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Acute Bacterial Rhinosinusitis

Evidence-Based Management and Optimizing Antibiotic Therapy

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Approximately 20 million cases of acute bacterial rhinosinusitis (ABRS) are managed annually in the United States. According to National Ambulatory Medical Care Survey (NAMCS) data, sinusitis is the fifth most common diagnosis for which an antibiotic is prescribed, and accounted for 7-12% of all antibiotic prescriptions written from 1992 to 1999. In 1996, the primary diagnosis of sinusitis lead to expenditures of approximately $3.39 billion in the United States.

Acute bacterial sinusitis is one of the 10 most common diagnoses encountered in ambulatory practice. Primary care physicians frequently approach sinusitis as the manifestation of acute bacterial infection and prescribe an antibiotic in 85-98% of cases. However, sinusitis commonly is caused by viral infection, and often will resolve without antibiotic treatment, even if it is bacterial in origin.

Acute rhinosinusitis is defined by symptom duration of fewer than four weeks. Acute bacterial sinusitis usually is a secondary infection resulting from sinus ostia obstruction, impaired mucus clearance mechanisms caused by an acute viral upper respiratory tract infection, or both. According to epidemiologic estimates, only 0.2-2% of viral upper respiratory tract infections in adults are complicated by bacterial rhinosinusitis. The accepted standard for the definitive diagnosis of bacterial sinusitis is sinus puncture, with Streptococcus pneumoniae *and* Haemophilus influenzae *bacteria most commonly isolated from infected maxillary sinuses. However, sinus puncture is an invasive procedure seldom performed in the primary care setting, and as a result, other criteria must be evaluated as triggers for antimicrobial therapy.*

Because no simple and accurate office-based test for acute bacterial sinusitis currently is available, emergency clinicians must rely on clinical findings and historical features to confirm the diagnosis. This may be problematic, since signs and symptoms of acute bacterial sinusitis and

those of prolonged viral upper respiratory tract infections are very similar, resulting in frequent misclassification of viral cases.

As expected, the diagnosis and management of acute bacterial sinusitis is a fiercely debated topic, with expert opinion varying as to when antibiotic therapy is appropriate and which specific antibiotics should be employed as first-line therapy. A recent set of recommendations published by the American College of Physicians (ACP) concluded that most cases of acute rhinosinusitis diagnosed in ambulatory care are caused by uncomplicated viral upper respiratory tract infections, and that specific clinical triggers should be utilized to initiate antibiotic-centered management.

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Given the importance of this commonly encountered outpatient infection, this issue of Emergency Medicine Reports *attempts to outline in evidence-based detail what the authors conclude to be optimal, risk-stratified, empiric treatment recommendations for this patient population. In addition, this review identifies key clinical findings, resistance patterns, risk factors, coexisting conditions, and other clinical triggers supporting referral of patients with ABRS to an otolaryngologist for more invasive—i.e., multimodal surgical and more intensive antimicrobial—management strategies.*

The ultimate goal is to provide a concise, practical, and clinically relevant schemata for day-to-day patient management in which evidence can be put into practice to optimize clinical outcomes in patients with ABRS.

—The Editor

Definition of the Problem

The term sinusitis refers to inflammation of the mucosa lining one or more of the paranasal sinuses. The condition is invariably associated with contiguous inflammation of the nasal mucosa. Viral or allergic rhinitis typically precedes sinusitis, and sinusitis without rhinitis is rare. Thus, rhinosinusitis may be the more appropriate term to describe this disease process and will be used throughout this review. Sinus inflammation occurs in 90% of individuals with the common cold.[1] Acute bacterial rhinosinusitis (ABRS) results when this sinus inflammation is complicated by bacterial infection and occurs in up to 2% of cases of patients with colds.[2] Most cases of rhinosinusitis affect the maxillary and ethmoid sinuses. Isolated infection of the sphenoid and frontal sinus is rare, more serious, and usually is bacterial in origin.[3-5]

Emergency physicians should be aware of several challenges associated with the diagnosis and management of ABRS. Although most cases of ABRS are caused by viral upper respiratory infections, bacterial and viral causes are difficult to differentiate on clinical grounds. The diagnosis often is presumptive, and the treatment is empirical.[6] The problem that arises is that many patients receive antibiotics for rhinosinusitis, which is not only ineffective against the more likely viral pathogen, but contributes to resistance of various bacteria to these drugs. Furthermore, the antibiotics that are prescribed frequently are used indiscriminately.[7] The purpose of this paper is to review rhinosinusitis, particularly acute bacterial rhinosinusitis in the adult population, and to provide simple guidelines for the emergency physician in the diagnosis and management of this common disease.

Classification

Rhinosinusitis is classified as acute, subacute, or chronic based on the duration of symptoms. Acute rhinosinusitis may last as long as 4 weeks. Symptoms of subacute rhinosinusitis may persist for 4-12 weeks. Chronic rhinosinusitis is defined by symptom duration of greater than 12 weeks. Recurrent acute rhinosinusitis requires four or more episodes of acute rhinosinusitis lasting at least seven days each in any one-year period.[8] Patients can have recurrent acute attacks or an acute exacerbation of chronic rhinosinusitis.

Table 1. Predisposing Events for Sinusitis

- Prior upper respiratory tract infection
- Concurrent group A streptococcal infection
- Allergic rhinitis
- Anatomic variation (adenoidal hypertrophy, deviated septum, cleft palate, nasal polyps)
- Barotrauma from swimming
- Dental infection
- Exposure to irritants such as smoke
- Iatrogenic factors (nasogastric tubes, mechanical ventilation, nasal packing, dental procedures)
- Secretory disturbance (cystic fibrosis)
- Abnormalities in mucociliary clearance
- Hormonal changes (pregnancy)
- Medication side effect (abuse of topical vasoconstrictors or cocaine)
- Immunodeficiency, including diabetes mellitus

Adapted from: Brook I. Medical management of acute bacterial sinusitis, recommendations of a clinical advisory committee on pediatric and adult sinusitis. *Ann Otol Rhinol Laryngol* 2000;109 (suppl 182):2-20.

Epidemiology

The symptoms attributable to sinusitis are one of the most common reasons for visits in the acute care setting. Sinus disease affects more than 10% of the population.[9] Approximately one billion cases of viral rhinosinusitis can be expected to occur annually in the United States alone, which can be complicated by 20 million cases of ABRS, assuming a 2% complication rate and an estimated 3-4 acute respiratory illnesses a year.[10] The prevalence of acute rhinosinusitis is increasing according to data from the National Ambulatory Medical Care Survey, up from 0.2% of diagnoses at office visits in 1990 to 0.4% of diagnoses at office visits in 1995.[11] Respiratory infections such as rhinosinusitis are a leading cause of morbidity and a significant financial burden to society.[12] There is considerable cost associated with the symptoms, diagnosis, treatment, and complications of acute bacterial rhinosinusitis. In addition to the direct costs of office visits, diagnostic tests, and treatment drugs and modalities, the spectrum of the financial impact also includes cost of time lost from work for illness and office visits, the injudicious use of antibiotics, and inpatient costs for complications or morbid infections. In 1992, Americans spent $200 million on prescription medications for rhinosinusitis and more than $2 billion on over-the counter medications.[11] Fortunately, patients with uncomplicated ABRS rarely need to be hospitalized. However, complications due to spread of infection into the periorbital space or central nervous system often require urgent hospitalization and aggressive treatment.

Pathophysiology

The paranasal sinuses are comprised of four paired air-filled cavities within the skull: maxillary, ethmoid, frontal, and sphenoid. The frontal sinuses are not present in young children and become fully developed around 12 years of age. The sphenoid sinuses are the last to develop and first appear around 7 years of age. The frontal and sphenoid sinuses become clinically important in the teenage years when they become infected in pansinusitis.[4] The frontal, anterior ethmoid, and maxillary sinuses drain into the ostiomeatal complex located in the middle meatus, lateral to the middle turbinate. The sphenoidal sinus and posterior ethmoidal cells drain into the sphenoethmoidal recess.

The sinuses and nasal cavity are lined with ciliated pseudostratified columnar epithelium that serves both a functional and protective role. This area is rich in goblet cells that secrete mucus onto the surface epithelium. This blanket of mucus acts as a lubricant for the cilia and helps to trap inhaled particles. Mucus and debris are transported toward the ostia by the beating of the cilia and are expelled into the nasal airway. The sinuses are normally sterile, even though the sinus epithelium is contiguous with the nasal passages which are colonized with bacteria. This sterility is maintained by mucociliary clearance and immunologic host defense mechanisms. If the sinus ostia are obstructed, mucociliary flow is impaired. Obstruction of these ostia results in an anaerobic, high-carbon dioxide, and stagnant environment that can facilitate bacterial growth.[13]

Sinus infection usually occurs when predisposing events (*see Table 1*) result in inflammation and obstruction of drainage into the ostiomeatal complex. Obstruction of these ostia appears to be a critical factor in precipitating infection.[14] The presence of bacteria in the absence of obstruction appears to be insufficient to cause disease.[15] The most common predisposing factor is mucosal inflammation from viral upper respiratory infection or allergic rhinitis.[16] Predisposing conditions may contribute to the increased incidence of ABRS as the inflammation impedes mucus flow and drainage.[17,18] This relationship is at least as strong in the pediatric population.[19]

Certain anatomic variants may decrease the mucus drainage from the sinuses, thus increasing pooling and the possibility of bacterial overgrowth. Nasal polyps are thought to predispose to rhinosinusitis in two ways. In addition to impeding sinus drainage, their association with asthma and the associated mucociliary inflammation both increase the incidence of infection and inflammation.[20-22] Immunodeficiency also is a risk factor for the development of ABRS.[23] Previous trauma, intranasal cocaine use, and swimming all can lead to rhinosinusitis, as they upset the natural anatomy and pH of the turbinates, meatus, and sinuses.[24]

Rhinosinusitis should not be viewed simply as an infection treated only with antibiotics, but rather a complex process involving inflammatory processes that may include bacterial, viral infection, and obstructive phenomena.[25]

Etiology

Episodes of ABRS almost always appear as complications of viral upper respiratory infections, and less frequently with allergic rhinitis. The most common pathogens isolated from sinus drainage cultures in acute rhinosinusitis are *Streptococcus pneumoniae*, *Hemophilus influenzae*, *Moraxella catarhallis*, and *Staphlyococcus aureus*.[26-29] The infection is polymicrobial in

Table 2. Diagnostic Criteria for Acute Bacterial Rhinosinusitis (ABRS)

Diagnosis of ABRS made with two or more major criteria, or one major and two or more minor criteria.

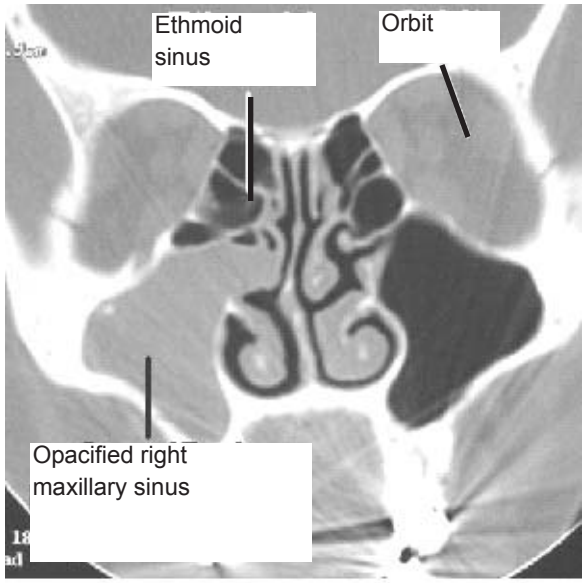
MAJOR CRITERIA	MINOR CRITERIA
• Facial pain or pressure	• Headache
• Facial congestion or fullness	• Dental pain
• Fever	• Cough
• Nasal purulent discharge or discolored postnasal drainage	• Fatigue
• Nasal obstruction or blockage	• Ear pain, pressure, or fullness
• Hyposmia or anosmia	• Halitosis
• Purulence in nasal cavity on examination	

*Facial pain/pressure alone does not constitute a suggestive history for ABRS in the absence of another major nasal sign or symptom.

**ABS is likely when symptoms worsen after five days or persist for more than 10 days

Adapted from: Lanza DC, Kennedy DW. Adult rhinosinusitis defined. *Otolaryngol Head Neck Surg* 1997;117(3 Pt 2):S1-7. Osguthorpe JD. Adult rhinosinusitis: Diagnosis and Management. *Am Fam Physician* 2001;63:69-76.

