Fig. 3—Picture of sibling A's pupils (current age 5 years) and sibling B's pupils (current age 2 years) in light and in darkness. Note the small physiological anisocoria in sibling A's pupils.

phenomenon in a 6-year-old female patient, and the series by Woods et al.[4] included a 5-year-old female patient.

The 2 sisters in this case are the youngest ever reported in the literature and were 24 months old and 8 weeks old, respectively, at onset. It is important to note that not only are they both first-degree relatives, but they had a first-degree relative (mother) with a history of migraines. This augments the very likely possibility of a familial link. As they were both very young when it occurred, it was not possible to ascertain if they were migraine sufferers themselves. Neuroimaging can be a difficult decision in this age group, and so understanding the natural history of this phenomenon is helpful in reassuring the family and determining clinical management, which in most cases is simply observation.

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Brown's syndrome during pregnancy: a case report and review of literature

Brown's syndrome is an uncommon strabismus characterized by restriction of elevation in adduction and can be congenital or acquired.[1–3] Clinical features include mild impaired elevation on upgaze, minimal/no elevation deficit on abduction, and minimal/no superior oblique overaction.[1–3]

Acquired Brown's syndrome can be due to superior oblique or trochlear etiology such as peritrochlear scarring in chronic sinusitis; tendon-trochlear inflammation in rheumatoid arthritis, often associated with superonasal orbital tenderness; superonasal orbital mass, such as neoplasm; or an inelastic superior oblique muscle as in thyroid disease. The differential diagnosis of deficient elevation in adduction includes monocular elevation deficiency, inferior oblique paresis, and superior oblique overaction.[2,3]

We report a rare case of Brown's syndrome in pregnancy.

A 34-year-old pregnant female presented to the emergency eye clinic with sudden-onset binocular vertical diplopia upon wakening with isolated right eye pain the previous day. She was 33 weeks pregnant with her second child. The current and previous pregnancies were both uncomplicated. Her medical history was significant for episodes of intermittent, nonerythematous, painful bilateral knee swelling 4 years prior with no definite diagnosis, controlled with oral nonsteroidal anti-inflammatory medications. Current medications were iron supplements. Her medical history was noncontributory, with no history of ocular trauma, rheumatoid arthritis, or sinusitis.

Fig. 1—Ocular motility examination at presentation showing an elevation deficit OD in straight up gaze, marked elevation deficit upon adduction OD; there was a slight right hypotropia in primary gaze (not shown).

On examination, visual acuity was 20/20 OU. Pupils were equal and reactive to light, with normal intraocular pressures. Ocular motility examination showed an elevation deficit OD in straight up gaze, −4 elevation deficit on adduction OD, and a slight right hypotropia in primary gaze (Fig. 1) and no compensatory head tilt. There was point tenderness over the right trochlea. Forced ductions showed marked restriction to elevation in adduction OD. Cranial nerves 2, 3, and 5–12 were intact bilaterally. Anterior and posterior segments were normal OU with no proptosis or palpable mass.

She was diagnosed with acute-onset right Brown's syndrome of unknown etiology. After discussing treatment options with the patient and her obstetrician, a peritrochlear triamcinolone injection 10 mg was administered with no complications. Inflammatory work-up revealed normal complete blood count (apart from mild normocytic anemia) and normal c-anti-neutrophilic cytoplasmic antibody, antinuclear antibody, rheumatoid factor, syphilis, and Lyme serology. However, erythrocyte sedimentation rate (104 mm/h), C-reactive protein (CRP; 38.6 mg/L), and p-anti-neutrophil cytoplasmic antibody (200 AU/mL, normal range 0–99) were elevated.

Over the following 2 weeks, her diplopia resolved and the elevation deficit on adduction also decreased considerably. On examination at 36 weeks of pregnancy in the adult strabismus clinic, she was orthotropic in all positions of gaze at distance, and near. She had a −2.5 limited elevation in adduction right eye, improved from initial presentation, with resolved tenderness over the right trochlea. Six weeks after her initial presentation, she underwent an uncomplicated labour, giving birth to a healthy baby.

Two months postpartum she was orthotropic in all positions of gaze with resolved diplopia. The −2 elevation deficiency in adduction OD persisted, and a −1 elevation deficiency in adduction OS was detected. Her motility examination 16 months after initial presentation was unchanged. Systemically, she had recently developed right knee swelling and neck stiffness and was diagnosed with a seronegative spondyloarthritis. The significance of our patient's elevated p-ANCA value is unclear. Follow-up with her rheumatologist found her to be HLA-B27 positive, with sacroiliitis and chronic synovitis. No systemic treatment or surgery has been required for symptom management.

When a pregnant patient presents with diplopia, worrisome neurological disorders that affect ocular motility must be excluded, including parasellar and posterior fossa tumours, parasellar aneurysmal expansion, carotid-cavernous fistulas, and myasthenia gravis.[4] Ocular motility disorders limited to the extraocular muscles with onset during pregnancy are rarely reported; we are aware of only 3 published reports in the literature, the details of which are summarized below.

