7

This 7-year-old girl presented 3 years after injury in a traffic accident. There was 20° right head tilt and a mydriatic, unreactive left pupil. She had 50^{Δ} left exotropia and 30^{Δ} left hypertropia in central gaze, and markedly limited left eye infraduction more in abduction (−4) than in adduction (−2), mildly limited left eye adduction, and normal supraduction.

Fundus examination showed right excyclotorsion. CT revealed left IR atrophy and right SO atrophy.[9]

Case 8

This 46-year-old woman had suffered left orbital trauma requiring globe rupture repair and placement of an orbital floor implant. There was 50^{Δ} left hypertropia and 60^{Δ} exotropia, with −4 left eye infraduction but normal saccades. MRI showed general commotion to the left extraocular muscles and an inferomedial fluid collection around the orbital implant. The anterior half of the left IR was not visible adjacent the orbital implant. The posterior half of the IR had retained bulk, with contractility on attempted infraduction (Figure 8). At orbitotomy, the distal end of the IR muscle was identified before drainage of the fluid collection and implant removal. Through a conjunctival incision, the IR tendon and ciliary vessels were identified and dissected to Lockwood's ligament. The distal IR was then pulled anterior through its pulley and sutured to the proximal end to restore muscle continuity.

Case 9

This 37-year-old women had endoscopic sinus surgery complicated by entry into the right orbit. She could fuse with 10° right head tilt. There was a 5° right hypertropia in primary position that reversed to 8^{Δ} left hypertropia in infraversion and 8^{Δ} esotropia in dextroversion that reversed to 12^{Δ} in levoversion. She had −4 supra- and infraduction, −2 abduction, and −3 adduction. MRI revealed a defect in the inferomedial wall of the right orbit and disrupted connective tissue near the MR and IR pulleys. The anterior quarter of the MR and third of the IR exhibited a T2 signal similar to orbital fat, with normal muscle signal more posteriorly but intact innervation (Figure 9).

Case 10

This 35-year-old man had diplopia after endoscopic surgery sinus complicated by entry into the left orbit. There was 12^{Δ} left esotropia and 15^{Δ} left hypotropia, with severe limitation to supraduction, abduction, adduction, and infraduction but normal saccades. MRI revealed an anterior defect in the inferomedial wall of the left orbit and extensive fibrosis, including the left MR, the IR, and the IO. The IR was inserted to the sclera just posterior to the equator with a defect passing diagonally through the muscle belly, but retained contractility. Nerves to the damaged muscles were normal.

Case 11

This 50-year-old woman experienced diplopia following bilateral laser transconjunctival blepharoplasty. She could fuse only by lowering her chin and turning to the left. She had 30^{Δ} left hypertropia that increased in infraversion, levoversion, and contralateral head tilt, and diminished in sursumversion, dextroversion, and ipsilateral head tilt. There was marked limitation to left eye infraduction, greatest in abduction. MRI revealed transection of the left IR with retained distal contractility. Left anterior orbitotomy with IR recovery and surgical reattachment and SR recession corrected the misalignment.[14]

Case 12

This 15-year-old boy had diplopia 1 month after repair of a right orbital fracture. There was 12^{Δ} right hypertropia that decreased in sursumversion and became very large in infraversion. There was −2 elevation in adduction and −4 infraduction, with slowed infraducting saccades in the right eye. Forced duction testing demonstrated mild limitation to full infraduction of the right eye; force generation testing showed marked weakness of the right IR. MRI revealed enlargement of the right IR to 4–5 times normal size, surrounded by a large highly vascularized mass, in normal continuity with the scleral insertion and displaced from the repaired fracture. The IR motor nerve was intact (Figure 10). Strabismus surgery was not recommended, and the strabismus, motility, and diplopia spontaneously improved.

Case 13

This 16-year-old boy had diplopia 6 months after surgical repair of a left orbital floor fracture. In central gaze there was 15^{Δ} esotropia and 25^{Δ} left hypertropia that increased in levo- and infraversion. He had marked limitation to infraduction greatest in abduction, but normal vertical saccades. MRI showed a left orbital floor implant, scarring in the inferior fornix, and a nearly completely disinserted left IR with normal distal contractility. The left IR was reattached with improvement in aligment.[8]

Case 14

This 49-year-old man presented 1 year after trauma repaired with a right orbital floor implant. He had 15^{Δ} exotropia and 60^{Δ} right hypertropia in central gaze, that increased in infraversion and decreased in sursumversion. There was markedly limited right eye infraduction with a slowed downward saccade. MRI revealed severe damage to the IR muscle. Horizontal rectus inferior transposition with posterior fixation and injections of botulinum toxin to the SR improved alignment.

Case 15

This 43-year-old woman had undergone multiple surgeries for severe head, facial, and left orbit trauma. There was 14^{Δ} esotropia and 10^{Δ} left hypotropia. There was −3 supraduction and −4 infraduction with −1 limitation to abduction in the left eye, with normal horizontal saccades. MRI demonstrated fracture of the medial and inferior walls, deep transection of the left IR, with bridging by a strand with the proximal stump, adherent to an orbital floor implant. There was deep damage to the left MR, SR, LR, and IO muscles that exhibited no contractility.

Summary of Mechanisms

Patients with congenital absence or hypoplasia of the IR exhibited A-pattern exotropia (cases 1–3). Additionally, 2 patients (cases 1 and 3) had abnormal orbital bands and 1 (case 1) had incyclotorsion of the pulley array. Except for case 7, who had oculomotor palsy, acquired IR denervation was associated with exotropia or slowed saccades as well as hypertropia that increased in the IR field of action. IR direct damage was associated with orbital fracture in 7 of 8 patients and with inadvertent surgical breach in case 11. Several patients with chronic IR trauma showed normal infraducting saccades.