Figure 1. Coronal CT Scan Showing Right Maxillary Sinus Opacification



Note the septal deviation to the right and the hypertrophy of the left inferior turbinate.
Used with permission from: Bechara Y. Ghorayeb, MD, www.ghorayeb.com/imagingmaxillarysinusitis.html.

about one-third of the cases.[30] Enteric bacteria are recovered less commonly.[30] If resolution of the acute phase of rhinosinusitis does not take place, anaerobic bacteria of oral flora origin become predominant over time.[6] Anaerobic infections typically also occur in association with dental disease.[31] Although the organisms responsible for ABRS have remained unchanged, their susceptibility to a variety of antibiotics has changed significantly.[32]

Certain populations are more susceptible to developing complicated sinus infections based on anatomic variants or co-morbid conditions. Loss of immunocompetence related to HIV infection, chemotherapy, posttransplant immunosuppression, insulin-dependent diabetes mellitus, or some connective tissue disorders predisposes patients to rhinosinusitis and increases the likelihood of its persistence.[4] *Pseudomonas aeruginosa* and other aerobic gram-negative rods are common in rhinosinusitis of nosocomial origin (e.g., nasogastric tubes or nasotracheal intubation), in immunocompromised individuals, and in patients with cystic fibrosis.[30] Fungal rhinosinusitis is uncommon and is found in either atopic individuals who develop an allergic reaction to certain fungal pathogens or in immunocompromised patients, including diabetics who develop a much more invasive form of this disease.[33]

Differential Diagnosis

The differential diagnosis for ABRS includes viral or allergic rhinosinusitis, chronic rhinosinusitis, and other head and neck infections. Because the clinical spectrum of these other etiologies overlaps, many practitioners treat empirically with antibiotics.

The etiology most commonly mistaken for ABRS is viral rhinosinusitis. This fact alone likely contributes to the overuse of antibiotics prescribed in the ambulatory, urgent care, and emergency services population. Viral infections frequently are bilateral and have concomitant upper respiratory infection symptoms, and are suggested by more clear mucus secretions. In making a distinction between viral and bacterial sources of sinus infection, one often has to treat empirically using adjunctive therapies (without antibiotics) based on signs and symptoms. If after seven days the patient has persistent or worsening symptoms, the diagnosis of bacterial infection is suggested. Chronic rhinosinusitis also is in the differential, and may be difficult to distinguish from both allergic and bacterial rhinosinusitis. Other infections of the head and neck initially may present in a similar fashion to that of acute ABRS, but a thorough history and physical exam (and occasionally necessary ancillary studies) usually can delineate the location of infection.

Helpful features in differentiating bacterial from allergic causes include unilaterality of the pain and secretions, acute onset, fever, and preceding upper respiratory infection symptoms in patients with ABRS. Patients suffering from allergic rhinosinusitis often are misdiagnosed with a bacterial infection. There are common features, in addition to the fact that patients prone to allergic rhinosinusitis have risk factors for developing bacterial rhinosinusitis. These include inflammation and mucosal thickening as well as subsequent impedance of meatal mucus flow. Finally, other causes for headache pain also should be in the differential. Migraine headaches may be associated with nasal symptoms and should be considered.[34]

Clinical Features

Rhinosinusitis is an inflammatory disease of the paranasal sinuses and nasal cavity that can be characterized by a variety of

Figure 2. Waters' View of the Sinuses Showing Partial Opacification of the Right Maxillary Sinus, with an Air-Fluid Level

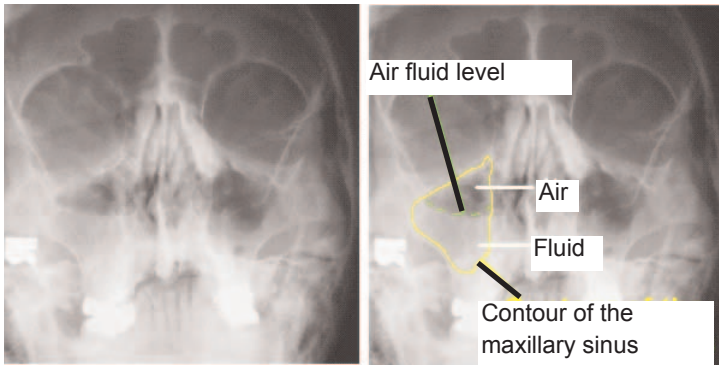


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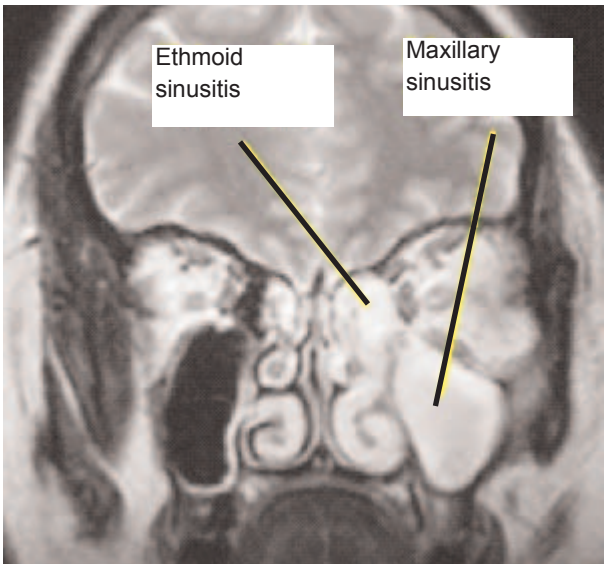
Figure 3. Coronal MRI Scan Showing Opacification of the Left Maxillary and Ethmoid Sinuses.



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presentations. An upper respiratory infection or a history of one may be present.[35] The diagnosis often is based on symptoms indicating maxillary or frontal sinus involvement. Headache is the most prevalent symptom of bacterial rhinosinusitis.[36] However, all rhinosinusitis pain is not the same. Maxillary rhinosinusitis pain most typically is located in the cheek, the gums, and the teeth of the upper jaw. Ethmoid rhinosinusitis pain is felt between the eyes. The eyeball may be tender, and pain may be aggravated by eye movement. Frontal rhinosinusitis pain is felt mainly in the forehead. Sphenoid rhinosinusitis pain is felt in the vertex, but has a more general localization.[4] According to the Task Force on Rhinosinusitis of the American Academy of Otolaryngology-Head and Neck Surgery, diagnosis of acute bacterial rhinosinusitis depends on the presence of at least two major or one major and two minor criteria.[8] (*See Table 2.*) Headache, fever, facial pain, and typical upper respiratory infection symptoms are the most common, although there are many different presentations. Unilateral facial pain suggests actual sinus infection because usually a single sinus is involved. On the other hand, bilateral facial pain and congestion would be seen more commonly with a straightforward viral upper respiratory infection. Blockage of sinuses often leads to other symptoms such as anosmia and halitosis, and the purulent rhinorrhea often may lead to a persistent cough.[23] Often, a patient presents with typical upper respiratory infection symptoms that have worsened over several days, and obstruction of secretions in the sinuses causes pain, especially upon bending forward.[37]

The physical examination may not be helpful, particularly in sphenoid rhinosinusitis in which the diagnosis often is missed.[4] Visualization of purulent nasal discharge on examination may be a strong indicator of ABRS;[38] however, pus is not always seen in sphenoid rhinosinusitis.[39] Sinus tenderness on physical examination is suggestive but not specific for rhinosinusitis.[40] Transillumination of the sinuses has low sensitivity and specificity,[35] and routine anterior rhinoscopy performed with a headlight and nasal speculum allows only limited inspection of the anterior nasal cavity.[4]

Diagnostic Studies

The diagnosis of acute bacterial rhinosinusitis can be made in several different ways. These include symptom complex, physical exam, endoscopy, sinus puncture and culture, and radiologically with computed tomography (CT), magnetic resonance imaging (MRI), plain films, or ultrasonography.

Many studies have been performed to try to identify diagnostic criteria for ABRS to determine the need for antibiotic treatment or further care. Predictors for bacterial infection include: purulent nasal discharge, unilaterality of pain on exam, maxillary toothache, and poor responsiveness to over-the-counter treatments.[41-46] A randomized double-blind trial found that history of purulent nasal discharge and signs of pus in the nasal cavity and throat were the most predictive of responsiveness to antibiotics. These criteria were better than radiography, which was even better than c-reactive protein or CRP in this study.[38] However, there is limited evidence to suggest that clinical criteria have better diagnostic accuracy than sinus radiography.[11]

Often, however, clinical exam cannot definitely detect a sinus infection. CT has become the most widely used diagnostic radiography for acute bacterial rhinosinusitis. Imaging must be performed in the coronal plane to adequately demonstrate the ethmoid complex. The mucosa of the normal, noninfected sinus approximates the bone so closely that it cannot be visualized on CT. Therefore, any soft tissue seen within a sinus is abnormal.[47] Findings consistent with sinus inflammation include air-fluid levels, sinus opacification, sinus wall displacement, and mucosal thickening of 4 mm or greater. (*See Figure 1.*) IV contrast may be required to evaluate central nervous system or orbital complications.[48] Unfortunately, CT findings of sinus inflammation are not diagnostic of ABRS. In a study by Gwaltney et al, 87% of

patients who presented with the "common cold" and no previous history of rhinosinusitis had maxillary sinus abnormalities; 65% had ethmoid sinus abnormalities; and 30-40% had frontal or sphenoid sinus abnormalities on CT. This same study reported that up to 40% of asymptomatic adults have abnormalities on sinus CT scans.[1] Sinus CT is helpful in persistent disease, treatment failures, or for guidance of therapy when done preoperatively.[49] CT has not been shown to be cost-effective for the diagnosis of rhinosinusitis.[50] Otolaryngologists order far more CTs, as they see patients in referral who already have been treated and perhaps have a higher prevalence of complicated rhinosinusitis.[51]

Plain films of the sinuses are not sufficient to evaluate drainage patterns in all sinuses and thus are used infrequently. Standard radiography is inadequate for the clinical evaluation of rhinosinusitis because it does not evaluate the anterior ethmoid air cells, the upper two-thirds of the nasal cavity, or the infundibular, middle meatus, or frontal recess air passages.[52] Visualization of the sphenoid sinus also is limited.[4] Although such studies can disclose sinus opacification or reveal air fluid levels in the sinuses, one of the most commonly infected areas, the anterior ethmoid region, is poorly visualized on plain film radiographs.[53] (*See Figure 2.*) MRI also has been occasionally used, although its use has been limited more to evaluating complications of acute and chronic rhinosinusitis.[54] (*See Figure 3.*) Ultrasonography rarely is used in the evaluation of sinus disease. Studies evaluating its use in the diagnosis of rhinosinusitis reveal substantial variation in test performance.[55]

Although not routinely performed in the emergency department, examination of nasal secretions also has been used to establish a diagnosis as well as to tailor therapy. While nasal cytology has been used to establish a diagnosis through neutrophil counts,[56] culture of secretions is employed more commonly. Traditionally, sinus puncture has been used. Maxillary sinus puncture is performed through the lateral wall of the interior mental meatus.[57] Because of the invasive nature of this procedure, newer endoscopic sampling, which is much less painful, has been established as a reasonable alternative.[58] Nasal endoscopic sampling has been advocated as an adjunct to diagnosis and guidance in cases of treatment failure.[53] Culture diagnosis often takes 2-3 days to obtain a specific etiology and antibiotic sensitivities. Other laboratory studies are unnecessary in the evaluation and management of most patients with uncomplicated rhinosinusitis.

