

PART I

HEENT (HEAD, EYES, EARS, NOSE AND THROAT)

1

Sore throat in a 29-year-old male

HISTORY OF PRESENT ILLNESS

A 29-year-old male with a medical history significant for type I diabetes presented to the ED complaining of a sore throat, inability to swallow solids and fevers to 103°F (39.4°C) for two days. He noted a hoarse voice and was able to tolerate only small sips of liquids. He denied significant neck swelling or stiffness, and was able to tolerate his secretions. He had immigrated to the United States from Mexico as a teenager, and his immunization status was unknown.

PHYSICAL EXAM

GENERAL APPEARANCE: The patient was a well-developed, nontoxic, moderately obese male who appeared slightly dehydrated, sitting upright and in no acute distress.

VITAL SIGNS

Temperature	103°F (39.4°C)
Pulse	100 beats/minute
Blood pressure	145/85 mmHg
Respirations	22 breaths/minute
Oxygen saturation	100% on room air

HEENT: Oropharynx was pink and moist, no erythema, exudates, tonsillar or uvular swelling noted.

NECK: Supple, anterior cervical lymphadenopathy noted, tenderness to palpation over cricoid cartilage noted.

LUNGS: Clear to auscultation bilaterally.

CARDIOVASCULAR: Regular rate and rhythm without rubs, murmurs or gallops.

ABDOMEN: Soft, nontender, nondistended.

EXTREMITIES: No clubbing, cyanosis or edema.

A peripheral intravenous line was placed and blood was drawn and sent for laboratory testing. Laboratory tests were significant for a leukocyte count of 24 K/ μ L (normal 3.5–12.5 K/ μ L) with 92% neutrophils (normal 50–70%). A soft-tissue lateral neck radiograph was obtained (Figure 1.1).



Figure 1.1 Soft-tissue lateral radiograph of the neck from a 29-year-old male with sore throat and inability to swallow solids.

What is your diagnosis?

ANSWER

The diagnosis is epiglottitis. The soft-tissue lateral neck radiograph demonstrates swelling of the epiglottis (“thumbprint” sign, Figure 1.2). The Ear, Nose and Throat (ENT) specialist was urgently consulted, and bedside nasopharyngoscopy demonstrated a swollen, red epiglottis with 90% obstruction of the upper airway. The patient received ceftriaxone 1 gm and decadron 10 mg intravenously, normal saline 1 liter IV bolus, and was admitted to the ICU for close monitoring and observation. The patient was discharged on hospital day #4 after his symptoms had improved. Repeat nasopharyngoscopy demonstrated significant improvement of the epiglottic swelling. He was continued on cefpodoxime proxetil (Vantin™) orally for ten days following discharge.

Epiglottitis in adults

Acute epiglottitis is a potentially life-threatening condition that results from inflammation of the supraglottic structures.^{1,2} Commonly considered a pediatric disease, the current incidence of epiglottitis in adults is 1 to 2 cases per 100,000, which is presently 2.5 times the incidence in children.¹ Epiglottitis occurs most frequently in men in the fifth decade; the disease is more common in countries that do not immunize against *Haemophilus influenzae* type B. Currently, the most common cause of epiglottitis is infection, although sources such as crack cocaine use have also been implicated.¹ Common pathogens include *H. influenzae* (Hib), β -hemolytic streptococci and viruses.

The clinical presentation of adult epiglottitis may differ significantly from that of the classic drooling child seated in a tripod position. The most common symptoms in adults are sore throat, odynophagia and muffled voice.³ Sore throat is the chief complaint in 75–94% of cases, whereas odynophagia may be present in as many as 94% of cases.⁴ The presence of stridor significantly increases the need for emergent airway intervention.⁵ Unlike children with epiglottitis, where emergency airway management is essential, most cases of adult epiglottitis do not require acute airway intervention due to the greater diameter of the adult airway.