Management

Based on orbital imaging, observation versus surgical treatment was selected. Lacerated, ruptured, or detached but functional IR muscles were recovered and reanastamosed (cases 8, 11, 13). One case of IR contusion recovered spontaneously (case 12). Patients with small-angle strabismus despite extensive IR damage (case 9 and 15) were managed with prisms. When multipositional MRI demonstrated residual IR contraction, IR reinforcement with SR weakening improved alignment (case 4).

Discussion

Clinical manifestations of congenital IR underaction may vary according to specific causes, combined with associated abnormalities, such as those of rectus pulleys. In this case series, congenital IR underdevelopment always presented with exotropia. Acquired IR denervation is less often associated with exotropia unless there is another lesion, such as oculomotor palsy or orbital trauma damaging other muscles or nerves. Trauma to the IR was often associated with orbital fracture or inadvertent surgical breech. Vertical saccadic examination was not reliably helpful in diagnosing IR underaction.

Surgical treatment of IR underaction includes options to address the functional IR deficit and associated horizontal, pattern, and torsional deviations. In trauma, multipositional MRI localized and characterized damage to the IR and connective tissue and demonstrated retained IR contractility, thus informing a decision between observation versus anatomically guided, prompt surgical intervention.

The functional deficit in IR palsy, permanent IR paralysis, and congenital IR agenesis can be remedied by horizontal rectus inferior transposition and/or inferior oblique anterior transposition (IOAT), with or without ipsilateral SR weakening. IOAT has an infraducting and antielevation effect, spares anterior segment circulation, and reserves horizontal rectus surgery to address horizontal strabismus, patterns or torsion.[15,16–18] In inferior transposition of the horizontal rectus muscles, infraducting force is created and horizontal duction is minimally decreased.[19] Because most cases of IR trauma interrupt its anterior ciliary arteries, plication of the horizontal rectus muscles with transposition of up the amount of plication may be preferred to resection.[20] The posterior SO folding technique described for case 1 aimed to enhance the infraducting effect of the SO and relieve the incyclotropia by moving the anterior torsional fibers posterior to the equator to change its effect from incycloduction to infraduction. Transposition of the SO also avoids risk of anterior segment ischemia.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

References

1. Chou TM, Demer JL. Isolated inferior rectus palsy caused by a metastasis to the oculomotor nucleus. Am J Ophthalmol 1998;126:737–40. [PubMed: 9822247]

2. Akbari MR, Ameri A, Keshtkar Jaafari A, Fard MA, Eshraghi B, Mirmohammadsadeghi A. Clinical features and surgical outcomes of isolated inferior rectus muscle paralysis. Strabismus 2014;22:58–63. [PubMed: 24738952]

3. Awadein A Clinical findings, orbital imaging, and intraoperative findings in patients with isolated inferior rectus muscle paresis or underaction. J AAPOS 2012;16:345–9. [PubMed: 22824489]

4. Young SM, Koh YT, Chan EW, Amrith S. Incidence and risk factors of inferior rectus muscle palsy in pediatric orbital blowout fractures. Craniomaxillofac Trauma Reconstr 2018;11:28–34. [PubMed: 29387301]

5. Khitri MR, Demer JL. Magnetic resonance imaging of tissues compatible with supernumerary extraocular muscles. Am J Ophthalmol 2010;150:925–31. [PubMed: 20801423]

6. von Noorden GK, Hansell R. Clinical characteristics and treatment of isolated inferior rectus paralysis. Ophthalmology. 1991;98:253–7. [PubMed: 2008286]

7. Astle WF, Hill VE, Ells AL, Chi NT, Martinovic E. Congenital absence of the inferior rectus muscle—diagnosis and management. J AAPOS 2003;7:339–44. [PubMed: 14566316]

8. Yip CC, Jain A, McCann JD, Demer JL. Inferior rectus muscle transection: a cause of diplopia after non-penetrating orbital trauma. Graefes Arch Clin Exp Ophthalmol 2006;244:1698–1700. [PubMed: 16555047]

9. Demer JL, Clark RA, Kono R, Wright W, Velez F, Rosenbaum AL. A 12-year, prospective study of extraocular muscle imaging in complex strabismus. J AAPOS 2002;6:337–47. [PubMed: 12506273]

10. Demer JL, Clark RA. Magnetic resonance imaging of human extraocular muscles during static ocular counter-rolling. J Neurophysiol 2005;94:3292–302. [PubMed: 16033934]

11. Demer JL, Miller JM. Magnetic resonance imaging of the functional anatomy of the superior oblique muscle. Invest Ophthalmol Vis Sci 1995;36:906–13. [PubMed: 7706039]

12. Demer JL, Dushyanth A. T2-weighted fast spin-echo magnetic resonance imaging of extraocular muscles. J AAPOS 2011;15:17–23. [PubMed: 21397801]

13. Clark RA, Demer JL. Functional morphometry of horizontal rectus extraocular muscles during horizontal ocular duction. Invest Ophthalmol Vis Sci 2012;53:7375–9. [PubMed: 22997285]

14. Pineles SL, Laursen J, Goldberg RA, Demer JL, Velez FG. Function of transected or avulsed rectus muscles following recovery using an anterior orbitotomy approach. J AAPOS 2012;16:336–41. [PubMed: 22835914]