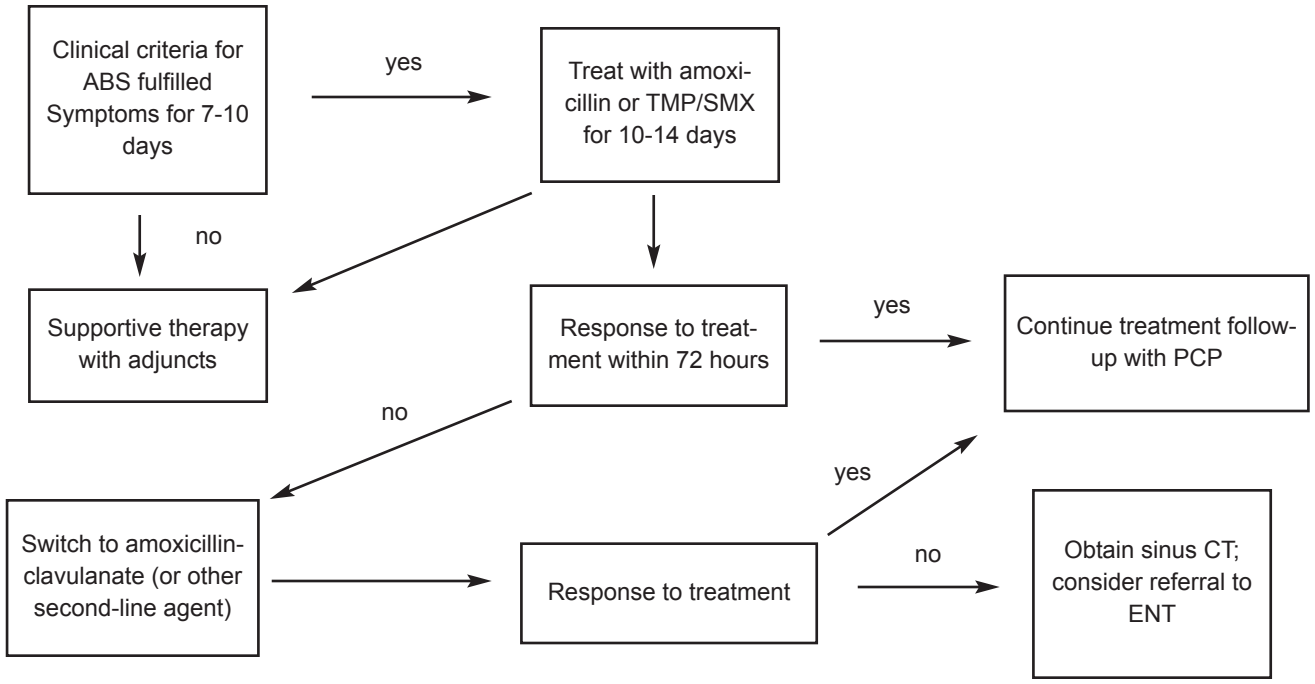
Management

The first and most important question to ask in treatment is whether to use antibiotics. It is extremely important to limit use of antibiotics to those infections requiring antimicrobials for recovery and clinical benefit. This is due to the very real phenomena of antibiotic overuse, increasing antibiotic resistance, and subsequent development of difficult-to-treat complications. Appropriate antibiotic therapy is of paramount importance, even though it is estimated that spontaneous recovery occurs in 48% of patients.[10] Most importantly, antibiotics are beneficial and effective in the prevention of the infectious complications of rhinosinusitis.

Most studies regarding antibiotic therapy in acute bacterial rhinosinusitis have been conducted in otolaryngology, non-emergency department settings. Some trials failed to find a difference between various antibiotics and placebo.[59-62] This is probably because antibiotics are unlikely to make a therapeutic difference in most cases of viral rhinosinusitis, which is most of the remainder of cases. After conducting its own systematic review, the Agency for Health Care Policy and Research (AHCPR) concluded that in patients with ABRS, more patients are cured and cured earlier, when treated with antibiotics rather than placebo.[11] Within the past several years, panels of experts have presented thorough reviews and made recommendations regarding the management of ABRS.[32,63-70] These consensus statements and systematic reviews support similar management guidelines for ABRS. They suggest that patients with rhinosinusitis symptoms lasting fewer than seven days are unlikely to have a bacterial infection. Treatment of ABRS should begin with the most narrow-spectrum agents active against the most likely pathogens, *S. pneumoniae* and *H. influenzae*. The currently recommended first line agents include amoxicillin or TMP-SMX. In areas in which resistance to *S. pneumoniae* is high, the dose of amoxicillin should be doubled (up to 80-90 mg/kg/day, maximum of 3 g/d).[32] Although the Sinus and Allergy Health Partnership recommends TMP-SMX only as an alternative in beta-lactam-allergic patients for the treatment of mild ABRS in adults and children who have not received antibiotics in the previous 4-6 weeks,[65] its use requires consideration of local resistance patterns and individual patient factors.[71] Prescribing physicians should consider factors that predispose patients to antibiotic-resistant bacteria, such as contact with children in daycare centers and recent antibiotic use. Despite these recommendations and consensus statements, some physicians still prescribe first-generation cephalosporins, such as cephalexin and doxycycline, for ABRS.

One study looked at antibiotic susceptibility for nasal swab samples of 16,213 cases of ABRS seen in outpatient primary care settings.[29] Of the four most common pathogens (*S. pneumoniae*, *H. influenzae*, *M. catarrhalis,* and *S. aureus*), only the fluoroquinolones had greater than 95% coverage of these pathogens. While the macrolides had decent coverage for *S. pneumoniae* isolates (64-68%), they did not fare so well against *S. aureus* (31-69%). Only 64% of *S. pneumoniae* isolates were susceptible to penicillin.[29] It is important to keep in mind that this study evaluated in vitro susceptibility of nasal swab isolates and did not measure clinical response. Because most of the studies evaluating clinical response of rhinosinusitis looked at penicillins (penicillin, amoxicillin or amoxicillin-clavulanate) or TMP-SMX, recommendations have been made based on these findings. Amoxicillin and TMP/SMX are recommended for first-line therapy for ABRS. Second-line agents include a variety of cephalosporins, fluoroquinolones, and macrolides. If symptoms do not improve in 72 hours, second-line antibiotic agents should be prescribed. For patients who fail to respond to initial therapy, sinus CT should be considered to confirm the diagnosis and assess for complications. In addition, referral to an ear, nose, and throat (ENT) specialist may be required. An algorithm for treatment of ABRS is found in Figure 4.

Figure 4. Algorithm for Treatment in Acute Bacterial Sinusitis (ABS)



While the consensus statements would suggest only a small proportion of patients with upper respiratory infection symptoms presenting to an acute care setting would benefit from antibiotic treatment, the reality is that many more are prescribed antibiotics. Although the prescription rate is improved somewhat from earlier studies showing between 85% and 98% prescription rates,[72,73] a more discriminate approach to antibiotic use is needed. Because many of the antibiotic vs. placebo trials have shown high cure rates without antimicrobials, it is important to limit antibiotic use. Recently, topical delivery of antibiotics has received attention as a method of decreasing systemic effects of antibiotic abuse. Nebulized solutions of locally delivered fosfomycin (*Monurol*) and tobramycin (*TOBI*) both have shown promise in treating ABRS. (The inhaled versions of both of these drugs are not FDA approved.)[74,75]

A multi-disciplinary panel consisting of emergency physicians, otolaryngologists, infectious disease experts, and primary care physicians have issued a set of guidelines based on evidentiary trials and other convenience, resistance, and patient toleration factors. (*See Table 3.*)

Other modalities useful in treating ABRS include nasal steroids, nasal saline, decongestants, and analgesics. Although most of these supportive therapies are unproven, these measures are inexpensive, uncomplicated, and do not have major side effects.[32] These work by eliminating causative factors and controlling inflammatory components. Steroids studied include beclomethasone diproprionate (*Beconase AQ, Beconase, Beclovent, Vancenase AQ, Vanceril*), mometasone furoate (*Elocon, Nasonex*), and fluticasone propionate (*Flovent, Flonase*).[76-81]

A randomized trial compared intranasal steroids to placebo and found that the addition of intranasal steroid to antibiotics significantly reduces symptoms of acute rhinosinusitis compared with antibiotic treatment alone.[76] Steroids also may be beneficial in prophylaxing against future episodes of rhinosinusitis by preventing impedance to mucus flow.[78] An interesting feature of macrolides is their anti-inflammatory properties, explaining a reason in addition to the antimicrobial spectrum, that they are successful in treating ABRS.[82,83] Decongestant nasal sprays (e.g., oxymetazoline [*Afrin*] and phenylephrine hydrochloride [*Neo-Synephrine*]) relieve acute symptoms by reducing mucosal edema. Their use should be limited to three days as they may cause rebound vasodilatation and inflammation (rhinitis medicamentosa). Oral decongestants such as pseudoephedrine also are effective, reasonable supportive alternatives. Mucolytic agents (e.g., guaifenesin) may facilitate mucociliary clearance by decreasing viscosity but there is no evidence that these may be useful. Saline spray or irrigation has a mild decongestant effect, liquifies viscous secretions, and is an important, often neglected adjunct to management.[80] (*See Table 4.*) Antihistamines should not be used routinely because they may dry nasal and sinus secretions. Even in allergic rhinitis, the traditional indication for antihistamine therapy, intranasal corticosteroids are the recommended first-line therapy.[84] Lastly, systemic analgesia, such as nonsteroidal anti-inflammatory agents, can provide relief from associated discomfort. Surgery also is an option for patients in more complicated cases of rhinosinusitis and requires referral to an ENT specialist. Surgery may help to facilitate drainage of the involved sinus and to remove diseased mucosa.

Table 3. Antibiotic Selection Guidelines Issued by ABRS Clinical Consensus Panel for Acute Bacterial Rhinosinusitis: Evidence-Based Antimicrobial Use in an Age of Emerging Drug Resistance

FIRST-LINE ANTIBIOTIC THERAPY[7]
Amoxicillin/clavulanate extended release 2000 mg/125 mg PO BID x 10 days[3]
(Alternative: amoxicillin/clavulanate 500 mg/125 mg PO TID x 10 days)
OR
Amoxicillin 875 mg PO BID x 10-14 days[4]
OR
Azithromycin 500 mg PO QD x 3 days
OR
Telithromycin 800 mg PO QD x 5 days

FIRST-LINE ALTERNATIVE ANTIBIOTIC THERAPY
Moxifloxacin[5,8] 400 mg PO QD x 10 days (preferred fluoroquinolone)
OR
Levofloxacin5 500 mg PO QD x 10-14 days
OR
Clarithromycin 500 mg PO BID x 14 days
OR
Doxycycline 100 mg PO BID x 10-14 days[6]

1 One or more severe symptoms present for less than 7 days which may prompt early antibiotic therapy may include the following: temperature > 102°; unilateral facial pain or pressure; bilateral facial pain, which may suggest pan-sinusitis; facial erythema; swelling over the sinus; maxillary teeth pain; and/or bimodal disease course.
2 Stronger consideration for initiating prompt antibiotic therapy should be given in the case of immunocompromised patients with symptoms of less than 7 days duration; clinical judgment should prevail in such cases, and earlier referral to ENT may be necessary.
3 Other beta-lactam antibiotics also may be considered, among them: cefpodoxime, cefuroxime, loracarbef, and ceftibuten.
4 Because of increasing resistance to amoxicillin among *S. pneumoniae* isolates from patients with bacterial respiratory tract infections, high-dose amoxicillin therapy is recommended for treatment of acute bacterial rhinosinusitis in adults. In addition, amoxicillin also is preferred as an initial agent when acquisition of the antibiotic may be compromised by cost considerations, resulting in medication noncompliance.
5 Fluoroquinolones are effective and safe agents for the treatment of acute bacterial rhinosinusitis, and produce similar outcomes when evaluated against comparator agents. However, recent practice guidelines for bacterial respiratory tract infections from the Infectious Disease Society of America (IDSA) and Centers for Disease Control and Prevention (CDC) note that effecting positive outcomes with potent, excessively broad-spectrum agents must be balanced against the pitfalls of inducing resistance to such agents, especially fluoroquinolones. In its Dec. 1, 2003, Practice Update Guidelines for community-acquired pneumonia (CAP), the IDSA committee expressed concern about misuse and over-use of fluoroquinolones, noting that if abuse of this class of drugs continues unabated, we may see the demise of fluoroquinolones as useful antibiotics within the next 5-10 years (*Clin Infect Dis*. 2003;37:1405-1433).
6 Doxycycline should be considered as an alternative agent when acquisition of the antibiotic may be compromised by cost considerations, resulting in medication noncompliance.
7 If a patient with presumed acute bacterial rhinosinusitis has received a previous course of antimicrobial therapy with either a beta-lactam (cefuroxime, amoxicillin, amoxicillin/clavulanate, etc.) or a macrolide within the past 3 months, excluding the current episode, a respiratory fluoroquinolone (i.e., moxifloxacin, levofloxacin) is recommended as the initial treatment. Conversely, recent use of a fluoroquinolone should dictate use of either an advanced generation macrolide (azithromycin or clarithromycin) or a beta-lactam (amoxicillin/clavulanate).
8 Among the advanced generation, respiratory fluoroquinolones, moxifloxacin is preferred because it has lower MICs against *S. pneumoniae* than levofloxacin, and because it has a more narrow (gram-positive organism-focused) spectrum of coverage.