The leukocyte count is greater than 10,000 in 80% of cases of adult epiglottitis. Soft-tissue lateral neck radiography, which may show an enlarged, misshapen epiglottis (“thumbprint” sign), has a sensitivity of 88% in establishing the diagnosis.³ Patients who appear ill or are in extremis should not leave the ED for radiographs, and airway management in patients in extremis should be the first and foremost responsibility. Direct laryngoscopy is the most accurate investigation to establish a diagnosis of epiglottitis.³ Management focuses on two important aspects: close monitoring of the airway with intubation (if necessary) and treatment with intravenous antibiotics.⁶ Antibiotics should be directed against Hib in every patient, regardless of immunization status. Cefotaxime, ceftriaxone or ampicillin/sulbactam are appropriate choices. Steroids are commonly used in the management of



Figure 1.2 Soft-tissue lateral radiograph of the neck from a 29-year-old male with epiglottitis, demonstrating “thumbprint” sign (arrow).

acute epiglottitis, although no randomized trials to date support this practice.⁶

KEY TEACHING POINTS

1. Acute epiglottitis is a potentially life-threatening condition resulting from inflammation of the supraglottic structures, with a current incidence of 1 to 2 cases per 100,000 adults in the United States.
2. Sore throat is the chief complaint in 75–94% of cases of adult epiglottitis, whereas odynophagia may be present in as many as 94% of cases.
3. Soft-tissue lateral neck radiography, which may show an enlarged, misshapen epiglottis (“thumbprint” sign), has a sensitivity of 88% in establishing the diagnosis.
4. The definitive diagnosis is made through direct laryngoscopic visualization of an enlarged, inflamed epiglottis.
5. Treatment of epiglottitis includes intravenous antibiotics and close airway monitoring in an ICU setting. Most clinicians treat acute cases with intravenous steroids.

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2

Left eye pain and discharge in a 44-year-old male

HISTORY OF PRESENT ILLNESS

A 44-year-old male with no significant medical history presented to the ED with several days of worsening left eye pain, discharge and decreased vision, as well as swelling and redness to the eyelid and surrounding tissue. He did not wear contact lenses or eyeglasses. The patient currently could not see from his left eye. He denied fevers, headaches or recent trauma to the eye, although he did report a foreign body sensation to the eye several days prior to the onset of these symptoms. He denied experiencing similar symptoms in the past.

PHYSICAL EXAMINATION

GENERAL APPEARANCE: The patient appeared to be in no acute discomfort.

VITAL SIGNS

Temperature	98.1°F (36.7°C)
Pulse	75 beats/minute
Blood pressure	135/85 mmHg
Respirations	22 breaths/minute
Oxygen saturation	100% on room air

EYES: The visual acuity of the right eye was 20/40; the right pupil was round and reactive to light. No vision or light perception was elicited from the left eye. The left upper and lower eyelids were swollen and erythematous. A thick yellow-green discharge exuded from the left orbit; the pupil could not be examined secondary to the thickness and adherence of the exudates. The left eye was proptotic (Figure 2.1).



Figure 2.1 A 44-year-old male with several days of swelling, redness and exudate of his left eye.

What is your diagnosis?

ANSWER

The diagnosis is endophthalmitis and orbital cellulitis of the left eye. A CT scan of the orbits revealed a markedly proptotic left ocular globe, as well as a markedly distended left anterior chamber with mild posterior displacement of the hyperdense lens (Figure 2.2). The CT also demonstrated enhancing infiltrate of the preseptal and periorbital soft tissues associated with the left orbit, consistent with orbital cellulitis. It was surmised that the patient had recently incurred a penetrating injury to the globe, which resulted in endophthalmitis with extension to orbital cellulitis.

The patient was started on intravenous antibiotics (ceftriaxone 1 gm q12 h and vancomycin 1 gm q12 h), as well as antibiotic eye drops (cefazolin and tobramycin, one drop each q1 h) and aggressive lubrication of the eye. He was admitted to the medical service with close ophthalmology involvement. By hospital day #10, no improvement in the patient's symptoms occurred despite aggressive antibiotic therapy, and the decision was made to surgically enucleate the left eye.