15. Gamio S, Tartara A, Zelter M. Recession and anterior transposition of the inferior oblique muscle [RATIO] to treat three cases of absent inferior rectus muscle. Binocul Vis Strabismus Q 2002;17:287–95. [PubMed: 12528659]

16. Almahmoudi F, Khan AO. Inferior oblique anterior transposition for the unilateral hypertropia associated with bilateral inferior rectus muscle aplasia. J AAPOS 2014;18:301–3. [PubMed: 24767828]

17. Nishikawa N, Ito H, Kawaguchi Y, Sato M, Yoshida A. Resection and anterior transposition of the inferior oblique muscle for treatment of inferior rectus muscle hypoplasia with esotropia. Am J Ophthalmol Case Rep 2017;7:70–73. [PubMed: 29260082]

18. Fan YY, Yang ML. Managing hypoplasia of the inferior rectus muscle by inferior oblique anterior transposition in children. J Pediatr Ophthalmol Strabismus 2017;54:e50–e53. [PubMed: 28837740]

19. Kushner BJ. Torsion and pattern strabismus: potential conflicts in treatment. JAMA Ophthalmol 2013;131:190–93. [PubMed: 23411884]

20. Pineles SL, Chang MY, Oltra EL, et al. Anterior segment ischemia: etiology, assessment, and management. Eye (Lond) 2018;32:173–8. [PubMed: 29148529]

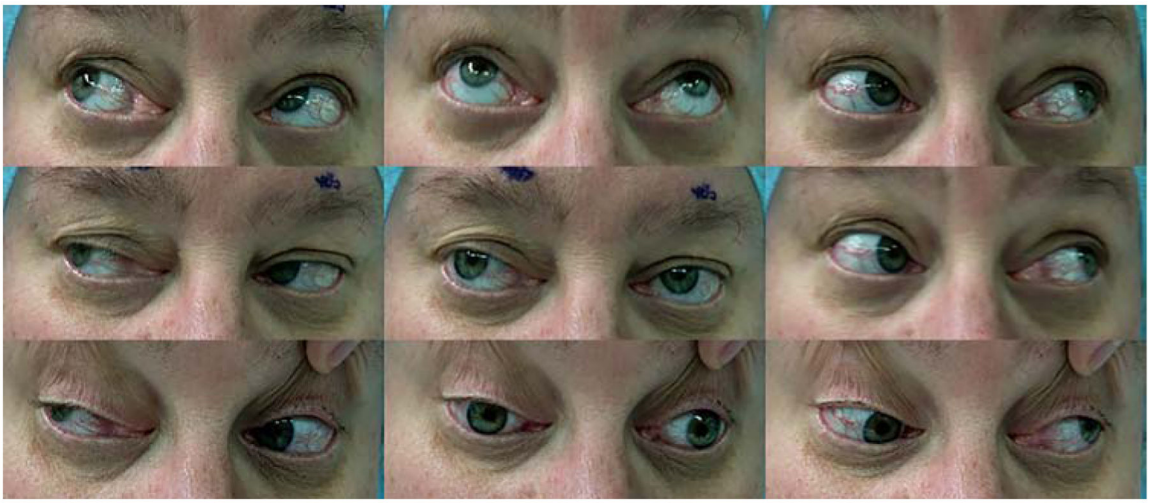

FIG 1.

Versions of case 1 with bilateral congenital absence of the inferior rectus muscle (IR), demonstrating limited infraduction bilaterally, especially in abduction, with A pattern exotropia reaching an estimated 100^{Δ} with maximal infraducting effort not fully captured here.

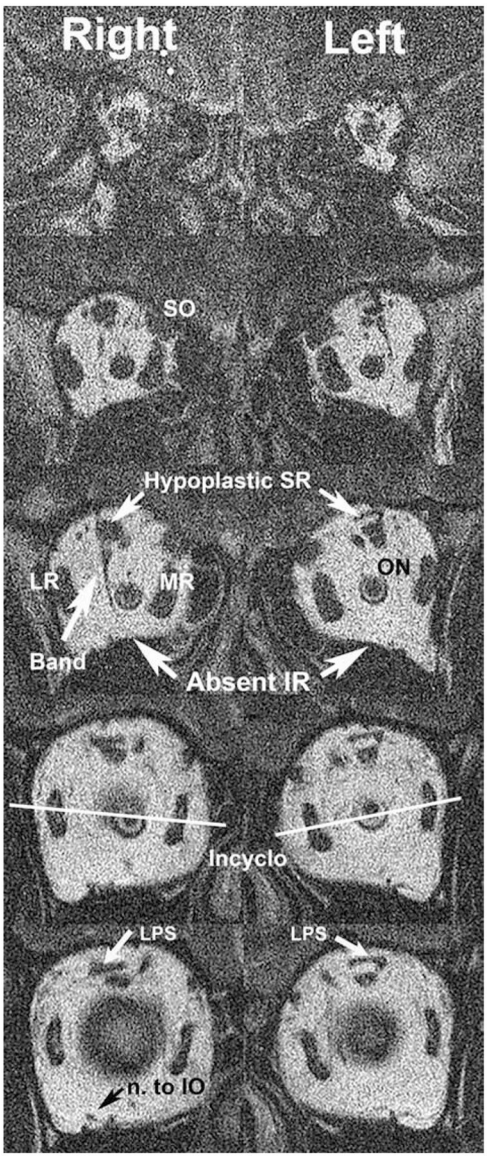

FIG 2.