Potential Complications

Acute bacterial rhinosinusitis can cause both intra- and extra-cranial complications such as meningitis, epidural or subdural empyema, cavernous sinus thrombosis, osteomyelitis, orbital cellulitis, or abscess.[85] Untreated or undertreated ABRS also can evolve into chronic rhinosinusitis, which carries significant morbidity.[29]

Patients treated both with and without antibiotics may suffer complications from ABRS. These range from infectious to anatomic. Because strict diagnostic criteria for ABRS are used so infrequently, it is difficult to quantify the rate of complications. One study found a 3.7% complication rate, but this was in patients hospitalized for rhinosinusitis, which was more common several years ago but is unusual today.[86] Patients who tend to suffer more complications, and thus usually would warrant otolaryngology referral, include those with immunocompromise, nasal polyps, or

Table 4. Adjunctive Therapy in ABRS[63]

THERAPEUTIC AGENT	COMMENT
Intranasal corticosteroids (mometasone, flunisolide)	Useful for rhinitis or associated bronchial hyperresponsiveness.
Oral steroids	No proven efficacy but consider if significant anatomic obstruction, invasive polyposis, or marked mucosal edema radiographically.
Nasal saline drops or spray	Helps to liquefy secretions and decreases crusting near the sinus ostia.
Topical decongestant (oxymetazoline and phenylephrine) Systemic decongestant (pseudoephedrine)	Reduces mucosal blood flow, decreases tissue edema and nasal resistance, and may enhance drainage of secretions from the sinus ostia.
Mucolytics	No conclusive evidence that these are useful.
Other comfort measures	Includes rest, adequate hydration, warm facial packs, steamy showers, sleeping with the head elevated, and analgesics as needed.

suggestion of chronic or repeated bacterial rhinosinusitis.[80]

One of the most common extra-nasal sites of infectious complications is the orbit, especially in the pediatric population. Untreated or inadequately treated rhinosinusitis often is the event leading to the development of orbital cellulitis in children. Other central nervous infectious complications, such as brain abscess or meningitis, are less common in the era of high antibiotic utilization, but still account for half of all infectious complications of ABRS.[5] Infection may gain access to the intracranial space by direct extension through a defect in the posterior wall of the frontal sinus caused by the infection itself. Retrograde thrombophlebitis of the valveless ophthalmic vessels also may offer a route of transmission for infected material into the intracranial cavity.[85] While antibiotics may have some impact on infectious complications, a review found that their use does not decrease the overall complication rate.[87] Osteomyelitis can be diagnosed with the use of head CT or culture/biopsy of paranasal bones. Pott's puffy tumor is osteomyelitis of the frontal bone, which causes a subperiosteal abscess. This usually is caused by *S. aureus* and its incidence also has dropped with the use of antibiotics.[5]

Anatomic complications from ABRS result from the changes that occur as the inflammation and tissue destruction change the structure and composition of the paranasal sinuses and surrounding area. The floor of the frontal sinus sits above the orbit, so disruption of the architecture predisposes to superior ophthalmic vein, dural vein, or cavernous sinus thrombosis or thrombophlebitis. Inflammation and scarring can contribute to the development of a mucocele, an expanded mucoid-filled sinus, or mucus retention cyst.

Complications of ABRS are not limited to individual instances, but can be expanded to include the impact on society that antibiotic overuse presents. The pharmacologic cost consideration is apparent, but increasing antibiotic resistance raises costs by the need for stronger, more expensive drugs and more resistant infections—including not only rhinosinusitis but also other infections. Penicillin-resistant *S. pneumoniae* is an increasing problem of variable magnitude in different communities, but will not retreat without extremely judicious use of narrow-spectrum drugs.

Disposition

The vast majority of patients with ABRS are treated successfully on an outpatient basis. The natural history of uncomplicated ABRS is resolution of symptoms in 4-10 days.[88] It is difficult to prove that antibiotics change the clinical course for many reasons, including: 1) many people presumptively diagnosed with ABRS in reality have viral or allergic rhinosinusitis; 2) antibiotics often used to treat ABRS are effective against some but not all pathogens and isolates; and, thus 3) studies looking at efficacy are in fact diluted by many cases that would not respond to antibiotics regardless.

A proportion of patients do not respond to conventional therapy, including antibiotics and adjuncts. After a confirmation of the diagnosis with CT scan, these patients usually are referred to otolaryngologists for further care. One option is endoscopic sinus surgery. This procedure removes nasal polyps and thus the anatomic risk factor for mucous flow impedance. This newer procedure improves sinus drainage instead of just removing diseased mucosa.[89] Another procedure, called the endoscopic modified Lothrop procedure, promotes patency in the frontal ostium. Symptomatic relief occurs in more than 90% of patients.[90] Most patients who do not respond adequately to medical therapy eventually receive some sort of endoscopic sinus surgery.[91]

Special Populations

Immunocompromised patients, diabetics, those with cystic fibrosis, and children deserve special attention. The immunodeficient patient should be managed in consultation with an otolaryngologist, as sinus aspirate culture should be considered early in the course to guide therapy.

Cystic fibrosis patients are more prone to sinus infections by virtue of the ciliary defect. *Staphylococcus aureus* and Pseudomonas are particularly common causes of recurrent infection and should be treated accordingly with the appropriate antibiotic therapy.[92] A culture diagnosis should be considered, owing to the frequent need for long-term antibiotics and chronicity of infections.

Although rhinosinusitis generally is more common in children than adults, frontal and sphenoid rhinosinusitis are rare in children. In the primary care setting, between 6% and 18% of children presenting with upper respiratory infections may have acute bacterial rhinosinusitis.[11] Children are infected more frequently with *M. catarrhalis* than adults, and this should direct therapy

effective against this pathogen. The American Academy of Pediatrics recommends a clinical diagnosis alone in children 6 years and younger. These symptoms should last more than 10 days but fewer than 30 days and include a fever of 39°C or higher, nasal or postnasal discharge, a daytime cough, and purulent nasal discharge present for at least 3-4 consecutive days.[93] Adjuvant therapies are not recommended except for saline spray. Imaging is recommended only for complications or persistent symptoms. First-line therapy is high-dose amoxicillin (90 mg/kg/day in two divided doses). Amoxicillin/clavulanate or cefuroxime is recommended in cases that do not improve with amoxicillin. The Sinus and Allergy Health Partnership added cefpodoxime to the guidelines for treatment, and recommend a macrolide for patients who cannot tolerate penicillins due to allergy.[66]

Summary

Most patients with ABRS typically present with a multitude of symptoms, including headache, unilateral face or tooth pain, fever, and purulent nasal drainage. Although the gold standard for diagnosis includes sinus CT scan and culture of sinus aspirates, this is needed only in cases of diagnostic dilemma, lack of response to treatment, or complication. Empiric antibiotic therapy consists of amoxicillin, but only after symptoms have persisted at least seven days—making the diagnosis of viral rhinosinusitis less likely. Adjuvant treatment to antibiotics includes nasal decongestants, analgesics, intranasal saline, and steroid sprays. Referral to an otolaryngologist should be made for treatment failures, complications, or in special populations at high risk for the aforementioned. Surgical techniques can treat and prevent complications. Antibiotic overuse has become a problem in large part due to the massive abuse of antibiotics for simple upper respiratory infections incorrectly diagnosed as ABRS.

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Physician CME Questions

11. Which of the following is one of the most common infectious complication of acute sinusitis in children?
    A. Intracranial abscess/meningitis
    B. Pott's puffy tumor (osteomyelitis)
    C. Cavernous sinus thrombosis
    D. Orbital cellulitis

12. Which of the following is considered a first-line antibiotic agent for acute sinusitis?
    A. Amoxicillin
    B. Cephalosporins
    C. Clindamycin
    D. Clarithromycin

13. Which of the following is a major criteria for ABS?
    A. Dental pain
    B. Purulent nasal discharge
    C. Halitosis
    D. Headache

14. Which of these adjunctive treatments is recommended in uncomplicated ABRS?
    A. Antihistamines
    B. Mucolytics
    C. Decongestants
    D. Debridement of nasal mucosal

15. Disturbance of normal sinus flora is a cause of ABRS.
    A. True
    B. False

16. Consensus statements and systematic reviews suggest that patients with rhinosinusitis symptoms lasting fewer than 7 days are unlikely to have a bacterial infection
    A. True
    B. False

17. If a patient clinically diagnosed with acute sinusitis fails initial treatment with 3 days of amoxicillin, what is the next step?
    A. Refer to an otolaryngologist
    B. Order a sinus CT
    C. Switch to a different antibiotic
    D. Perform sinus puncture and culture

18. Which of the following is *not* part of the criteria for clinical diagnosis of acute sinusitis in children?
    A. Symptoms lasting fewer than 30 days
    B. Frontal headache
    C. Fever of 39°C or more
    D. Purulent nasal discharge present for more than 3-4 days

19. Which of the following is true regarding acute bacterial sinusitis?
    A. ABS most frequently is a complication of viral or allergic rhinosinusitis.
    B. Rhinitis is not a common finding.
    C. The finding of maxillary sinus thickening on CT scan is always diagnostic.
    D. *S. aureus* is the most common pathogen.

20. Plain film x-rays:
    A. are the gold standard of imaging diagnosis.
    B. are not sufficient to evaluate drainage patterns in all sinuses.
    C. have the advantage of sphenoid sinus imaging.
    D. are superior for the diagnosis of anterior ethmoid involvement.

CME Answer Key

11. D	16. A
12. A	17. C
13. B	18. B
14. C	19. A
15. B	20. B

Emergency Medicine Reports
The Practical Journal for Emergency Physicians

Acute Bacterial Rhinosinusitis

Antimicrobial Therapy for ABRS

Length of therapy will vary depending on FDA-approved course of therapy for specific antibiotics, with length of therapy varying between 3 days and 14 days.

ANTIBIOTIC	DOSE
Adults	
First-line	
• Amoxicillin (Amoxil, Trimox, Wymox)*	500-1000 mg tid or 875 mg bid
• TMP/SMX (Bactrim, Septra)**	160 mg/800 mg (double-strength) bid
Second-line	
• Amoxicillin-clavulanate (Augmentin)*	500 mg tid or 875 mg bid
• Cefpodoxime proxetil (Vantin)	200-400 mg bid
• Cefprozil (Cefzil)	250-500 mg bid
• Cefuroxime axetil (Ceftin)	250-500 mg bid
• Cefdinir (Omnicef)	300 mg bid
• Clarithromycin (Biaxin)	500 mg bid
• Levofloxacin (Levaquin)	500 mg qd
• Gatifloxacin (Tequin)	400 mg qd
• Moxifloxacin (Avelox)	400 mg qd
Children	
First-line	
• Amoxicillin*	45 or 90 mg/kg/d in two divided doses
Second-line	
• Amoxicillin-clavulanate*	45 or 90 mg/kg/d in two divided doses
• Cefuroxime axetil	30 mg/kg/d in two divided doses
• Cefpodoxime proxetil	10 mg/kg qd
• Ceftriaxone (Rocephin)	50 mg/kg/d intravenously
If penicillin-allergic	
• Azithromycin (Zithromax)	10 mg/kg first day then 5 mg/kg qd
• Clarithromycin	15 mg/kg/d in two divided doses

*Use higher doses of amoxicillin if *S. pneumoniae* resistance suspected.
**Chosen less often due to increasing bacterial resistance and the rare but serious complications.