Periorbital cellulitis, orbital cellulitis and endophthalmitis

Periorbital cellulitis (also called preseptal cellulitis) is an infection that occurs anterior to the orbital septum. The orbital septum is a layer of fibrous tissue arising from the periosteum of the skull and continues into the eyelids. Because this layer provides an effective barrier against the spread of infection from the preseptal tissues into the orbit, periorbital cellulitis does not progress to orbital cellulitis.¹ Periorbital tissue may become infected either by trauma or due to primary bacteremia, which is especially common in young children at high risk for pneumococcal bacteremia.¹

Orbital cellulitis is post-septal, with involvement of the orbit itself. It is most commonly the result of a complication of ethmoid sinusitis, which accounts for more than 90% of all cases.² Orbital cellulitis can also be caused by the direct extension of infection from the globe, eyelids, ocular adnexae and other periocular tissues, in addition to the sinuses. Orbital cellulitis is more common in children but can also occur in adults.³ Along with eyelid edema and erythema seen in periorbital cellulitis, patients with orbital cellulitis have proptosis, chemosis (edema of the bulbar conjunctiva), impairment of and pain with extraocular movements, and decreased extraocular movements.¹⁻⁴ The presence of fever, systemic signs, and toxicity is variable in patients with orbital cellulitis.⁴ Bacterial causes of orbital cellulitis are most commonly *Streptococcus* species, *Staph aureus* and *Haemophilus influenzae* Type B.² *Pseudomonas*, *Klebsiella*, *Eikenella* and *Enterococcus* are less common culprits. Polymicrobial infections with aerobic and anaerobic bacteria are more common in patients 16 years or older.²

Endophthalmitis is a serious intraocular inflammatory disorder resulting from infection of the vitreous cavity.⁵ Exogenous endophthalmitis occurs when infecting organisms gain entry into the eye by direct inoculation, such as from



Figure 2.2 CT of the orbits of a 44-year-old male demonstrating a markedly proptotic left ocular globe (dark arrow), as well as a markedly distended left anterior chamber with mild posterior displacement of the hyperdense lens (white arrow).

intraocular surgery, penetrating trauma, or contiguous spread from adjacent tissues. Endogenous endophthalmitis occurs when infectious agents are hematogenously disseminated into the eye from a distant focus of infection.⁶ Progressive vitritis is a hallmark of any form of endophthalmitis. Histologically, there is massive infiltration of the vitreous cavity with inflammatory cells, primarily neutrophils.⁵ In most instances, vitreous infiltration is accompanied by progressive intraocular inflammation associated with loss of vision, pain, and hypopyon. Further progression may lead to panophthalmitis, corneal infiltration and perforation, orbital cellulitis, and phthisis bulbi (atrophy and degeneration of a blind eye).⁵ Decreased vision and permanent loss of vision are common complications of endophthalmitis. Patients may require enucleation to eliminate a blind and painful eye. The most important laboratory studies are Gram stain and culture of the aqueous and vitreous humor.⁶

Diagnostic testing for orbital cellulitis starts with contrast-enhanced CT imaging of the orbits.³ Findings on CT indicating orbital cellulitis include any of the following: proptosis, inflammation of the ocular muscles, subperiosteal abscess or frank orbital abscess.¹ Ipsilateral (or bilateral) sinusitis should also be evident (except in cases where orbital cellulitis is caused by extension of endophthalmitis). Sinus disease is important in the pathogenesis of orbital cellulitis but not of periorbital cellulitis.¹ Purulent material from the nose should be collected with cotton or calcium alginate swabs and submitted for Gram stain and culture on aerobic and anaerobic media; any material obtained from the sinuses or directly from an orbital abscess should be assessed in the same manner.²

Uncomplicated post-traumatic periorbital cellulitis can generally be treated with oral antimicrobials directed against Gram-positive bacteria (e.g., cephalexin, dicloxacillin or clindamycin).¹ The patient with orbital cellulitis should be promptly hospitalized for treatment, with ophthalmology and infectious disease consultations. Historically, the presence of subperiosteal or intraorbital abscess was an indication for surgical drainage in addition to antibiotic therapy; however, medical management alone is successful in many cases.² Intravenous broad-spectrum antibiotics (e.g., second- and third-generation cephalosporins or ampicillin/sulbactam) should be started immediately until antibiotics can be tailored to pathogens identified on culture.^{2,3} Typically, intravenous antibiotic therapy should be continued for 1 to 2 weeks, followed by oral antibiotics for an additional 2 to 3 weeks.^{1–3}

