

PediatricsⁱⁿReview[®]

Index of Suspicion

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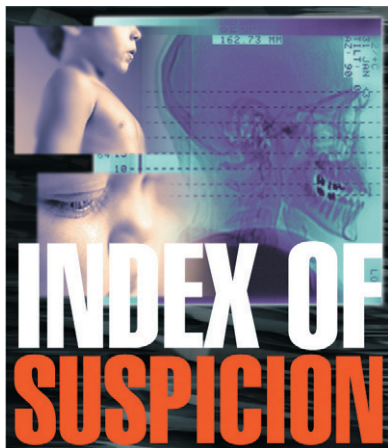
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The reader is encouraged to write possible diagnoses for each case before turning to the discussion.

The editors and staff of *Pediatrics in Review* find themselves in the fortunate position of having too many submissions for the Index of Suspicion column. Our publication slots for Index of Suspicion are filled through 2013. Because we do not think it is fair to delay publication longer than that, we have decided not to accept new cases for the present. We will make an announcement in *Pediatrics in Review* when we resume accepting new cases. We apologize for having to take this step, but we wish to be fair to all authors. We are grateful for your interest in the journal.

Author Disclosure

Drs Ananthanarayanan, Gereige, Al-Owain, Nguyen, Kansagra, Shaw, and McLean have disclosed no financial relationships relevant to these cases. This commentary does not contain a discussion of an unapproved/investigative use of a commercial product/device.

Case 1: Lymphadenopathy, Prolonged Hematuria, Proteinuria, and Weight Loss in a Teenage Boy

Case 2: Red, Swollen, Painful Eye in a 12-year-old Boy With Methylmalonic Acidemia

Case 3: Ptosis and Diplopia After a Respiratory Infection in a 7-year-old Girl

Case 1 Presentation

A 14-year-old African American boy presents with fever, right-sided neck swelling, and weight loss of 20 lb over 2-month period. He denies night sweats, illness contacts, or recent travel. He was seen 1 month ago for fatigue, body aches, decreased appetite, and 10 lb weight loss over the previous month. Due to a family history of diabetes, urinalysis was performed, which revealed moderate hemoglobin, protein of 200 mg/dL, white blood cell (WBC) count of 13/high power field, and red blood cell (RBC) count of 5/high power field. He was scheduled to be re-evaluated in 1 week but has been lost to follow-up.

The boy appears tired. His weight is 54.2 kg (>50th percentile), height is 165 cm (75th percentile), temperature is 38.9°C, heart rate is 106 beats/minute, respiratory rate is 20 breaths/minute, and blood pressure is 113/64 mm Hg. Physical examination reveals a 3 × 4 cm, tender, non-erythematous, nonfluctuant, slightly mobile right cervical mass located behind the sternocleidomastoid muscle. Findings on the rest of the examination are normal.

Complete blood count reveals a WBC count of $5.5 \times 10^3/\mu\text{L}$, with 65% granulocytes, 30% lymphocytes, and 5% monocytes, platelet count of $267 \times 10^3/\mu\text{L}$, hemoglobin level 9.5 g/dL, hematocrit 29%, mean corpuscular volume 71 fL, RBC distribution width 15.2%, and normal peripheral smear. His erythrocyte sedimentation rate (ESR) is 65 mm/h,

and C-reactive protein (CRP) level is 1.081 mg/dL. Metabolic panel reveals a sodium concentration of 141 mEq/L, potassium 4.5 mEq/L, chloride 111 mEq/L, bicarbonate 24 mEq/L, serum urea nitrogen 33 mg/dL, creatinine 1.7 mg/dL, total protein 6 g/dL, and albumin 1.9 g/dL. Urinalysis reveals a protein of 200 mg/dL and rare granular casts. Chest radiograph is normal. Blood and urine cultures are obtained, and a tuberculin test is performed on admission. Additional evaluation reveals the diagnosis.

Case 2 Presentation

A 12-year-old boy with methylmalonic acidemia and secondary renal impairment presents to the emergency department with 4-day history of a red, swollen, painful left eye. The swelling has been gradual in onset and worsens with eye movements. He was diagnosed as having periorbital cellulitis in a private clinic and treated with oral amoxicillin/clavulanate for 2 days before this visit.