Antibiotic Selection Guidelines Issued by ABRS Clinical Consensus Panel for Acute Bacterial Rhinosinusitis: Evidence-Based Antimicrobial Use in an Age of Emerging Drug Resistance

FIRST-LINE ANTIBIOTIC THERAPY[7]

Amoxicillin/clavulanate extended release 2000 mg/125 mg PO BID x 10 days[3]
(Alternative: amoxicillin/clavulanate 500 mg/125 mg PO TID x 10 days)
OR
Amoxicillin 875 mg PO BID x 10-14 days[4]
OR
Azithromycin 500 mg PO QD x 3 days
OR
Telithromycin 800 mg PO QD x 5 days

FIRST-LINE ALTERNATIVE ANTIBIOTIC THERAPY

Moxifloxacin[5,8] 400 mg PO QD x 10 days (preferred fluoroquinolone)
OR
Levofloxacin5 500 mg PO QD x 10-14 days
OR
Clarithromycin 500 mg PO BID x 14 days
OR
Doxycycline 100 mg PO BID x 10-14 days[6]

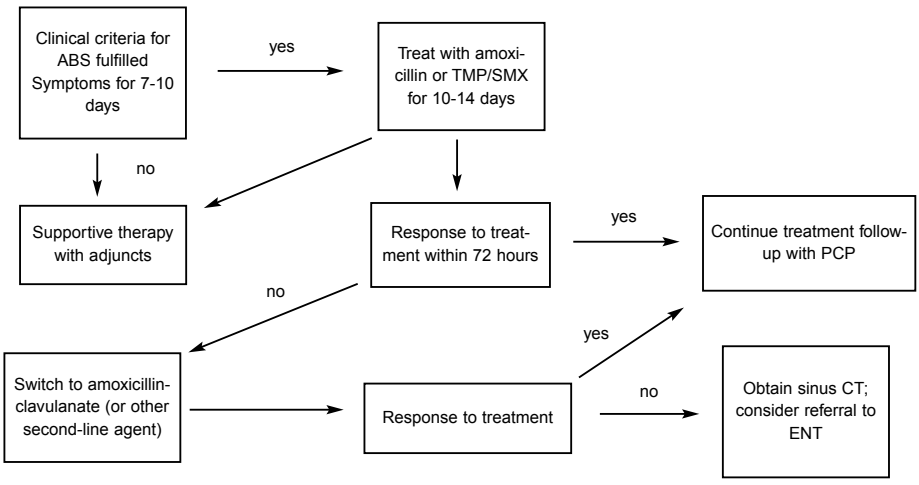
1 One or more severe symptoms present for less than 7 days which may prompt early antibiotic therapy may include the following: temperature > 102°; unilateral facial pain or pressure; bilateral facial pain, which may suggest pan-sinusitis; facial erythema; swelling over the sinus; maxillary teeth pain; and/or bimodal disease course.
2 Stronger consideration for initiating prompt antibiotic therapy should be given in the case of immunocompromised patients with symptoms of less than 7 days duration; clinical judgment should prevail in such cases, and earlier referral to ENT may be necessary.
3 Other beta-lactam antibiotics also may be considered, among them: cefpodoxime, cefuroxime, loracarbef, and ceftibuten.
4 Because of increasing resistance to amoxicillin among *S. pneumoniae* isolates from patients with bacterial respiratory tract infections, high-dose amoxicillin therapy is recommended for treatment of acute bacterial rhinosinusitis in adults. In addition, amoxicillin also is preferred as an initial agent when acquisition of the antibiotic may be compromised by cost considerations, resulting in medication noncompliance.
5 Fluoroquinolones are effective and safe agents for the treatment of acute bacterial rhinosinusitis, and produce similar outcomes when evaluated against comparator agents. However, recent practice guidelines for bacterial respiratory tract infections from the Infectious Disease Society of America (IDSA) and Centers for Disease Control and Prevention (CDC) note that effecting positive outcomes with potent, excessively broad-spectrum agents must be balanced against the pitfalls of inducing resistance to such agents, especially fluoroquinolones. In its Dec. 1, 2003, Practice Update Guidelines for community-acquired pneumonia (CAP), the IDSA committee expressed concern about misuse and over-use of fluoroquinolones, noting that if abuse of this class of drugs continues unabated, we may see the demise of fluoroquinolones as useful antibiotics within the next 5-10 years (*Clin Infect Dis*. 2003;37:1405-1433).
6 Doxycycline should be considered as an alternative agent when acquisition of the antibiotic may be compromised by cost considerations, resulting in medication noncompliance.
7 If a patient with presumed acute bacterial rhinosinusitis has received a previous course of antimicrobial therapy with either a beta-lactam (cefuroxime, amoxicillin, amoxicillin/clavulanate, etc.) or a macrolide within the past 3 months, excluding the current episode, a respiratory fluoroquinolone (i.e., moxifloxacin, levofloxacin) is recommended as the initial treatment. Conversely, recent use of a fluoroquinolone should dictate use of either an advanced generation macrolide (azithromycin or clarithromycin) or a beta-lactam (amoxicillin/clavulanate).
8 Among the advanced generation, respiratory fluoroquinolones, moxifloxacin is preferred because it has lower MICs against *S. pneumoniae* than levofloxacin, and because it has a more narrow (gram-positive organism-focused) spectrum of coverage.

Predisposing Events for Sinusitis

- Prior upper respiratory tract infection
- Concurrent group A streptococcal infection
- Allergic rhinitis
- Anatomic variation (adenoidal hypertrophy, deviated septum, cleft palate, nasal polyps)
- Barotrauma from swimming
- Dental infection
- Exposure to irritants such as smoke
- Iatrogenic factors (nasogastric tubes, mechanical ventilation, nasal packing, dental procedures)
- Secretory disturbance (cystic fibrosis)
- Abnormalities in mucociliary clearance
- Hormonal changes (pregnancy)
- Medication side effect (abuse of topical vasoconstrictors or cocaine)
- Immunodeficiency, including diabetes mellitus

Adapted from: Brook I. Medical management of acute bacterial sinusitis, recommendations of a clinical advisory committee on pediatric and adult sinusitis. *Ann Otol Rhinol Laryngol* 2000;109 (suppl 182):2-20.

Algorithm for Treatment in Acute Bacterial Sinusitis (ABS)



Diagnostic Criteria for Acute Bacterial Rhinosinusitis (ABRS)

Diagnosis of ABRS made with two or more major criteria, or one major and two or more minor criteria.

MAJOR CRITERIA	MINOR CRITERIA
• Facial pain or pressure	• Headache
• Facial congestion or fullness	• Dental pain
• Fever	• Cough
• Nasal purulent discharge or discolored postnasal drainage	• Fatigue
• Nasal obstruction or blockage	• Ear pain, pressure, or fullness
• Hyposmia or anosmia	• Halitosis
• Purulence in nasal cavity on examination	

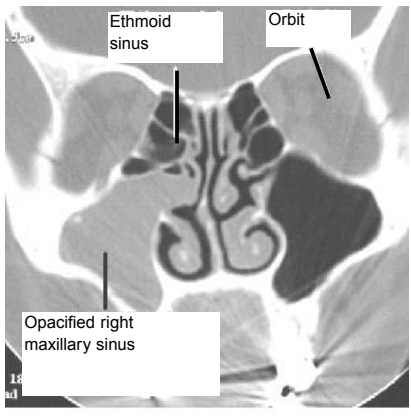
*Facial pain/pressure alone does not constitute a suggestive history for ABRS in the absence of another major nasal sign or symptom.
**ABS is likely when symptoms worsen after five days or persist for more than 10 days

Adapted from: Lanza DC, Kennedy DW. Adult rhinosinusitis defined. *Otolaryngol Head Neck Surg* 1997;117(3 Pt 2):S1-7. Osguthorpe JD. Adult rhinosinusitis: Diagnosis and Management. *Am Fam Physician* 2001;63:69-76.

Adjunctive Therapy in ABRS

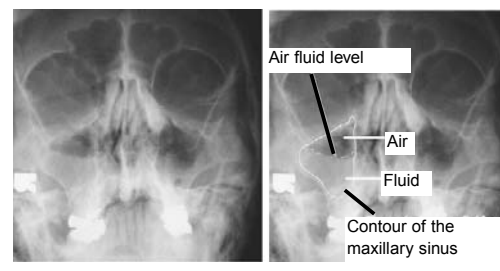
THERAPEUTIC AGENT	COMMENT
Intranasal corticosteroids (mometasone, flunisolide)	Useful for rhinitis or associated bronchial hyperresponsiveness.
Oral steroids	No proven efficacy but consider if significant anatomic obstruction, invasive polyposis, or marked mucosal edema radiographically.
Nasal saline drops or spray	Helps to liquefy secretions and decreases crusting near the sinus ostia.
Topical decongestant (oxymetazoline and phenylephrine) Systemic decongestant (pseudoephedrine)	Reduces mucosal blood flow, decreases tissue edema and nasal resistance, and may enhance drainage of secretions from the sinus ostia.
Mucolytics	No conclusive evidence that these are useful.
Other comfort measures	Includes rest, adequate hydration, warm facial packs, steamy showers, sleeping with the head elevated, and analgesics as needed.

Coronal CT Scan Showing Right Maxillary Sinus Opacification



Note the septal deviation to the right and the hypertrophy of the left inferior turbinate.
Used with permission from: Bechara Y. Ghorayeb, MD, www.ghorayeb.com/imagingmaxillarysinusitis.html.

Waters' View of the Sinuses Showing Partial Opacification of the Right Maxillary Sinus with an Air-Fluid Level



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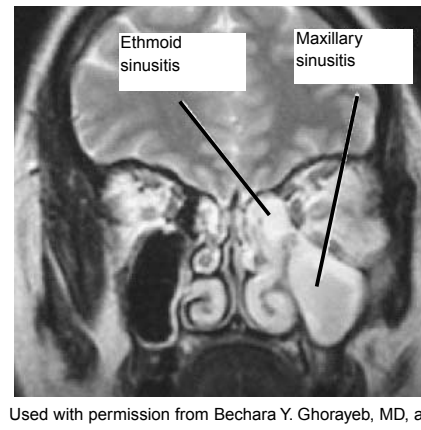
Coronal MRI Scan Showing Opacification of the Left Maxillary and Ethmoid Sinuses



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Supplement to *Emergency Medicine Reports*, January 10, 2005: Acute Bacterial Rhinosinusitis: Evidence-Based Management and Optimizing Antibiotic Therapy." *Authors:* **Lynn P. Roppolo, MD**, Assistant Professor, Division of Emergency Medicine, University of Texas Southwestern, Parkland Memorial Hospital, Dallas; and **Riva L. Rahl, MD**, Division of Emergency Medicine, University of Texas Southwestern Medical Center, Parkland Memorial Hospital, Dallas.

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Eye injuries present a significant challenge to emergency personnel. Patient stress and coexisting periorbital findings can complicate any evaluation, and many of the signs of serious injury may be quite subtle. Because the majority of eye injuries present between 10 p.m. and 4 a.m.,[1] when ophthalmology consultation is not available immediately in most hospitals, a tremendous burden is placed on the emergency health care provider to identify and manage potential vision-threatening disorders. The following is a review of ocular trauma with a focus on clinical findings, their implications, and management.

— The Editor

Initial Evaluation of the Traumatized Eye

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Epidemiology and Introduction

More than 2.5 million eye injuries occur in the United States annually.[2] From 1988- 2000, the United States Eye Injury Registry (USEIR) reported more than 10,000 major eye injuries,[3] an annual hospitalized incidence of 13.2 per 100,000.[4,5] Eighty percent of those injured were male, with a mean age of 29 years. Traditionally, the workplace presented the most common site of ocular injuries, but the USEIR reports 40% of these injuries occurring in the home, 13% in the workplace, and another 13% during recreation. Of these injuries, 31% were caused by a blunt object (e.g., rock, fist, baseball, or lumber), 18% by a sharp object, and 12 % were attributed to projectiles such as firearms and pellet (BB) guns.[6-8] Alcohol consumption was involved in 10% of cases, and 57% of all eye injuries occurred duirng warm weather months (spring and summer).[9] In the United States, 1.7% of all eye injuries will progress to permanent visual loss,[9] resulting in 60,000 new cases of monocular blindness related to trauma annually.[10]

Among the pediatric population (birth–16 years of age), eye injuries occur at an annual rate of 15.2 per 100,000 with males outnumbering females four to one. Males, ages 11-15, have the highest incidence of eye injuries at 23.7 per 100,000 annually, and 40% of these injuries are attributed to sports.[11] In victims of child abuse, an ocular injury will be the presenting injury in up to 40% of all cases[12] and in 95% of cases of shaken baby syndrome.[13]

Physical Exam

Dannenberg et al reported that 33% of all registered penetrating eye injuries were in the setting of multisystem trauma.[6] Thus, Advanced Trauma Life Support (ATLS) guidelines and a thorough general examination must precede any ocular evaluation. Secondly, any eye with a potential exposure to hazardous materials (e.g., acids, alkali, particulate matter, or heat) should be irri-

gated prior to evaluation. Whenever possible, a thorough history should be obtained detailing the context, mechanism, time of injury, and use of eye protection. Past medical history should include previous ophthalmologic conditions, surgeries and trauma, current ocular medications, and pre-injury vision status (e.g., glasses/ contact lenses wearer, and visual acuity).

Figure 1 demonstrates a simple guide for the trauma examination of the eye. The first step is an assessment of visual acuity, which can be accomplished with a Snellen Eye Chart or near card. For pre-literate children, Allen cards or HOTV vision test letters may be used. For infants, fixation and smooth pursuit can be assessed using a hand light or colorful target. In many cases, the visual acuity may be too poor to assess by standard charting, and thus a gross assessment of visual acuity should be obtained using finger counting, motion perception, and light perception.

Most patients, regardless of mechanism, will complain of some degree of eye pain, but care must be taken to differentiate between globular and periorbital sources of pain. On inspection, examine the orbit for any step-off deformities or crepitus that may reveal an orbital wall fracture. Although there may be evidence of significant periorbital ecchymosis and edema, the lids still should be examined closely for lacerations and inverted when possible to remove retained foreign bodies. In the setting of prominent periorbital edema, a speculum or retractor may be employed to visualize the globe; however, if there is any evidence of a globe rupture (e.g., prolapse of intraocular contents, hemorrhagic chemosis, or enophthalmos), then this step should be foregone in favor of imaging studies and immediate ophthalmologic consultation. When possible, examine the anterior chamber for hyphema or pupil irregularities, findings that require further examination by slit lamp. Globe pain or foreign body sensation imply, at the minimum, corneal irritation and can be evaluated using fluorescein dye with a Wood's lamp to reveal corneal abrasions, ulcerations, or lacerations. All corneal exams that require fluorescein should be followed by a slit-lamp examination for occult foreign bodies.