Case 1. Quasi-coronal T2 MRI of the right (left column) and left (right column) orbits showing bilateral absence of the inferior rectus (IR), narrowing and hypoplasia of the superior rectus (SR), incyclorotation of rectus pulley array in the right more than in the left orbit, and an abnormal band from the SR to the inferior oblique (IO) in the right orbit. *IR*, inferior rectus; *LPS*, levator palpebrae superioris muscle; *LR*, lateral rectus; *MR*, medial rectus; *N to IO*, nerve to inferior oblique; *ON*, optic nerve; *SO*, superior oblique.

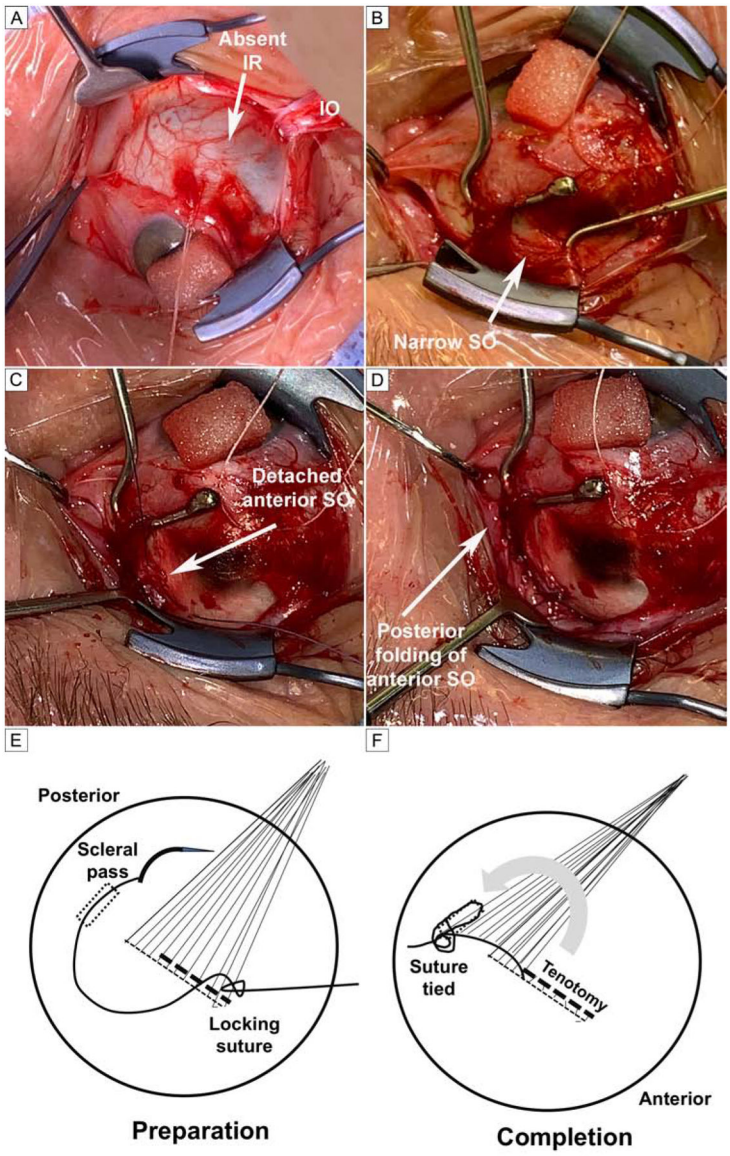

FIG 3.

Surgical exposure of right orbit of case 1. A, Inferior sclera shows absence of IR muscle, tendon, and ciliary vessels. B, Abnormally narrowed SO tendon. C, A 6–0 polyglactin 910 suture was placed just posterior and lateral to the terminal portion of the SO insertion, and then passed through the anterior portion of the SO tendon at the insertion. The anterior three-quarters of the SO tendon were then divided posterior to the suture and detached. D, The suture was then tied to fold and rotate the anterior portion of the SO tendon posteriorly to the scleral suture site to enhance infraduction action. E, Diagram of step C, suturing the anterior tendon margin and posterior scleral pass before tenotomy. F, Diagram of step D: posterior folding of the anterior SO tendon. *SR*, superior rectus.