Physical examination reveals an alert boy whose height is 128 cm and weight is 27 kg, both below the 3rd percentile. His temperature is 37.2°C, respiratory rate 20 breaths/minute, heart rate 79 beats/minute, and blood pressure 125/91 mm Hg. The left upper eyelid is erythematous, swollen, hot, and tender. He has mild exophthalmos and bulbar conjunctivitis of the left eye (Fig 1). There is ophthalmoplegia of the left eye, more pronounced on lateral gaze.



Figure 1. Chemosis and bulbar conjunctivitis associated with redness around the eye with mild proptosis. Note that the vascular engorgement is located more toward the lateral aspect of the left eye (lateral rectus position).

The remaining findings are normal except for mild hypotonia associated with the methylmalonic acidemia.

Laboratory evaluation reveals a WBC count of $7.81 \times 10^3/\text{mcL}$ with 64.9% neutrophils and 27.2% lymphocytes, hemoglobin of 10.2 g/dL, and platelet count of $151 \times 10^3/\text{mcL}$. His serum creatinine level is elevated at 116 $\mu\text{mol/L}$. However, serum levels of serum urea nitrogen, electrolytes, ammonia, creatine kinase, lactate dehydrogenase, aspartate aminotransferase, alanine aminotransferase, angiotensin converting enzyme (ACE), thyroid stimulating hormone, free thyroxine, and antithyroid

Frequently Used Abbreviations

ACE:	angiotensin converting enzyme
CNIII:	cranial nerve III
CNS:	central nervous system
CSF:	cerebrospinal fluid
CT:	computed tomography
ESR:	erythrocyte sedimentation rate
Ig:	immunoglobulin
LN:	lupus nephritis
RBC:	red blood cell
SLE:	systemic lupus erythematosus
TB:	tuberculosis
WBC:	white blood cell

peroxidase antibody are within normal range. His ESR is 30 mm/h, and CRP is 5.9 mg/dL. An imaging study leads to the correct diagnosis.

Case 3 Presentation

A previously healthy 7-year-old girl is hospitalized for evaluation of right eyelid droop and double vision for 2 days. She denies eye pain, redness, or drainage. Two weeks before presentation, she had several days of runny nose, fever to 38.3°C, and cough. Those symptoms resolved; however, she has since experienced intermittent headaches. She denies recent travel or known tick exposure. She reports no trauma, vertigo, syncope, weakness, numbness, paresthesias, dysphagia, or dysarthria. Her family history is significant for a grandmother who had a brain aneurysm.

Physical examination reveals an alert, oriented, obese girl who has normal cardiovascular, pulmonary, and abdominal findings. The neurologic examination reveals a right eye that is deviated downward and laterally with notable ptosis. She is able only to abduct the right eye, with no vertical movement or adduction past midline noted. The left eye has normal extraocular movement. The right pupil is dilated to 5 mm, is minimally reactive to light, and has no consensual response when light is shone into the left eye. The left pupil is 3 mm and reacts normally, with a normal consensual response. Her visual acuity is 20/100 in the right eye and 20/30 in the left eye. The remainder of her neurologic examination is completely normal. Her gait is intact.

Complete blood count, serum electrolyte levels, and cerebrospinal fluid (CSF) studies are normal. Her ESR is 21 mm/h and CRP level is 0.48 mg/dL. Further laboratory and imaging studies reveal the diagnosis.

Case 1 Discussion

The patient was started empirically on clindamycin for lymphadenitis. The differential diagnosis at this point included cervical lymphadenopathy or lymphadenitis, malignancy, and an autoimmune disorder. Ultrasonography of his neck revealed right posterior cervical and supraclavicular lymphadenopathy. Rheumatoid factor was <20, antinuclear antibody was positive, and antidouble stranded DNA antibody titer was positive at 3663. He was diagnosed as having systemic lupus erythematosus (SLE). It was important to rule out malignancy and active tuberculosis (TB) before starting corticosteroids. The tuberculin test was negative and this result was attributed to low T lymphocyte numbers (absolute CD3 count of $511/\text{mm}^3$ [71%], absolute CD4 count of $252/\text{mm}^3$ [35%], absolute CD8 count of $238/\text{mm}^3$ [33%]).

Therefore, a serum interference- γ release assay was done and came back positive. This test measures a cell-mediated immune response as a sign of TB infection. Right cervical lymph node biopsy revealed lupus lymphadenopathy, and kidney biopsy revealed stage III lupus nephritis (LN). The patient was presumed to have latent TB and started on isoniazid and pyridoxine and treated for 9 months. He also was started on lisinopril and corticosteroids. He was placed on a high calorie/high protein diet and has started gaining weight.