Ocular motility should be assessed in both the vertical and horizontal planes. Classically, orbital wall fractures will present with deficits of ocular motility, but a proptotic eye with any motion deficit is the hallmark of a retrobulbar hemorrhage. For this reason, any defects in ocular motion should be evaluated further with a computerized tomography (CT) scan.

Finally, the posterior segment should be assessed by pupil reactivity to direct light and accommodation. Even in cases of prominent trauma to the anterior segment, the retina and optic nerve can be assessed by direct light to the affected eye and monitoring accommodation in the unaffected eye, known as the swinging flashlight test. A pupil that fails to constrict with direct light stimulation, but responds to stimulation of the other eye implies a defect in the posterior segment. Children may have a corresponding lack of a red reflex (Bruckner's test) in the affected eye. Both should be investigated further with an ophthalmoscope, when possible, or imaging studies such as ultrasound or CT scan.

In theory, any patient with a painless eye with intact visual acuity, a normally reactive pupil, and intact ocular motility in the absence of any anatomic deformities following a traumatic event may be discharged without further study and appropriate follow up. Nevertheless, an examining physician should have a low threshold for advanced study in the setting of high energy trauma or any projectiles where these findings may be masked by either trauma elsewhere or the delicate size of the particulate matter. Any new visual deficits or any impairment in mental status that complicates the examination should prompt a full evaluation.[10,14-17]

Classification of Eye Trauma

In 1993, a group of ophthalmologists from Birmingham, Ala., developed a standardized classification of eye trauma that would ease communication between peers and provide prognostic significance. In 1995, the International Society of Ocular Trauma, The USEIR, and the American Academy of Ophthalmology endorsed this new system. Ocular Trauma Classification (Tables 1 and 2) involves identifying the type of injury (open vs closed globe), the grade of the injury (visual acuity), pupil response, and

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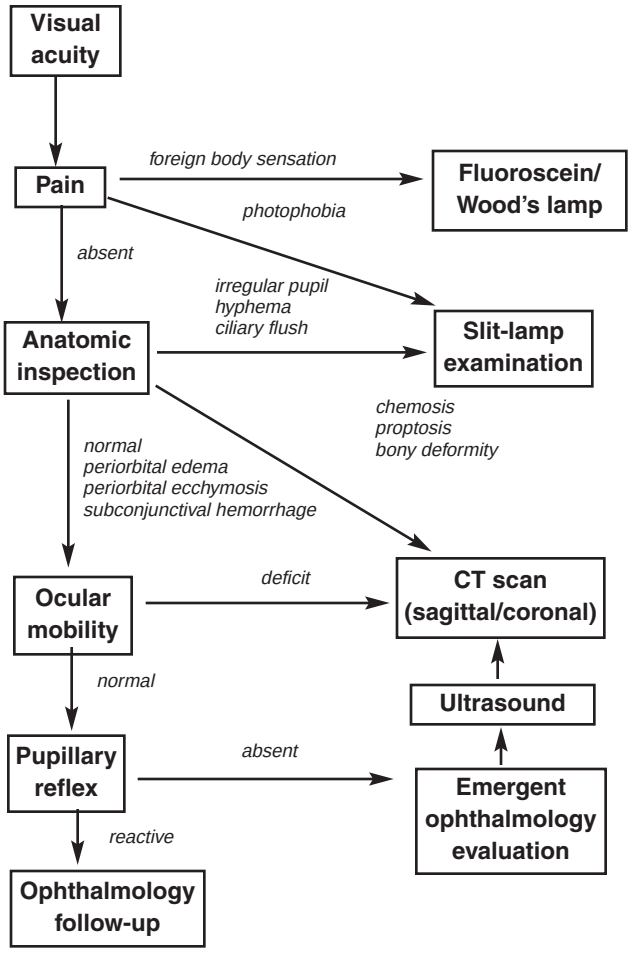
Figure 1. Trauma Examination of the Eye



the zone of the injury. Zone defines the anterior-posterior relationship of the injury on the globe.

An *open-globe injury* contains a full thickness wound of the eyewall (sclera or cornea). Open-globe injuries are then divided into lacerations (sharp mechanism) and ruptures (blunt mechanism). Lacerations are further divided into penetrating injuries (i.e., one entrance wound only), perforating injuries (i.e., two full thickness entrance and exit wounds), and intraocular foreign bodies. A *closed-globe injury* has no full thickness wound of the eyewall, and these injuries are divided into contusion (blunt mechanism), lamellar laceration (sharp mechanism), and superficial foreign body categories.

The Ocular Trauma Classification System provides a universal means of communicating the type and severity of an eye injury to an ophthalmology consultant or between emergency personal. In addition to quantifying an injury, this system, within the ophthalmology community, provides a foundation for management requirements as well as prognostic value for regaining vision.[18-21]

Superficial Injuries of the Eye

Superficial injuries of the eye, such as periorbital edema and ecchymosis, are very common sequelae of eye trauma, and often are quite dramatic on initial presentation. Despite the stress they may cause patients, these injuries involving the eyelids are quite benign in the absence of any globe injury and can be managed conservatively. For this reason, evaluation of lid injuries, regardless of mechanism, should be delayed for the evaluation and management of intraocular injuries.

Eyelid lacerations are divided into three types: vertical, horizontal, and canilicular (involving the medial lacrimal duct). Essentially, all eyelid laceration repairs may be delayed safely for 24-48 hours.[22-23] Thus, it is recommended that these injuries be irrigated to remove any foreign bodies, covered with a semi-moist pressure dressing, and ophthalmology evaluation arranged within one day. This is acceptable management for all pediatric and uncooperative patients as well.

Partial thickness injuries may be closed by emergency personnel, especially if ophthalmology evaluation will be delayed more than two days. However, all canalicular lacerations should be left to a specialist; the most superficial lacerations require proper exploration prior to closure.

Horizontal lacerations are more serious than vertical lacerations due to potentially significant impairment of the levator muscle. Evidence of ptosis or prolapsing fat indicates violation of the orbital septum, and, due to the close proximity of the levator aponeurosis, is highly suggestive of a levator injury. These repairs also should be delayed for a specialist.

Partial or full thickness vertical lacerations can be repaired by first approximating the tarsoconjunctival edge with simple, interrupted polyglycolic acid suture material with a D-1 needle.[22,24] The muscle layer can be sutured with 6-0 catgut, and the external eyelid can be closed with 6-0 silk. Once sutured, a pressure patch should be placed to reduce eye swelling and horizontal tension on the eyelid.[17] Although suture removal is recommended in 7-10 days, an ophthalmology follow-up evaluation should be scheduled in 2-3 days to ensure adequate wound healing.

Injury by Presentation: Pain

Pain is the hallmark of a corneal injury. Corneal abrasions are the single most common ocular condition evaluated in the ED,[25] as well as the most common eye injury related to airbag deployment.[26] For injuries confined to the superficial corneal epithelium, patients will present with a painful eye with conjunctival injection, ciliary flush, and tearing. Prior to examination, the eye should be anesthetized with tetracaine or proparacaine. Findings on fluorescein dye examination of the eye under a cobalt blue light or Wood's lamp will vary depending on the mechanism of the injury. Large abrasions with sharp borders are most often due to blunt trauma. Diffuse, punctuate lesions represent mild burn injuries from chemical exposure or ultraviolet light. Foreign bodies under the eyelid will produce multiple linear defects. Treatment includes removal of any foreign bodies, broad-spectrum antibiotics for the eye (e.g., ciprofloxacin/polymyxin B-trimethoprim), oral analgesia such as non-steriodal anti-inflammatory drugs (NSAIDS) or acetaminophen, tetanus prophylaxis as needed, and ophthalmology follow-up in 24-48 hours.[20]

Burns. Corneal burns account for up to 18% of all ocular trauma, with 68% of these injuries occurring in an occupational setting.[27] Burning agents can be divided into three broad cate-

gories (alkaline chemical agents, acidic chemical agents, and thermal agents), and the severity of the injury is related to the composition of the offending agent, volume, pH, and duration of exposure. Alkaline agents, accounting for 58% of all burn injuries, damage the cornea by saponifying the fatty acid components of the corneal cell membranes. This induces a liquefactive necrosis and allows deeper penetration into the globe. Ammonia is the most common alkaline agent causing eye injuries, but others include lye, potassium hydroxide, and lime.

Acids damage the cornea by inducing protein precipitation and denaturation into the epithelium and superficial stroma. The resultant coagulative necrosis tends to protect deeper intraocular structures. The most common acid-burning agents include sulfuric, sulfurous, hydrofluoric, and hydrochloric acids.[22]

Thermal injuries are usually the result of splash injuries from hot liquids or molten metal. A temperature greater than 100° C is required to injure the corneal epithelium. Cigarette burns to the cornea are a common injury in children ages 2-4 years. Usually the result of a child running into a cigarette at eye level; this burn pattern is rarely a manifestation of abuse.[10]

Eye burns are classified into four grades. A Grade I injury presents with hyperemia, conjuntival ecchymosis, and a cornea with a ground-glass appearance. A cigarette burn will produce this pattern of injury. In addition, a Grade II injury will have conjunctival chemosis and minor eschar formation. Grade III burns are deeper injuries characterized by conjunctival ischemia, thrombosed blood vessels, and reduced corneal clarity with preservation of the remaining anterior segment. Complete opacification of the cornea and lens, in addition to diffuse conjunctival ischemia and a grey iris in mydriasis, typify a Grade IV injury. Necrosis of the conjunctiva is accompanied by a dramatic inflammatory response that will induce further corneal ulceration within 4-6 weeks.

The mainstay of burn therapy is irrigation, preferably with a solution that is isotonic to the corneal stroma. Ideal solutions include lactated Ringers, balanced saline solution, or diphoterine; however, any irrigation (water, or normal saline) is far better than none at all. Upon presentation, patients should be anesthetized topically and then irrigated promptly for 15 minutes or 1L of fluid using a Morgan lens. Irrigation should not stop until normalization of the corneal pH (7.4). Lime and cement products will react with water to produce calcium hydroxide (pH 12.4), exacerbating their effect. These patients will require aggressive cleaning of the cul-de-sac with a cotton-tipped applicator dipped in 1% ethylenedinitrilotetraacetic acid (EDTA).[22]

Grade I and II burns may be discharged home on topical antibiotics and oral analgesics with ophthalmology follow-up within 24 hours. Grade III burns require admission for operative microscopy to determine the depth of tissue destruction. Grade IV lesions require steroids (oral or IV), as well as topical antibiotics prior to admission. Grade III lesions may be admitted to a local hospital with ophthalmology services; however, a Grade IV burn will most likely require a tertiary center with expertise in plastic reconstruction of the eye.[22]

Table 1. Open-Globe Injury Classification

TYPE

- Rupture
- Penetrating
- Intraocular foreign body
- Perforating
- Mixed

GRADE (VISUAL ACUITY)

- > 20/40
- 20/50 to 20/100
- 19/100 to 5/200
- 4/200 to light perception (LP)
- No light perception (NLP)

PUPIL

- *Negative*: Relative afferent pupillary defect absent in affected eye
- *Positive*: Relative afferent pupillary defect present in affected eye

ZONE

- I: Isolated to cornea (including corneoscleral limbus)
- II: Corneoscleral limbus to 5 mm posterior into the sclera
- III: Posterior to the anterior 5 mm of sclera

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Ultraviolet Keratitis. Ultraviolet keratitis is another form of exposure injury to the cornea classically known as snow blindness. Caused by the cumulative effect of ultraviolet light from electric arcs (welding) or tanning lamps, this disorder initially will present as a bilateral foreign body sensation and photophobia. Symptoms progress to severe bilateral eye pain with conjunctival erythema and tearing. Fluorescein staining reveals diffuse punctuate lesions on the cornea. Symptoms usually will resolve in 24-36 hours once removed from the inciting agent. Management includes topical antibiotics and narcotic analgesia.[20]