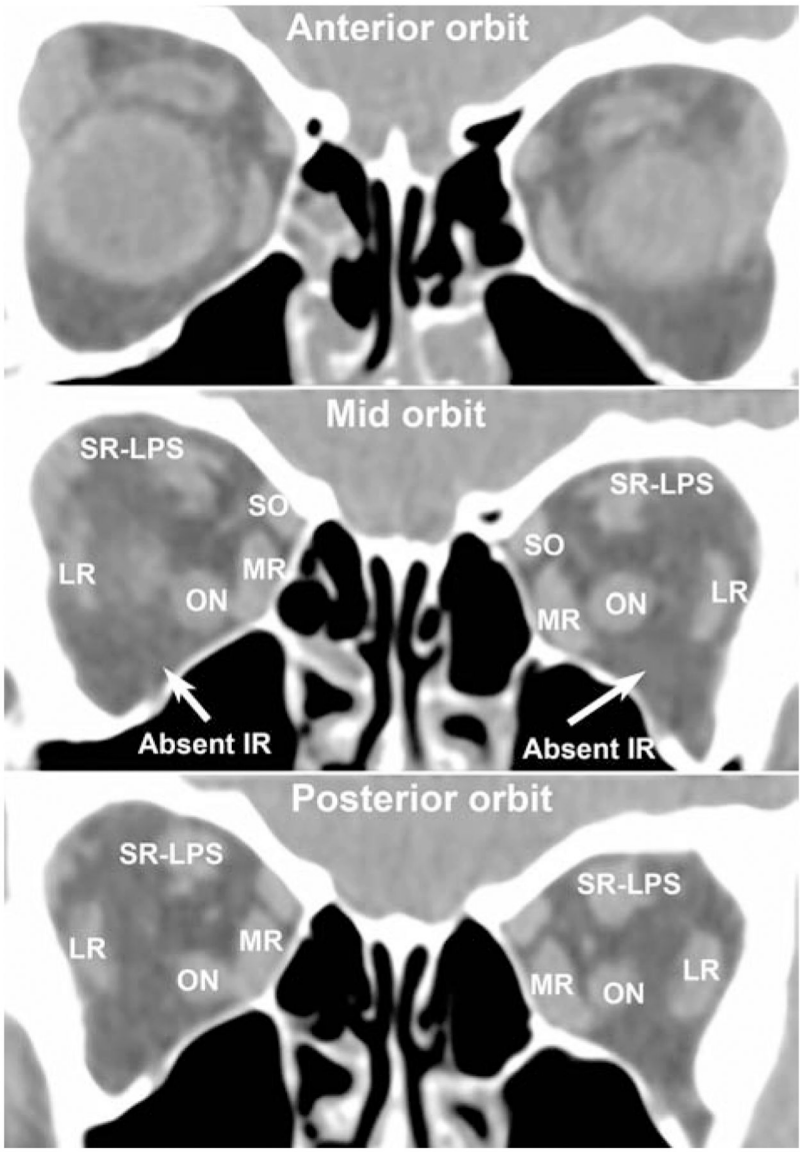

FIG 4.

Case 2. Quasi-coronal T2 MRI of the orbits showing moderately hypoplastic right inferior rectus muscle. *SR-LPS*, superior rectus–levator palpebrae superioris complex.

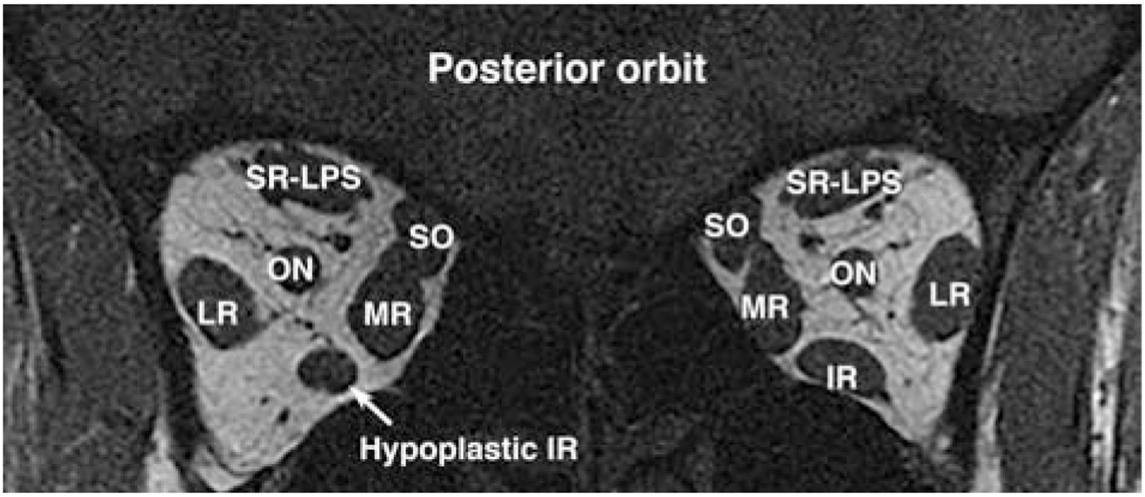

FIG 5.

Case 3. One mm thickness coronal CT scan demonstrate bilateral absence of the inferior rectus (IR).