Jacobson[5] reported isolated superior oblique palsy in 3 pregnant women, with third-trimester onset, believed to be due to pregnancy-induced decompensation of a latent superior oblique palsy. Jacobson postulated that increases in extracellular fluid compartment during pregnancy caused sufficient expansion of the venous-filled cavernous sinus to exert mild compression or traction on the thin trochlear nerve residing in the lateral dural wall. Von Noorden[6] reported a case of nonresolving Brown's syndrome with onset during the first trimester, with no reported trochlear tenderness.

Christiansen and Thomas[7] described a case of postpartum Brown's syndrome with no clinical evidence of systemic inflammatory disease, treated with oral prednisone followed by peritrochlear injection of dexamethasone.[7] They suggested that pregnancy-associated fluid expansions, blood volume increase, and labour-associated Valsalva maneuvers might have exacerbated a previously subclinical trochlear stenosis or tenosynovitis.

Our case is the first to report acute-onset Brown's syndrome with diplopia in the third trimester of pregnancy with resolution of diplopia and improved ductions after peritrochlear steroid injection. The cause of our patient's Brown's syndrome is postulated to be a pregnancy-induced exacerbation of an underlying subclinical inflammatory process; the trochlear tenderness, response to steroids, elevated CRP,[8] and seronegative spondyloarthritis support this hypothesis.

In summary, Brown's syndrome should be included in the differential diagnosis of new-onset diplopia in pregnancy. It may be the first presentation of an underlying inflammatory process warranting a referral to rheumatology.

Treatment with peritrochlear steroids may resolve diplopia while limiting systemic exposure to medications that may affect the fetus.

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Endogenous endophthalmitis due to *Klebsiella pneumoniae* from an infected gallbladder

Endogenous endophthalmitis from *Klebsiella pneumoniae* carries both a poor visual and ocular prognosis.[1] Risk factors include concurrent liver abscesses, diabetes mellitus, old age, and an immunocompromised state.[2] The incidence of endophthalmitis in patients with liver abscesses is as high as 11%,[2] and this has led to recommendations for universal screening for patients with *Klebsiella* sepsis.[3]

We report a case of *Klebsiella* endophthalmitis in a patient with sepsis due to acute cholecystitis. Metastatic spread from an infected gallbladder is extremely rare. To our knowledge, we present only the second reported instance of *Klebsiella* endophthalmitis from an infected gallbladder.[4] Our findings demonstrate the importance of considering *Klebsiella* infection in a patient presenting with a painful, red eye in the setting of cholecystitis.

A 68-year-old male presented for ophthalmologic assessment after a 3-day history of redness, photophobia, and reduced vision in his left eye. His medical history was remarkable for non–insulin-dependent diabetes mellitus, chronic kidney disease, and a simple liver cyst identified in 2014. He was not on any regular medications, and he had no known allergies. He had been admitted to the hospital several days earlier with symptoms of right-upper-quadrant abdominal pain, fever, and intermittent confusion. Initial work-up had revealed an elevated white blood cell count and elevated liver enzymes. Abdominal ultrasonography and subsequent endoscopic retrograde cholangiopancreatography confirmed cholelithiasis with acute cholecystitis, with purulent material identified in the common bile duct. Both blood cultures and fluid from a gallbladder aspiration confirmed *K. pneumoniae* infection.

On ophthalmologic examination, his visual acuity was 20/30 OD and hand motions OS. His intraocular pressures were 16 mm Hg OD and 46 mm Hg OS by Tono-Pen (Reichert Technologies, Buffalo, N.Y.). His pupil was reactive OD but was fixed and irregular and responded minimally to light OS. There was no afferent pupillary defect. He had significant conjunctival injection and chemosis with 5 mm of proptosis OS. He had mild limitation in all positions of gaze OS. Anterior segment examination revealed diffuse microcystic corneal edema OS with a shallow, fibrinous anterior chamber with 4+ cells (SUN classification)[5] and a 1-mm hypopyon. There was a moderate cataract OS. Examination findings of the right eye were unremarkable, aside from a moderate cataract. Given the limited view of the posterior segment of the left eye, a B-scan ultrasound was performed (Sonomed; Escalon Medical Corp., Wayne, Pa.) and revealed marked vitritis, scleral thickening, and retinal detachment. Posterior segment examination of the right

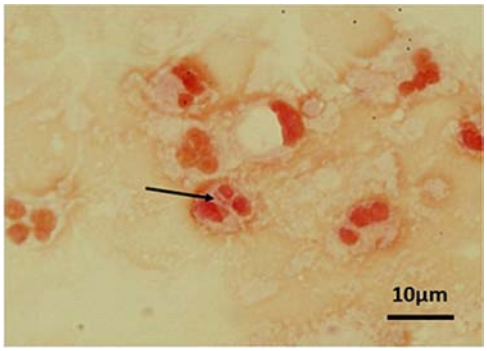

Fig. 1—Gram stain of the aspirate of intravitreal fluid from the left eye. Multiple neutrophils are apparent in this frame. There are 2 intracellular coliform gram-negative bacilli, identified by the solid black arrow, in the cytoplasm of a neutrophil in the centre of the image. In this clinical setting, these bacteria are consistent with *Klebsiella pneumoniae* infection.