Lacerations. A corneal laceration is defined as a traumatic disruption of the cornea involving all three layers (epithelium, stroma, and endothelium) due to a penetrating mechanism; 75% of these injuries in children were caused by a sharp object.[28] Like other corneal injuries, pain is the most common presenting complaint. An examination of the eye may reveal a shallow anterior chamber or teardrop-shaped pupil directed toward the laceration. Prolapse of intraocular contents is a confirmatory finding; however, in many cases, the eye will appear normal due to the self-sealing properties of the elastic corneal stroma. Fluorescein evaluation of the cornea under blue light will reveal a bright green stream (Seidel test) caused by egress of fluid from the anterior chamber. A slit-lamp examination on oblique illumination may reveal the length and depth of a laceration. Nevertheless, a normal fluorescein study does not completely eliminate the possibil-

Table 2. Closed-Globe Injury Classification

TYPE

- Contusion
- Lamellar laceration
- Superficial foreign body
- Mixed

GRADE (VISUAL ACUITY)

- > 20/40
- 20/50 to 20/100
- 19/100 to 5/200
- 4/200 to light perception (LP)
- No light perception (NLP)

PUPIL

- *Negative:* Relative afferent pupillary defect absent in affected eye
- *Positive:* Relative afferent pupillary defect present in affected eye

ZONE

- I: External (limited to bulbar conjunctiva, sclera, and cornea)
- II: Anterior segment
- III: Posterior segment

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ity of a corneal laceration. Partial-thickness lacerations can be managed like corneal abrasions.[20,29]

Foreign Bodies. Forty-one percent of all open-globe injuries involve an intraocular foreign body.[30] The majority of these projectiles (80%) are generated in the setting of hammering either a chisel, nail, or stone.[30,31] Other causes of ocular foreign bodies include motor vehicle collisions, explosions, projectile weapons, and activities involving power tools such as drilling. Magnetic metals (iron, lead, and copper) account for the majority of intraocular foreign bodies (IOFB); however, nonmetallic items such as glass, wood, and especially organic materials in pastoral settings, may be a nidus for a disastrous Bacillus infection. More than 80% of IOFBs will penetrate the cornea and settle within the vitreous.[32] Other common sites for foreign bodies include the underside of the upper lid, the infracorneal recess within the lid fissure, and the inferior angle of the anterior chamber.[33]

Missed foreign bodies constitute 56% of all eye trauma-related legal claims, and therefore, IOFBs should be excluded in any patient with a suspicious mechanism. One-fifth of all patients with ocular foreign bodies will have no pain and intact visual acuity.[30] Many patients, especially children, will be unaware of the exposure, resulting in a significant delay to presentation. Like other corneal injuries, pain or foreign body sensation will be the most common complaint, but visual deficits will vary depending on the size of the projectile, extent of the injury, and delay in presentation. Fluorescein examination of the cornea should be followed by a slit lamp examination of the cornea, anterior chamber, and the area under the lids. Any superficial foreign body may be removed with either a cotton-tipped applicator (lid) or a 27-gauge needle.[34]

Assault-related Injuries. Dannenberg et al reported that one-third of all occupational eye injuries[11] and 56% of all assault-related injuries involve the sclera.[5] Scleral rupture is a traumatic disruption of the sclera commonly in the supranasal quadrant at the insertions of the rectus muscles on the globe. Most often this injury is the result of a blunt mechanism and has a prevalence of 3.5% in blunt trauma.[35] In the absence of obvious prolapse of intraocular contents, evidence of a scleral rupture may be quite subtle. Low intraocular pressures (< 6 mmHg), reduced visual acuity on initial presentation, an afferent pupillary defect, and a shallow anterior chamber are clinical findings highly specific to scleral ruptures, and thus, warrant further investigation. In addition, one-quarter of hyphemas and 22% of bloody chemosis cases are associated with a full-thickness scleral injury. [12,30,36-38]

Because full thickness injuries of the eyewall (e.g., corneal lacerations, scleral rupture, and intraocular foreign bodies) are open-globe injuries, any suspicion of such requires prompt ophthalmology evaluation. CT imaging (axial and coronal views) may be diagnostic (sensitivity, 93%; specificity, 75%; PPV 95%)[39,40] of scleral injuries, but is the modality of choice to detect intraocular foreign bodies (sensitivity 100% for objects more than 0.06 mm).[3,41] Magnetic resonance imaging (MRI) is contraindicated for IOFB due to migration of metallic fragments.

The examiner should avoid placing any pressure on a suspected globe rupture to limit further prolapse of intraocular content; therefore, tonometry should be deferred despite the value of confirming low intraocular pressures. An eye shield should be placed promptly pending further evaluation. Tetanus vaccinations should be updated, and prophylactic antibiotics should be started in the ED. Up to 16% of open-globe injuries may progress to bacterial endophthalmitis caused by Staphylococcus, Streptococcus, or Bacillus species, which are particularly aggressive. Current antibiotic recommendations include ceftazidime (1 g every 8 hours) and vancomycin (1 g every 12 hours). Ciprofloxacin (400 mg every 12 hours) and vancomycin may be used in cases of penicillin allergy.[42]

Metallic intraocular foreign bodies may induce similar localized inflammatory reactions known as metallosis (chalcosis for copper IOFB; siderosis for iron IOFB), which can progress to hypopyon, retinal detachment, and irreversible visual deficits within hours. Therefore, prompt surgical evaluation is the definitive management for open-globe injuries, ideally performed by an ophthalmology specialist familiar with anatomical reconstruction to optimize visual outcomes.

Injury by Presentation: Diplopia

The two major components of the external eye are the extraocular muscles and the bones of the orbit. Post-traumatic deficits in ocular motility are suggestive of extraocular injuries leading to the common complaint of binocular diplopia.

The conical orbit comprises six bones that integrate to create a floor (maxilla), roof (frontal bone), lateral wall (sphenoid and zygoma), and medial wall (maxilla, lacrimal bone, ethmoid, and sphenoid). Orbital wall fractures are largely the result of blunt trauma from an object greater in diameter than the orbital rim such as a fist, ball, or dashboard. The energy from the trauma is dispersed across the orbital rim and elastic globe, which will transmit this force into the orbit. The evolutionary shape of the orbit minimizes the effect of these sudden elevations in intraocular pressure by collapsing, like a safety valve, at its weakest points – the floor and ethmoid aspect of medial wall (lamina papyracea). Sports-related injuries (e.g., baseball, softball, and soccer) are the most common causes of orbital wall fractures in children.[43] In adults, these injuries often are attributed to motor vehicle collisions, assault with a blunt object, and falls.[44]

Blow-out Fractures. Impairment of upward gaze due to entrapment of the inferior rectus muscle is the classic presentation of a blow-out fracture, a fracture of the floor or medial wall of the orbit. Patients commonly will complain of binocular diplopia with periorbital edema, ecchymosis, and an acutely proptotic eye. This can be associated with periorbital crepitus due to subcutaneous emphysema,[45] as well as hypoesthesias of the upper lip and maxillary teeth secondary to infraorbital nerve injury. Enophthalmos, usually a delayed finding, may develop proportional to the size of the maxillary fracture.

Blow-in Fractures. A blow-in fracture is a fracture of the orbital roof (frontal bone) caused by high-velocity blunt trauma directed at the superior orbital rim. A rare fracture (5% of all facial fractures), orbital roof fractures have a strong association with other facial fractures (73% of cases) and intracranial injuries (44% of cases).[46] Because this fracture reduces orbital space, exophthalmos will persist after the periorbital edema has regressed. As in a blow-out fracture, patients will complain of diplopia, but this may be associated with supraorbital hypoesthesia and ptosis secondary to entrapment of the levator palpabrae. In rare cases, intracranial contents may herniate inferiorly, and cerebrospinal fluid (CSF) may leak from the eye.

A CT scan with fine-cut axial and coronal images of the face and orbit is the modality of choice for the diagnosis of orbital bone fractures. Due to the bony immaturity, children will have a higher rate of false negatives despite obvious clinical signs, and management should proceed as if a fracture is present.[47] Surgery is the definitive treatment, and indications include fractures greater than 50% of the orbital floor, extraocular muscle entrapment, and enophthalmos greater than 2 mm. In most cases, intervention will be delayed 7-10 days to allow reduction of post-traumatic edema; thus, emergent management is conservative. Ice packs with head elevation are primary, and the patient should be instructed to avoid nose blowing. Nasal decongestants and a 10-day course of antibiotic prophylaxis against sinusitis may be initiated. Because practice varies, ophthalmology should be notified, but does not require urgent consultation unless the patient demonstrates evidence of an orbital roof fracture, elevated orbital pressures, or stimulation of the oculocardiac reflex (bradycardia/hypotension).[17,22,33,40,43-45,48-52]

Extraocular Muscular Avulsion. Another cause of post-traumatic diplopia is extraocular muscle avulsion. The superior oblique most often is affected due to the close proximity of the trochlea to the superior orbital rim. Penetrating mechanisms are almost always the cause including projectiles (e.g., pellets [BBs], bullets), bone fragments from orbital wall injuries, and dog bites in children. Presentation will vary depending upon the degree of transection. Patients will complain of diplopia, and examination will reveal a motion deficit or, in cases of complete transection, deviation of the globe. CT imaging may confirm a muscle injury, and isolated injuries may be managed conservatively with ophthalmology follow-up within 24-48 hours.[33,44,52]

Retrobulbar Hemorrhage. Because the orbit is a rigid, enclosed space, it is susceptible to a compartment syndrome. Retrobulbar hemorrhage is a very rare injury with serious consequences. Unfortunately studies in the United Kingdom have suggested that an overwhelming majority (73%) of emergency personnel are not familiar with the presentation and management of this disorder.[54] Post-traumatic retrobulbar hemorrhage will occur in the setting of either penetrating or blunt mechanism. Like compartment syndrome elsewhere, pain is the hallmark of this injury with rigid, rock-hard proptosis, which may be accompanied by painful ocular motility progressing to diplopia. Increasing intraocular pressures will reduce perfusion pressures to the optic nerve causing a progressive visual deficit and an afferent pupillary defect that is irreversible within 1-2 hours of onset.[55]

Management begins with recognition of a painful proptotic eye. CT imaging will confirm the presence of an intraorbital hematoma, but if the patient has any visual or pupillary deficits on presentation, immediate intervention is necessary. Concerning presentations with negative CT scans can be assessed with tonometry to confirm normal ocular pressures prior to discharge.

An ophthalmologist should be notified. The patient's head should be elevated, and he should be instructed to avoid any valsalva maneuvers (e.g., coughing, sneezing, and straining). Mannitol, acetazolamide, and steroids should be given to reduce intraocular pressures and protect the optic nerve. If symptoms continue to progress, a lateral canthotomy should be performed with lateral cantholysis of the inferior crus. If intraocular pressures are normalized within a timely manner, symptoms typically resolve within hours, including a return to baseline vision.[44-46,56-58]

Injury by Presentation: Photophobia

The uvea is composed of the iris, ciliary body, and choroids. Because its primary role is pupillary regulation, injuries usually will present as pain with pupillary constriction, and patients will complain of photophobia.

Traumatic Iritis. Traumatic iritis is acute inflammation of the anterior uvea, secondary to blunt or penetrating trauma. The chief complaint will be photophobia, but patients also may complain of headache, blurred vision, or light sensitivity due to traumatic mydriasis. They also will have pain in the affected eye when a light is shone in the unaffected eye, termed consensual photophobia. Slit-lamp examination will reveal ciliary flush with characteristic cell and flare. Treatment consists of symptomatic

relief with a cycloplegic agent such as 5% homatropine and ophthalmology follow-up in 2-3 days.[16,33]

Sympathetic Ophthalmia. Sympathetic ophthalmia is a bilateral, autoimmune, granulomatous uveitis incited by penetrating injuries to the eye. The incidence ranges from 1% in open-globe injuries[59] to 10 cases per 100,000 penetrating eye wounds.[61] Onset can range from five days to 66 years, but the average is 4-8 weeks following trauma.[61] Patients will complain of photophobia with tearing and visual deficits, but the key element in the history is the bilateral presentation (consensual photophobia) despite sustaining an injury to only one eye. If left untreated, the inflammatory conditions related to sympathetic ophthalmia can progress rapidly to profound visual impairment from secondary cataract, glaucoma, or retinal detachment. Slit-lamp examination will reveal cell and flare, and the standard of care in the ED is steroid therapy with prompt ophthalmology follow-up.[62]

Blurred Vision with Intact Afferent Pupillary Reflex

A normal pupillary response is indicative of an intact posterior segment, mainly the retina and optic nerve. Visual deficits with normal accommodation reflect injury anterior to the retina. These injuries obscure the visual axis to the posterior eye; therefore, patients will complain of aberrant vision without a complete deficit.