Treatment of traumatic endophthalmitis includes admission to the hospital with ophthalmologic consultation, intravenous antibiotics (including vancomycin and an aminoglycoside or third-generation cephalosporin) and topical fortified antibiotics.⁶ Intravitreal antibiotics should be administered by an ophthalmologist, with consideration for pars plana vitrectomy.^{5,6} Tetanus immunization is necessary if immunization is not current.

KEY TEACHING POINTS

1. Clinical signs and symptoms of orbital cellulitis include eyelid edema and erythema, proptosis, chemosis, pain with extraocular movements and ophthalmoplegia.
2. The diagnostic test of choice for orbital cellulitis is a contrast-enhanced CT scan of the orbits, demonstrating proptosis, inflammation of the ocular muscles, and subperiosteal or orbital abscess.
3. Treatment of orbital cellulitis includes administration of broad-spectrum intravenous antibiotics with close ophthalmology involvement.
4. Endophthalmitis, a serious intraocular inflammatory disorder resulting from infection of the vitreous cavity, may progress to orbital cellulitis.
5. Treatment of traumatic endophthalmitis includes hospital admission and administration of intravenous, topical and intravitreal antibiotics.

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3

Sudden, monocular vision loss in a 62-year-old female

HISTORY OF PRESENT ILLNESS

A 62-year-old female with no significant medical history presented to the ED complaining of seeing “floaters” and flashing lights in her right eye associated with loss of vision in the right inferolateral visual field (affecting only her right eye) for the past three days. She did not wear eyeglasses or contact lenses, and denied pain, redness or discharge from the eye. She denied trauma to the eye or headaches.

PHYSICAL EXAMINATION

GENERAL APPEARANCE: The patient appeared well developed and in no acute discomfort.

VITAL SIGNS

Temperature	98.6°F (37°C)
Pulse	80 beats/minute
Blood pressure	135/85 mmHg
Respirations	20 breaths/minute
Oxygen saturation	100% on room air
Visual acuity	OD 20/100 OS 20/20 OU 20/40

EYES: PERRL, EOMI and no afferent pupillary defect. Lids, lashes and lacrimal glands normal, no conjunctival or scleral injections, no ocular discharge. The cornea was clear, without edema, fluorescein uptake or cloudiness. No cell or flare on slit lamp examination. Right inferolateral visual field deficit affecting the right eye only. Retinal examination of both undilated eyes was normal. Intraocular pressures were 12 mmHg OD and 16 mmHg OS, respectively.

A linear, 10-MHz ultrasound probe was gently placed over the closed right eye using a small amount of water-soluble gel (Figure 3.1).



Figure 3.1 Ultrasound image of the right eye from a 62-year-old female with 3 days of floaters and an inferolateral visual field deficit of the right eye.

What is your diagnosis?

ANSWER

The diagnosis is retinal detachment. The ultrasound image demonstrates an inferolateral detachment of the retina (arrow, Figure 3.2). The ophthalmologist was urgently consulted, and confirmed the presence of retinal detachment upon dilated retinal examination of the right eye. The patient underwent microsurgical repair of the retinal detachment, regaining normal vision of her right eye.

Retinal detachment

Retinal detachment (RD) involves separation of the retina from the underlying retinal epithelium (Figure 3.3). It affects approximately 2 in 10,000 people per year.¹ Risk factors for the development of RD include increasing age, previous cataract surgery, focal retinal atrophy, myopia, trauma, diabetic retinopathy, family history of RD, uveitis, and prematurity.¹ Patients complain of new floaters, squiggly lines or cobwebs that appear abruptly, associated with visual field loss.² Over time, the patient may report a shadow in the peripheral visual field, which, if ignored, may spread to involve the entire visual field in a matter of days.³ Vision loss may be filmy, cloudy, irregular or curtain-like.