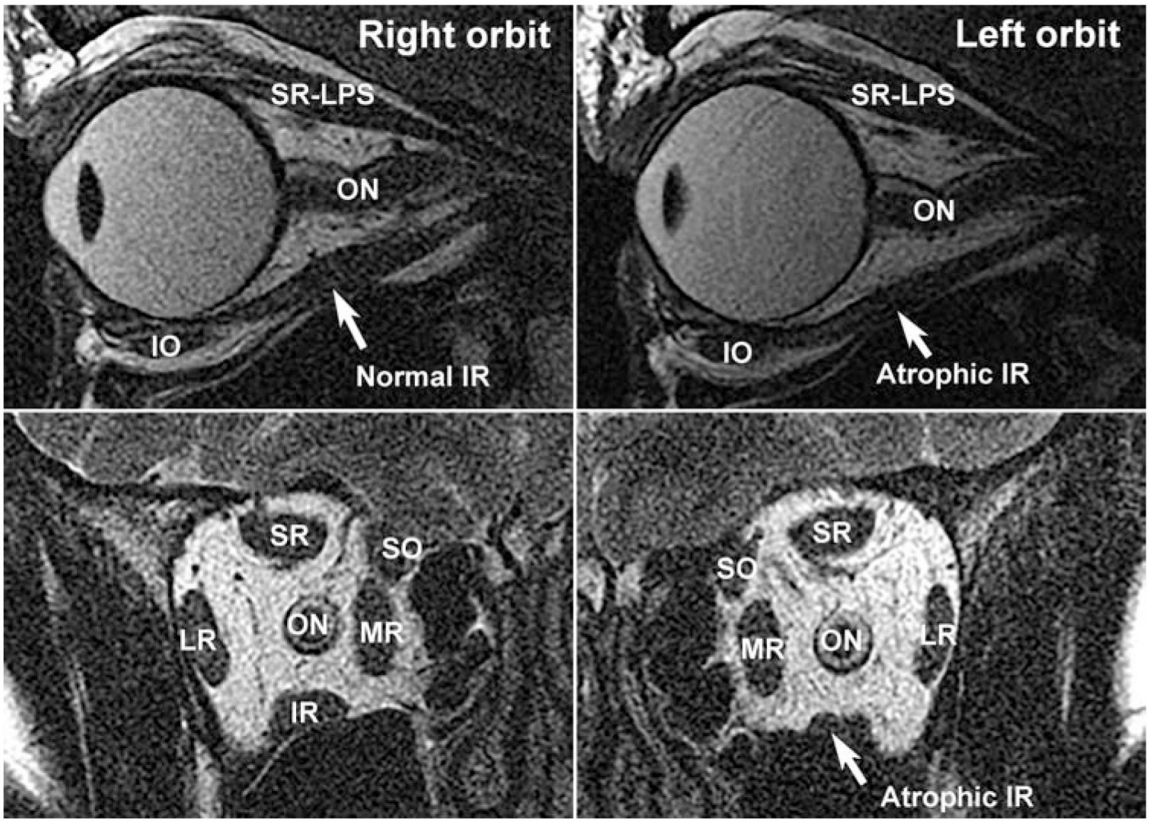

FIG 6.

Case 4. Sagittal (top row) and coronal (bottom row) MRI of right (left column) and left (right column) orbits in central gaze, demonstrating atrophic left IR with inferior bowing.

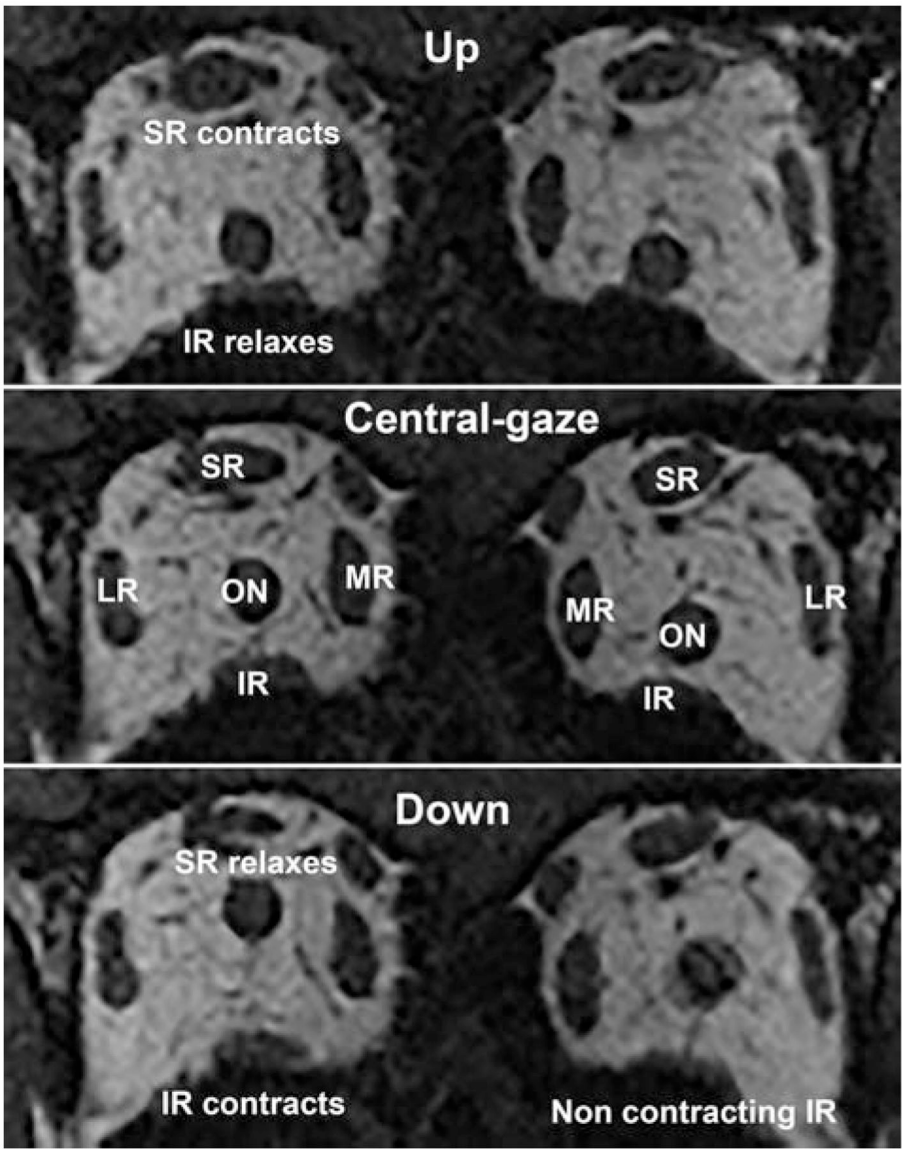

FIG 7.

Case 5. Quasi-coronal T2 MRI of right (left column) and left (right column) orbits of case 1 in supraduction (top row), central gaze (middle row), and infraduction (bottom row) showing atrophy of the left IR that lacks contractile changes with attempted infraduction.