The Condition

LN is one of the most serious manifestations of SLE and usually arises within 5 years of the diagnosis of SLE. LN is an inflammatory process caused by glomerular deposition of autoantibodies. The incidence is 3:10,000 in the general population and occurs in 30% to 90% of individuals who have SLE. LN is more common

in African American individuals, Asian individuals, children older than 15 years, and females.

The causes of SLE are unknown. SLE is more common in first-degree relatives of patients who have the disorder, with higher concordance in monozygotic than in dizygotic twins. The concordance rate in monozygotic twins is not 100%, suggesting an environmental trigger. Other risk factors include human lymphocyte antigen class II, cytokine and mannose-binding lectin genes, and complement C2, C4 deficiencies. The pathogenesis of LN is based on at least three potentially overlapping mechanisms: (1) circulating DNA anti-DNA immune complexes deposited in the kidney; (2) complement activation and leukocyte-mediated injury triggered by in situ antigen-antibody complexes; and (3) renal injury caused by antibodies against specific cellular targets.

Clinical Manifestations

Fewer than one-half of patients have symptoms such as fever, fatigue, weight loss, rash, arthritis, serositis, and anemia of SLE at the time of diagnosis of LN. Patients with active LN often present with proteinuria, hypoalbuminemia, peripheral and sometimes periorbital edema, normal or mildly elevated creatinine levels, and hypertension. Clinically, the disease is evaluated by urinalysis, concentrations of serum creatinine and albumin, anti-DNA titers, and serum complement levels. Abnormal urinary findings include albuminuria, leukocyturia, hematuria, casts (granular, hyaline, RBC, and fatty), and oval fat bodies. A rising anti-DNA titer, together with hypocomplementemia and a low C3 level, is a strong indicator of active lupus renal disease. Hypoalbuminemia with significant proteinuria and hypercholesterolemia are markers of the nephrotic syndrome, which may accompany active lupus renal disease.

LN is defined also histologically by renal biopsy, light microscopy, immunofluorescence, and electron microscopy. A kidney biopsy is used to confirm the diagnosis, determine the stage, and guide the choice of appropriate treatment. Patients with “silent” LN have normal urinalyses and serum creatinine levels but may show evidence of mesangial to proliferative nephritis on renal biopsy.

Management

Treatment depends on clinical manifestations and the results of renal biopsy. Treatment is indicated for patients with membranous changes and severe nephrotic syndrome. Patients generally are started on corticosteroids, ACE-inhibitors, lipid lowering agents, and anticoagulants as needed. Immunosuppressive therapy is used in patients with membranous LN who have persistent severe and symptomatic nephrotic syndrome; in patients with nephrotic syndrome experiencing protein excretion >3.5 g/day; and in patients with mixed membranous and proliferative lesions on biopsy. Agents used are cyclophosphamide, cyclosporine, mycophenolate mofetil, and chlorambucil. Maintenance of adequate nutrition with high protein and calories is very important. Despite treatment, some patients with LN develop progressive loss of kidney function leading to renal failure requiring dialysis and eventually transplantation.

Complications and Prognosis

Complications include nephrotic syndrome, acute renal failure, chronic renal failure, and end-stage renal disease. Prognosis depends on the specific form of LN. Better outcomes are reported in patients with pure membranous LN. Patients with proliferative lesions are more likely to develop end-stage kidney disease.

Lessons for the Clinician

- In patients with LN, active or latent TB must be ruled out before starting corticosteroids.
- An interference- γ release assay should be considered as the test of choice in patients with an SLE flare-up and negative tuberculin skin test to rule out active or latent TB.
- Dietary modification is very important and includes restrictions in sodium intake in patients with hypertension, restrictions in protein intake in patients with renal insufficiency, and restrictions in fat intake in patients with hypercholesterolemia. In the absence of the conditions mentioned above, a diet high in protein, fat, and calories is recommended.