Examination of the eyes should begin with an assessment of the visual acuity. The external eye examination should include inspection for any signs of trauma and confrontational visual field testing; the latter can assist in isolating the location of the RD.³ Pupillary reaction should be checked, and a slit lamp biomicroscopy performed. Intraocular pressures should be measured in both eyes, as hypotony of more than 4–5 mmHg less than the unaffected eye is common in RD.³ Finally, a fundoscopic examination with ophthalmoscopy is required. This examination may not reveal the RD, particularly in an undilated eye, as the detachment may be at the periphery of the retina where the retina is the thinnest.²

Patients with new onset visual loss can be rapidly assessed for RD using bedside ultrasound, readily performed by

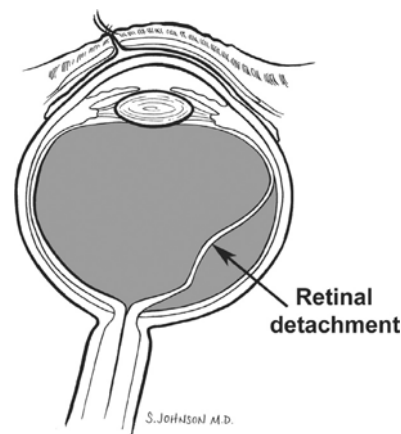


Figure 3.3 Retinal detachment.

emergency physicians.^{4–8} The diagnosis of retinal detachment can be made using almost any ultrasonographic probe, although a linear probe such as the 7.5- to 10-MHz probe is preferred. The patient is asked to look straight ahead with closed, but not clenched, eyelids. The probe is placed perpendicular to the orbit, using conduction gel and minimal pressure to obtain the image.⁵ Care should be taken to avoid placing excessive pressure on the globe while performing the scan, particularly if there is concern for globe rupture (in the setting of trauma).

RD requires urgent consultation with an ophthalmologist. Surgical repair of retinal detachments, typically performed by a retinal specialist, has a high success rate.¹ More invasive therapies, such as scleral buckling and posterior vitrectomy, have success rates of nearly 90%, whereas less invasive therapies, such as pneumatic retinopexy, may be performed in an office setting in select cases. If the repair is technically successful, visual acuity is often restored to predetachment levels.¹

KEY TEACHING POINTS

1. Retinal detachment (RD) involves separation of the retina from the underlying retinal epithelium, and affects approximately 2 in 10,000 people per year.
2. RD is an ophthalmologic emergency, requiring urgent ophthalmology consultation.
3. Patients with RD complain of new floaters, squiggly lines or cobwebs that appear abruptly, associated with visual field loss.
4. Key components of the ocular examination include visual acuity testing, gross and slit lamp inspection of both eyes, visual field confrontation and extraocular movement assessment, pupillary response to light and accommodation, testing of intraocular pressures, and fundoscopic examination (best performed on a dilated eye).
5. Bedside ultrasound is a useful and readily available tool for making the diagnosis of RD in the emergency department.

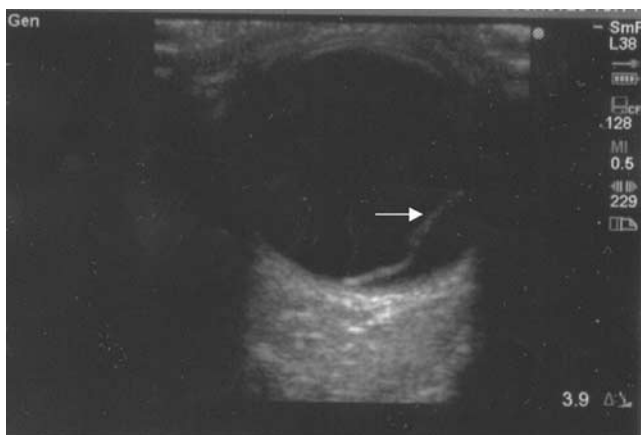


Figure 3.2 Ultrasound image of right eye from a 62-year-old female demonstrating inferolateral retinal detachment (arrow).

