

Treatment of acquired Brown syndrome in a child with a single intramuscular systemic depot steroid injection

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A 10-year-old girl presented with a complaint of diplopia and mild superomedial orbital pain in the left eye of 2 weeks' duration. She had limited elevation of the left eye, especially in adduction, moderate limitation of elevation in the primary position, mild limitation of elevation in abduction, downshoot in adduction, mild hypotropia in the primary position, and normal abduction. There was mild swelling and tenderness in the superomedial aspect of her left orbit. Fundus examination revealed intorsion of the left fundus on upgaze. She was diagnosed with acquired Brown syndrome, due presumably to a local inflammatory cause, and treated with a single intramuscular depot injection of betamethasone in her deltoid muscle. One week later, her symptoms were resolving, and there was marked improvement of elevation of the left eye in adduction, with near normal elevation in the primary position and in abduction. There was no recurrence 3 months later.

Brown syndrome is characterized by limitation of elevation of the affected eye in adduction due to shortening or inelasticity of the superior oblique muscle-tendon complex.^{1,2} It has been described as both congenital and acquired. Reported causes of acquired Brown syndrome include idiopathic, surgical or nonsurgical trauma, and inflammation due to sinusitis or tenosynovitis that can be associated with connective tissue diseases.¹⁻⁵ We report, to our knowledge, the first case of acquired Brown syndrome in a child successfully treated with a single intramuscular systemic depot steroid injection.

Case Report

A 10-year-old girl presented at Cairo University Hospital with double vision of acute onset and progressive

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course of 2 weeks' duration. She also complained of mild aching orbital pain in the left eye that started around the same time. Her parents noticed that she had developed ocular deviation especially on looking in certain directions. She had no past ocular history but was noted to have a cold and sinusitis a few days prior to this event. Examination revealed a corrected distance visual acuity of 20/20 in both eyes. Ocular motility examination revealed left hypotropia in the primary position of gaze, with limited elevation of the left eye especially when adducted (Figure 1A). Motility of the right eye was normal. Orbital examination revealed mild swelling and tenderness in the superomedial aspect of the left orbit. Anterior segment and posterior segment examination were unremarkable except for intorsion of the left fundus, more marked on upgaze. The patient was diagnosed as having acquired Brown syndrome, with a suspected local inflammatory cause, and a single dose of 1 ml of betamethasone (7 mg/ml) was injected in her left deltoid muscle. Blood samples were simultaneously drawn, and a complete blood picture, erythrocyte sedimentation rate, and antinuclear antibodies test were performed and proved normal a few days later. We performed laboratory tests to exclude an underlying connective tissue disease. One week later, there was improvement of symptoms, with recovery of normal muscle balance and ocular motility (Figure 1B). On follow-up 3 months later, there was no recurrence of symptoms and no complications.

Discussion

Acquired Brown syndrome in the present case was suspected, from the history and clinical examination, to have been caused by the spread of local inflammation from sinusitis or by idiopathic tenosynovitis, which was reported to have had a prolonged course.^{1,2,6} Limited elevation of the affected eye in adduction is the main feature of Brown syndrome. Other clinical features include mild deficient elevation in abduction and in primary position, downshoot in adduction, and hypotropia in primary position, all of which were also present in our patient.^{2,7} We did not perform imaging studies, because the clinical picture was clear, and current opinion does not favor routine imaging.²

Previously, local orbital steroid injections have been reported to be effective in the treatment of acquired Brown syndrome using 40 mg of methylprednisolone acetate.^{6,8} The reported cases, however, required frequent retreatment, which may be unsuitable or risky for children, who may be uncooperative or require sedation or general anesthesia during the local injections. Although orbital steroid injections are probably less associated with systemic side effects compared to systemic steroid administration, they are not free of side effects, and cases of systemic complications following

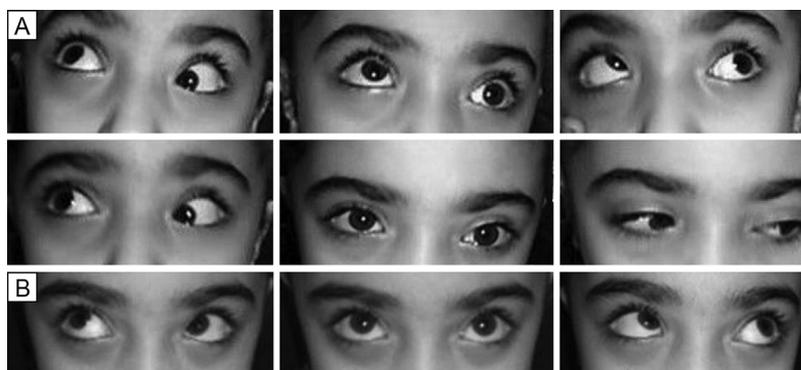


FIG 1. A, Ocular motility examination at presentation. There was marked limitation of elevation of the left eye in adduction, moderate limitation of elevation in the primary position, and mild limitation of elevation in abduction. There was also downshoot in adduction, mild hypotropia in the primary position, with normal abduction. B, Ocular motility 1 week following treatment. There was marked improvement of elevation of the left eye in adduction, with near normal elevation in the primary position, and in elevation in abduction.

repeated orbital methylprednisolone acetate injections have been reported in children.⁹

While spontaneous improvement of acquired Brown syndrome has been previously reported,² the rapid and dramatic improvement of the condition following treatment in this case suggests that improvement was due to treatment with a single intramuscular systemic depot steroid injection.

Literature Search

PubMed was searched on January 5, 2019, without date restriction, for English-language results, using the following terms: *acquired Brown syndrome, treatment, systemic steroids, intramuscular systemic steroid, and depot steroid injection.*

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Circumferential retinal hemorrhages after ophthalmic examination with scleral depression in an infant with anti-VEGF treated retinopathy of prematurity

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We report a case of retinal hemorrhages in a baby with retinopathy of prematurity (ROP) following examination with indirect ophthalmoscopy and scleral depression. There have been rare reports of examination-induced retinal hemorrhages during ROP screening, although those hemorrhages were diffusely scattered in the posterior pole. In this report the hemorrhages were found on the surface of the neovascular ridge. Changes in intraocular pressure caused by scleral depression may result in rupture of the fragile and immature retinal vessels, which have poor autoregulation in these premature babies. Ophthalmologists performing ROP screening examinations should be

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