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Case 2 Discussion

The symptoms and findings on physical examination (see Fig 1) suggested the diagnosis of orbital cellulitis in this patient. The normal thyroid studies and ACE level excluded orbital thyropathy and sarcoidosis, respectively. Both computed tomography (CT) and MRI are valuable for diagnosing orbital pathology. The orbital CT in this patient revealed moderate enlargement of the lateral rectus muscle on the left with associated enlargement of the muscle tendon at the insertion into the sclera (Fig 2). The medial, superior, and inferior rectus muscles appeared normal. There was no sinus involvement or abnormality in the course of the optic nerve. This pattern was diagnostic of orbital pseudotumor.

The Condition

The best definition of orbital pseudotumor is an inflammatory mass of

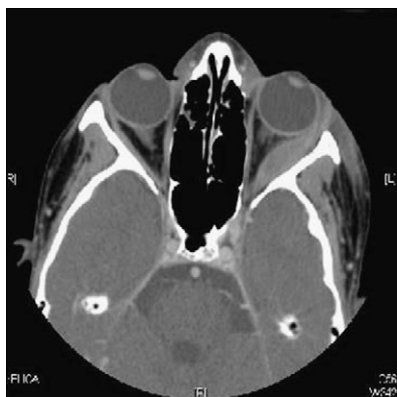


Figure 2. CT scan of the orbit revealing fusiform swelling of the left lateral rectus.

unknown etiology that involves orbital tissue and simulates a tumor. Orbital myositis, in which there is nonspecific orbital inflammation that involves primarily the extraocular muscles, often is included under the broad description of orbital pseudotumor. Sometimes these two terms are used interchangeably.

Orbital pseudotumor is more prevalent in adults than children. In two large series, only 6% to 16% of patients who had orbital pseudotumor were younger than 20 years of age. The condition usually is unilateral, and the majority of studies indicate no sexual predilection.

The cardinal feature of orbital pseudotumor is acute orbital pain exacerbated by eye movement. Other characteristics include decreased ocular motility, lid or conjunctival edema, and proptosis of varying degrees. Unusual symptoms include cluster headache. Bilaterality, trauma, and iritis are more common in children than in adults with orbital pseudotumor.

The cause of orbital pseudotumor still remains obscure. The condition is likely to be autoimmune in origin, with viral, genetic, and environmental factors implicated as possible triggers. Involvement can range from a single extraocular muscle to the entire orbit. Pathologically, the pseudotumor is

characterized by a mixed infiltrate of plasma cells, lymphocytes, macrophages, and polymorphonuclear cells, with fibrosis seen in the chronic form.

Diagnosis

Orbital pseudotumor should be considered in a patient with an acutely painful, proptotic eye. Leukocytosis and elevated ESR and CRP levels are nonspecific findings that suggest an active inflammatory process. Thyroid stimulating hormone and free thyroxine tests are performed to exclude thyroid-related ophthalmopathy. Testing for antinuclear antibody and more specific autoantibodies may be warranted if autoimmune diseases such as SLE are suspected. Imaging with orbital CT or MRI is essential for the diagnosis. Fine needle aspiration and biopsy should be reserved for patients having an atypical clinical course, a poor response to systemic steroid therapy, or recurrence of the disorder. These imaging techniques are helpful also in ruling out orbital malignancies or other diseases.

Orbital pseudotumor is associated with SLE, myasthenia gravis, Crohn disease, Lyme disease, Kawasaki disease, and paraneoplastic syndromes.

Differentiating orbital tumor from orbital cellulitis is extremely important (Table). In orbital pseudotumor, usually there is no associated sinus infection, the condition does not respond to antibiotic therapy, and characteristic CT findings are present.

Management

Systemic corticosteroid therapy is the accepted primary treatment for the pseudotumor of the orbit. Recurrent or chronic orbital myositis may be difficult to treat, requiring long-term corticosteroids, immunotherapy, chemotherapy, radiation therapy, or surgery. Failure to institute early therapy may result in permanent restriction of ocular mobility. Although orbital

pseudotumor is considered a self-limiting disease, the duration without treatment is unpredictable. The condition responds rapidly to corticosteroids, and complete resolution occurs within 1 to 2 weeks. However, the process may recur in up to 30% of patients, usually within the first 6 months.

This patient was started on intravenous ceftazidime and clindamycin and did not reveal any response for 48 hours. Once the diagnosis of orbital pseudotumor was established, he was started on oral prednisone (2 mg/kg per day) for 3 weeks, followed by tapering over another 3 weeks. He showed dramatic improvement within 48 hours and full resolution within 2 weeks. Follow-up at 3, 6, and 12 months revealed normal eye findings without recurrence.