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4

Syncope and monocular vision loss in a 76-year-old female

HISTORY OF PRESENT ILLNESS

A 76-year-old female with a medical history significant for hypertension, hypothyroidism, hyperlipidemia and chronic renal insufficiency presented to the ED after fainting while shopping. She reported feeling lightheaded and dizzy while walking in a grocery store, at which time she experienced a brief loss of consciousness. She fell to the floor, bystanders called emergency services but she quickly regained consciousness. She denied head trauma or neck pain after the fall. She also denied chest pain but had been experiencing some shortness of breath with exertion over the past several weeks, as well as low back pain and bilateral thigh pain worse in the morning. She also noted gradually worsening vision in her right eye over the past week, increasing redness to that eye, and a mild, right-sided headache; her vision prior to this had been normal. Upon presentation to the ED, she could perceive light and vague shapes with the right eye. Her medications included metoprolol, hydrochlorothiazide, Cozaar, levothyroxine, lovastatin and ibuprofen 400 mg orally twice daily for low back pain. She denied tobacco use, drank alcohol occasionally, lived alone and drove a car for transportation.

PHYSICAL EXAMINATION

GENERAL APPEARANCE: The patient appeared well hydrated and well nourished, and in no acute discomfort.

VITAL SIGNS

Temperature	98°F (36.6°C)
Pulse	54 beats/minute
Blood pressure	138/53 mmHg
Respirations	22 breaths/minute
Oxygen saturation	98% on room air
Visual acuity	OS 20/50 OD light perception and hand motion only (unable to count fingers)

HEENT: Atraumatic, normocephalic, PERRL, EOMI, no afferent pupillary defect. Sclera of the right eye red and injected, no discharge. Tenderness to palpation over right forehead. No facial lesions or asymmetry noted.

NECK: Supple, no jugular venous distension.

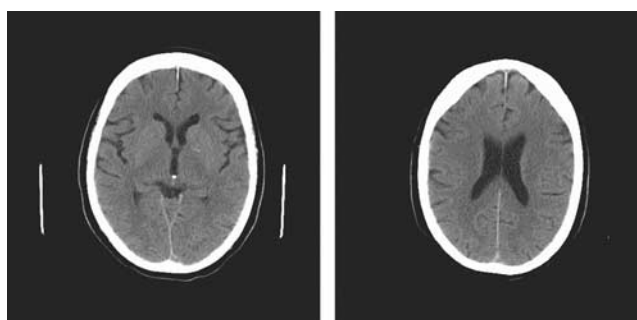


Figure 4.1 Noncontrast CT of the brain from a 76-year-old female with syncope and monocular vision loss.

CARDIOVASCULAR: Bradycardic rate, regular rhythm without rubs, murmurs or gallops.

LUNGS: Clear to auscultation bilaterally.

ABDOMEN: Soft, nontender, nondistended.

RECTAL: Normal tone, brown stool, hemoccult negative.

EXTREMITIES: No clubbing, cyanosis or edema.

NEUROLOGIC: Visual acuity deficit of right eye (cranial nerve II) as described; remaining neurologic examination nonfocal.

A peripheral intravenous line was placed and blood was drawn and sent for laboratory testing. A 12-lead ECG demonstrated sinus bradycardia, rate 56, without the presence of ST-T wave changes. A noncontrast CT of the brain was obtained (Figure 4.1). Laboratory tests were significant for a leukocyte count of 12.6 K/ μ L (normal 3.5–12.5 K/ μ L) with 81% neutrophils (normal 50–70%), hematocrit of 27% (normal 34–46%), creatinine of 1.7 mg/dL (normal < 1.1 mg/dL), erythrocyte sedimentation rate (ESR) of 120 mm/hr (normal 0–20 mm/hr) and C-reactive protein (CRP) of 18.2 mg/dL (normal < 0.9 mg/dL). The electrolytes, glucose and troponin I were within normal limits, and a urinalysis did not show signs of infection.

What is your diagnosis?