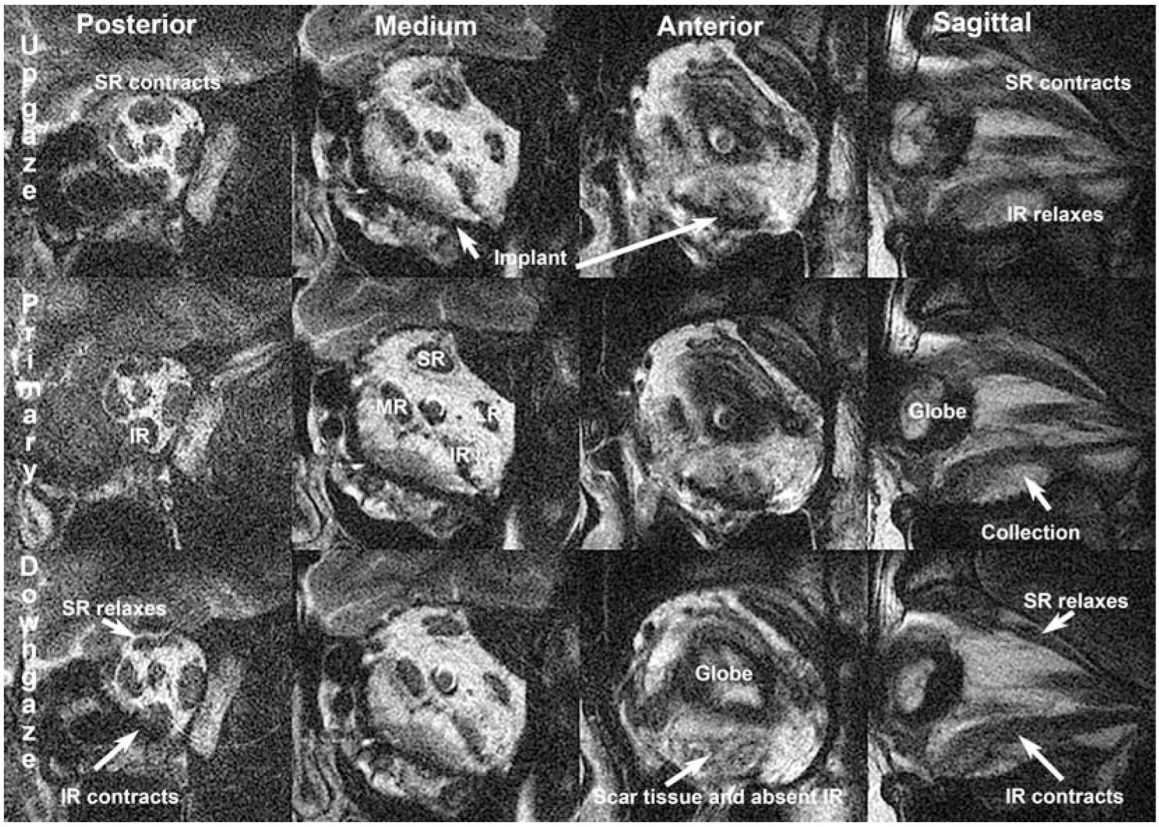

FIG 8.

Case 8. Quasi-coronal of posterior, medium and anterior (left and middles columns) and sagittal (right column) T2 MRI of the left orbit in up (top row), primary (central row), and down gaze (bottom row) showing disorganization of the left intraocular content with total retinal detachment, general commotion to the extraocular muscles and an inferomedial fluid collection near the orbital implant. The anterior half of the left IR was not clearly visible. The posterior half of the IR has retained bulk with retained contractility.