Lessons for the Clinician

- Orbital pseudotumor, also known as orbital myositis, is an idiopathic orbital inflammation.
- The diagnosis relies on clinical, radiologic, and sometimes pathologic findings.
- The condition should be differentiated from orbital cellulitis and neoplasms.
- Orbital pseudotumor has a good response to systemic corticosteroid therapy.

(Mohammed Al-Owain, MD, Department of Medical Genetics, King Faisal Specialist Hospital and Research Center, Riyadh, Saudi Arabia)

Case 3 Discussion

MRI of the head revealed thickening and abnormal enhancement of the cisternal segment of the right oculomotor nerve (Fig 3). Magnetic resonance angiography did not reveal a vascular aneurysm. MRI/magnetic resonance angiography was chosen over

Table 1. Differential Diagnosis of Orbital Pseudotumor

Diagnosis	Important Features
Orbital cellulitis	Characterized by proptosis, chemosis, ophthalmoplegia, and potentially decreased visual acuity and commonly caused by direct bacterial extension from the ethmoid sinus. Aggressive and prompt treatment is mandatory. Very uncommon in the setting of normal sinuses in children.
Preseptal (periorbital) cellulitis	No limitation of eye movement and no proptosis. CT scan reveals swelling of the lids and subcutaneous tissue anterior to the orbital septum. Antibiotic therapy is the treatment of choice.
Contiguous sinusitis	Nasal congestion and discharge, cough, fever, halitosis, and less commonly periorbital edema and facial pain. There is sinus tenderness sometimes. Sinus CT scan helps in establishing the diagnosis.
Dacryoadenitis (inflammation of the lachrymal gland)	Acute form can be viral or bacterial. Chronic form is seen with systemic diseases such as syphilis, tuberculosis, and sarcoidosis.
Orbital neoplasm	Benign tumors include hemangiomas and dermoid and epidermoid cysts. The most common malignant tumors are rhabdomyosarcoma, retinoblastoma (orbital extension), lymphosarcoma, metastatic neuroblastoma, and optic glioma. Biopsy is required for definitive diagnosis. Although lymphoid tumors probably are the most common orbital neoplasms in adults, they are exceedingly rare in children.
Trauma	Should be suspected in the pediatric age group, especially in the presence of stigmata of child abuse.
Foreign body	Conjunctival foreign body may give a picture indistinguishable from pseudotumor. Thorough ophthalmologic examination is necessary for diagnosis.
Orbital thyroid disease	Secondary to immune mechanism leading to inflammation of the extraocular muscles and fat. Characterized by exophthalmos, proptosis, and lid retraction and associated with late motility problems. Usually bilateral and not accompanied by constitutional symptoms.

CT/CT angiography for greater diagnostic specificity of structural brain lesions as well as less radiation exposure; additionally, there was low suspicion for a hemorrhage or vasculopathic



Figure 3. Contrasted T-1 axial image of patient's brain MRI, revealing thickening and abnormal enhancement of the cisternal segment of the right oculomotor nerve.

process. These images could not distinguish tumor from an infectious or inflammatory condition.

Blood and CSF samples were obtained in search of an infectious agent and for markers of inflammation. CSF studies were negative for evidence of neurosyphilis, Lyme disease, acid fast bacilli, *Cryptococcus*, fungal infections, cytomegalovirus, Epstein-Barr virus, and herpes simplex virus. The CSF also revealed no evidence of leukemic cell infiltrate, oligoclonal bands suggestive of multiple sclerosis, or ACE levels suggestive of neurosarcoidosis. Serology for Lyme disease was negative and serum ACE levels were normal. However, serum immunoglobulin M (IgM) and IgG antibody titers against *Mycoplasma* were elevated by enzyme immunoassay. The positive IgM *Mycoplasma* titer was later confirmed by immunofluorescence antibody assay.

Differential Diagnosis

The differential diagnosis of isolated cranial nerve III (CNIII) palsy is extensive, ranging from acutely life-threatening to relatively benign conditions. One important point of discrimination is the presence or absence of isolated extraocular movement involvement, isolated pupillary involvement, or both. The parasympathetic fibers that mediate pupillary constriction run along the outside of the oculomotor nerve, whereas innervation to extraocular muscles and the levator palpebrae muscle are more interior. Therefore, structural lesions that are compressive are more likely to present initially with pupillary involvement with sparing of extraocular movements, whereas vascular injuries to the nerve are more likely to affect extraocular movements with pupillary sparing. The following discussion

applies to processes that may lead to complete CNIII palsies.

The location of injury to the oculomotor nerve can be within its midbrain nucleus or anywhere along the nerve as it courses through the brainstem, subarachnoid space, and cavernous sinus, then into the orbit. A brainstem process is likely to affect additional cranial nerves, but this involvement may not always occur. It is worth noting that many cases of isolated oculomotor nerve palsies are idiopathic.

The causes of CNIII palsy include vascular, malignant, infectious or parainfectious, inflammatory, traumatic, congenital, toxic, and migranous conditions. Vascular causes merit particular attention, because an expanding aneurysm, most commonly of the posterior communicating artery, needs prompt intervention. Although more common in adults, aneurysms must be excluded in children. Other vascular causes include infarct or ischemia of the nucleus or nerve, vasospasm of the arterial blood supply, carotid artery-cavernous sinus fistula, and vascular malformation within the midbrain.

Malignant causes include schwannoma, carcinomatous meningitis, teratoma, meningioma, lymphangioma, and leukemia. Infectious causes are numerous but most notable are *Mycoplasma pneumoniae* infection, cysticercosis, measles, *Cryptococcus* infection, Lyme disease, TB, and HIV. The CNIII palsy may be due to either direct infection or result from a parainfectious process that may be immune-mediated. Abscess formation within the brainstem may selectively affect only a single cranial nerve.

Inflammatory causes include Wegener granulomatosis, Tolosa-Hunt syndrome, vasculitis, sarcoidosis, SLE, and demyelinating processes such as multiple sclerosis. Trauma to the orbit and ophthalmoplegic migraines can lead to isolated CNIII

palsy but should be diagnosed with caution in the appropriate clinic setting and only after other conditions have been carefully excluded. The same caution applies to the diagnosis of congenital CNIII palsy.

The Condition

Central nervous system (CNS) manifestations are the most common extrapulmonary manifestations of *Mycoplasma pneumoniae* infections, occurring in 1 in 1000 cases. CNS involvement more commonly presents as acute encephalitis, although meningitis, myelitis, facial nerve palsy, and radiculitis have been reported. A literature search reveals only one case report of another patient having specifically a CNIII palsy associated with *Mycoplasma* infection. (1) The pathophysiologic mechanism still is debated. *Mycoplasma* has been found in CSF, but the organism has never been recovered from brain tissue, making direct invasion less likely. The most accepted pathophysiologic explanation remains a postinfectious autoimmune inflammatory response targeting the CNS, often appearing ~1 week after a respiratory illness, usually accompanied by evidence of edema, perivascular infiltration, microthrombi, or areas of demyelination of CNS tissue.

Management and Prognosis

There are no published guidelines or case reports that discuss treatment of *Mycoplasma*-associated CNIII palsy. This patient was given initial treatment of prednisone 30 mg twice a day (0.75 mg/kg per day) on day 2 of admission, due to concern for a progressive inflammatory or parainfectious process. Within 24 hours, she had subtle improvement in her ptosis and pupillary constriction to light. She was started on azithromycin once the *Mycoplasma* result was

obtained. On follow-up 1 week after discharge, improvement had continued and she was able to adduct her involved eye past midline. At this point, she was placed on a corticosteroid wean, to be completed over a 2-week period.

She was lost to follow-up after this visit, so recovery could not be documented. A previous case report reveals recovery of function by 4 months (1); similarly, a report of a *Mycoplasma*-associated abducens nerve palsy revealed spontaneous resolution within 21 days and no recurrence at 10 months. (2)

Lessons for the Clinician

- There are many causes for isolated oculomotor nerve palsy.
- It is important to determine whether there is extraocular involvement, pupillary involvement, or both in order to help guide evaluation.
- Evaluation of isolated oculomotor nerve palsy may involve neuroimaging and lumbar puncture.
- *Mycoplasma pneumoniae* infection has been associated with many CNS disorders, including isolated oculomotor nerve palsy.

(Lisa Nguyen, MD, MPH, Sujay Kansagra, MD, Andrew Shaw, MD, Heather McLean, MD, Duke University Medical Center, Durham, NC.)

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