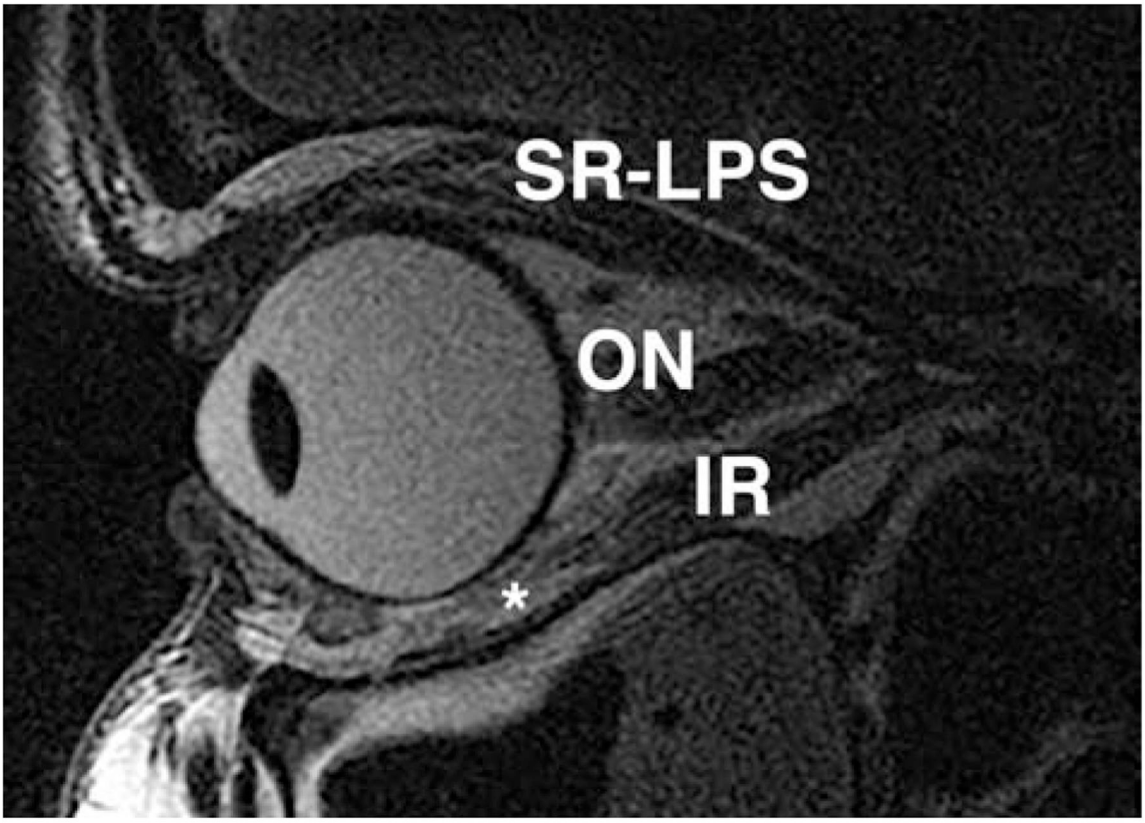

FIG 9.

Case 9. Sagittal T2 MRI showing disrupted connective tissue in the region of the IR pulleys. The anterior third of the IR exhibits a T2-weighted imaging signal (*) similar to that of the orbital fat, with normal signal more posteriorly and intact innervation.

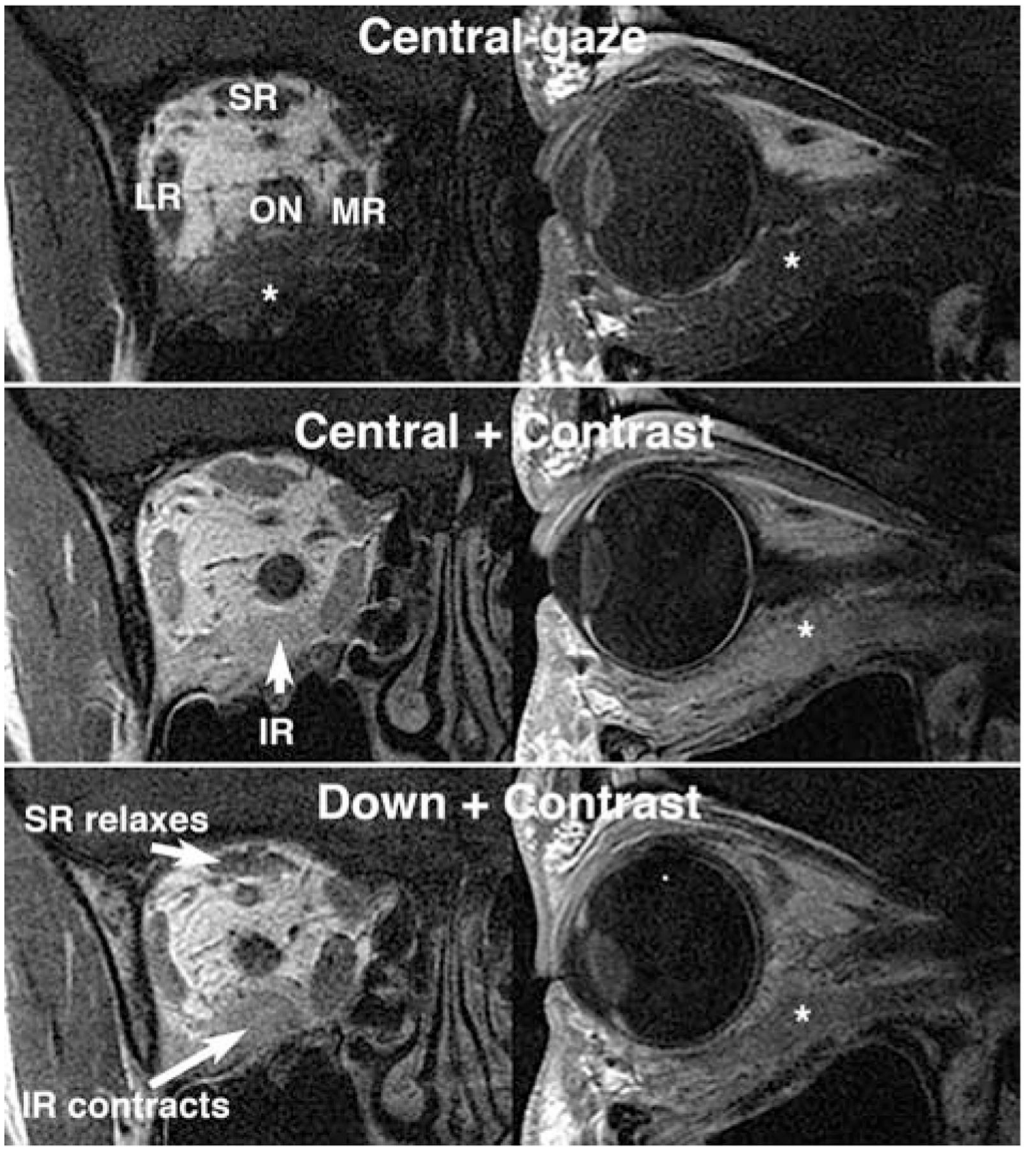

FIG 10.

Case 12. Coronal (left column) and sagittal (right column) T2 MRI in central (top and mid rows) and down gaze (bottom row) showing a functional IR in normal continuity with the scleral insertion surrounded by a large contrast enhanced mass (*). Right IR shows changes in attempted infraduction.