Traumatic Hyphema. Traumatic hyphema is the presence of blood in the anterior chamber secondary to trauma. The annual incidence is 17 to 20 per 100,000 with a peak age range of 10-20.[63,64] Two-thirds of all hyphemas are due to blunt mechanisms with 44% percent due to assaults,[5,62] 44% sports-related,[59,65] and the remainder associated with motor vehicle collisions and the workplace.[1] Hyphemas are also the most common eye injury associated with paintball trauma.[66] In the pediatric population, 65% of these injuries are sports-related.

A hyphema results from shearing forces on the uvea, leading to disruption of the iris, ciliary body, or choroid with subsequent hemorrhage into the anterior chamber. It is the hallmark of a severe ocular injury, and 25% are associated with a scleral rupture.[35] Most patients will present with eye pain and visual deficits, but the degree of visual impairment will be proportional to the percentage of the anterior chamber occupied by blood. For that reason, hyphemas are graded:

- A Grade I lesion occupies less than one-third of the anterior chamber;
- a Grade II lesion occupies between one-third and one-half of the anterior chamber;
- a Grade III lesion occupies more than half of the anterior chamber; and
- a Grade IV lesion is a total hyphema (black ball or eight ball).[63]

The severity of hyphemas is related to the corresponding complications. One-third of hyphemas are associated with elevated intraocular pressures, which may progress to acute angle-closure glaucoma secondary to obstruction of the trebecular meshwork by blood products or direct compression of outflow channels. Sickle cell disease or trait may exacerbate this effect, progressing to dramatic increases in intraocular pressure even in low volume hyphemas. Persistently elevated intraocular pressures can lead to optic nerve atrophy, and irreversible visual deficits. Finally, large hyphemas may cause corneal blood staining, a yellow discoloration of the cornea contributing to permanent visual deficits.

The first step in examining a hyphema is to rule out an open-globe injury. In the absence of any signs or symptoms of a full-thickness eyewall injury, a thorough history should be obtained, focusing on a medical history of blood disorders such as sickle cell disease, leukemia, Von Willebrand disease, or hemophilia. The history also should note any use of anticoagulant or antiplatelet medications such as aspirin, NSAIDS, clopidogrel, warfarin, or enoxaparin. In children, any delay in presentation or historical inconsistency warrants concern of child abuse.

A CT scan may be necessary to rule out associated orbital fractures or open-globe injury. All hyphemas require slit-lamp examination to evaluate the anterior chamber, and tonometry once an open-globe injury is ruled out.

Twenty-five percent (Grade I) to 67% (Grade III) of hyphemas will rebleed, usually 2-5 days post-trauma.[63] For this reason, management is directed at limiting rebleeding and subsequent elevations in intraocular pressure. This management may vary among specialists; ophthalmology should be notified prior to initiating treatment. The patient should be positioned with the head of the bed elevated to promote settling of the hyphema, and a rigid shield should be placed over the eye to prevent further injury. Topical cycloplegics, such as 1% atropine and topical steroids, may be given to relieve photophobia and intraocular inflammation. To limit rebleeding, recommendations include a five-day regimen of either aminocaproic acid or prednisone. Aminocaproic acid is favored in the setting of sickle cell disease, and may require concomitant, antiemetic therapy. For elevated intraocular pressures (> 25 mmHg), topical beta-adrenergic antagonists, alpha-adrenergic agonists, and carbonic anhydrase inhibitors are recommended. Table 3 outlines indications for outpatient management of hyphemas. If a patient is discharged, he should be instructed to discontinue all anticoagulant/antiplatelet medication, avoid any rigorous activity, and follow up with ophthalmology within 24 hours.[12,62,63,67]

Cyclodialysis. Cyclodialysis is a disruption of the ciliary muscle attachment at the sclera that results in a cleft allowing extravasation of aqueous humor into a potential space within the choroids. It will present in an estimated 4% of blunt eye traumas.[67] Patients will complain of poor vision with possible pain, erythema, and tearing. Because of the loss of volume, the anterior chamber will appear shallow on slit-lamp examination, and intraocular pressures will be low. This hypotony can lead to corneal edema, choroidal detachment, and optic disc edema. All cases of cyclodialysis will require surgical correction, but most of the sequelae are chronic manifestations. Normal vision still can be restored within eight weeks of the injury. Thus, patients may be discharged home with topical cycloplegics, and follow-up with an ophthalmologist within 3-4 days.[58,68]

Lens Subluxation. Lens subluxation is a partial displacement of the lens off the visual axis due to disruption of the lens zonule fibers that anchor it to the ciliary body. Blunt trauma is the most common mechanism. A complete dissociation of the lens from the ciliary body is known as a lens dislocation.

Patients may present with reduced visual acuity, monocular diplopia, or a visual glare, but symptoms may be delayed until months after the initial trauma. Conditions that predispose to lens dislocation with minimal trauma include Marfan's syndrome, homocystinuria, syphilis, Weill-Marchesani syndrome, and retinitis pigmentosa. A slit-lamp examination with pupillary dilation will reveal a displaced lens, prolapse of vitreous into the anterior chamber, or iridodonesis (a trembling of the iris with rapid eye movements). In children, an asymmetric red reflex will characterize lens abnormalities. Subluxation and posterior dislocation are benign conditions that can be managed with a corrective lens. Anterior displacement of a dislocated lens into the anterior chamber is an ocular emergency leading to pupillary obstruction and elevated intraocular pressures (pupillary block glaucoma). Any concern for anterior dislocation requires immediate ophthalmology evaluation for surgical correction, assessment of intraocular pressures, and management with topical beta-adrenergic antagonists, alpha-adrenergic agonists, or carbonic anhydrase inhibitors.[16,17,69]

Traumatic Cataracts. A traumatic cataract results from swelling and opacification of the lens secondary to disruption of the external capsule. This is the most common lens injury in trauma occurring in 39% of open-globe injuries and 11% of closed-globe injuries.[69] In addition, traumatic cataracts accounts for 10% of eye injuries secondary to assault,[6] 32% of workplace-related injuries,[1] and 12% of sports-related injuries.[2] Symptoms tend to be delayed in onset (weeks to months) with a common complaint of a unilateral, progressive blurring of the vision. Because the majority are associated with open-globe injuries and associated vitreoretinal injuries, many traumatic cataracts are diagnosed and managed in the operating room. Chronic, isolated injuries will be apparent on slit-lamp examination and are managed conservatively with ophthalmology follow-up in 2-3 days. Because a swollen, deformed lens may obstruct the pupil lending to pupillary block glaucoma, intraocular pressures should be assessed prior to discharge.[16,70]

Vitreous Hemorrhage. Vitreous hemorrhage is the extravasation of blood into the vitreous space. It is one of the most common post-traumatic ocular injuries, accounting for 40% of assault-related eye injuries,[6] 42% of work-related eye injuries,[1] and more than 60% of paintball-related injuries.[65] Vitreous hemorrhage also has a strong association with shaken baby syndrome.[17] The sources of bleeding include the iris, ciliary body, choroids, and retina. Although the mechanism may be blunt or penetrating, vitreous hemorrhage most often is associated with a closed-globe injury. Visual deficits represent the most common presentation, with patients complaining of a haze, floaters, cobwebs, smoke signals, or simply a shadowy appearance to their vision. This may be associated with pain or photophobia, depending on the site of disruption. Work-up is directed at excluding retinal detachment. Direct ophthalmoscopy should be employed, but may be obscured by blood. Ultrasound is useful in diagnosing a corresponding retinal detachment, but CT imaging will provide additional information on the integrity of the choroids, sclera, or any evidence of intraocular foreign body. Treatment of an isolated vitreous hemorrhage is conservative with symptomatic relief of pain as needed. Patients can be discharged home with instructions for bed rest with head elevated and ophthalmology follow-up in 2-3 days.

Complications related to vitreous hemorrhage include retinal detachment secondary to traction from the hemorrhage and ghost cell glaucoma, elevated intraocular pressures secondary to obstruction of aqueous humor outflow tracts by the byproducts of hemoglobin degradation. Onset is usually 1-3 weeks post-trauma, and patients will present with a painful eye with a beige collection of cells within the anterior chamber that often is confused for a hypopion. The presence of ghost-cells, elevated intraocular pressures, and impending retinal detachment are all indications for immediate surgical evacuation of the vitreous.[33,71,72]

Table 3. Traumatic Hyphema: Indications for Outpatient Management

- No associated ocular injury mandating hospitalization
- Hyphema less than Grade II
- Intraocular pressure < 35 mmHg
- No history of sickle cell disease, blood dyscrasias, or coagulopathic disorders
- No concern regarding the safety of home environment (including child abuse), ability to comply with limited activity, ability to comply with medication regimen, or ability to follow up with ophthalmology within 24 hours

Vision Loss with an Afferent Pupillary Defect

Trauma to the posterior segment of the eye (retina/optic nerve) may impair the pupillary light reflex, causing a pupil that will fail to constrict with direct light stimulation, known as a relative afferent pupillary defect.[73] In addition, posterior segment injuries also may have profound visual deficits, including complete loss of vision.

Retinal Detachment. Retinal detachment is a separation of the superficial neurosensory layer of the retina from the underlying pigmented layer. Fifteen percent of all retinal detachments are caused by trauma with blunt mechanisms (70-85% of cases), the most common etiology.[74] The development of a detached retina is a three-step process. The first is a break in the retinal layer generated during the initial trauma. Retinal dialysis (detachment from ciliary body) is the most common of these precipitants, but others include operculated holes, horseshoe flaps, or areas of necrosis. Secondly, fluid will seep into this defect and weaken the bond between the two superficial layers. Finally, traction must be provided to separate the two layers. During the initial trauma, this is provided by a blunt mechanism and the subsequent elastic recoil of the globe that places traction at the vitreous base. In many instances, this traction is a delayed process caused by the contraction of the vitreous secondary to healing.

Due to the separation from its blood supply in the choroids, the separated layer becomes ischemic and irreversibly atrophies.

In many cases, a retinal detachment will be delayed months to years after the initial injury. Patients will complain of seeing light flashes progressing to a "falling curtain of darkness." This may lead to profound visual deficits with a relative afferent pupillary defect, an unreactive iris to direct stimulation that constricts with stimulation of the opposite eye (consensual response). Examination in children may reveal a diminished red reflex. On direct ophthalmoscopy, detachments appear as grayish billowing of the retina; however, many smaller or peripheral detachments may not be seen with simple fundoscopy (even after dilating the pupil). Ultrasound will reveal a smooth membrane within the vitreous cavity or a triangular shape extending from the optic disc in cases of total detachment. Immediate surgical intervention is indicated, thus, any pupillary deficit or suspicion of retinal detachment requires immediate ophthalmology evaluation.[16,71,72,75-77]

Because of the high incidence of ocular findings in child abuse, retinal detachment in children should raise suspicion, but retinal hemorrhage in children younger than 3 years is pathognomonic for shaken baby syndrome.

Optic Nerve Injuries. Optic nerve injuries have a 7% incidence in major trauma[9] and will result in permanent visual loss in 50% of cases.[78] The optic nerve can be injured by two major mechanisms. Direct injuries cause trauma to the nerve usually by penetrating, open-globe mechanisms such as bone fragments, foreign bodies, or projectiles (e.g., bullets, BBs). Walsh and Hoyt described indirect injuries as a "traumatic loss of vision which occurs without external or initial ophthalmoscopic evidence of injury to the eye or the nerve."[79] These injuries are caused by transmitted forces upon the optic nerve due to bony apposition or globe mobility. Indirect injuries are the most common cause of traumatic optic neuropathy usually attributed to blunt, deceleration injuries with impact at the supraorbital or frontal regions of the head. Such forces are encountered in motor vehicle collisions, bicycle accidents, assaults, and falls, but optic nerve injuries also have been described in lower energy mechanisms such as falling debris and skateboard accidents.[77,80,81]

The optic nerve can be divided into four anatomic sections: the intraocular portion that we recognize as the optic disc; a mobile intraorbital section; a fixed intracanalicular section that is accompanied by the ophthalmic artery as it runs through the optic canal; and an intracranial section that joins the complimentary optic nerve to form the optic chiasm. The intracanalicular portion is the most common site of injury and follows a dual insult paradigm. The primary insult, at the time of initial trauma, results from shearing forces due to a mobile globe and intraorbital nerve section placing stress on an immobile intracanalicular section and vessels. This results in permanent axonal injury, as well as, disruption of the vasculature with possible hemorrhage. This loss of blood supply produces a secondary ischemia and circulation of toxic metabolites such as free radicals, bradykinin, and calcium. Any axons that survive the initial insult may succumb to the secondary effects of ischemia, contributing to the delayed presentation of optic nerve injury.[75,77,79,82,83]

The most common presentation for traumatic optic neuropathy is a visual deficit with an afferent pupillary defect (Marcus-Gunn pupil) on exam. Typically, patients present with vision of 20/400 or less, with 10% having a delayed-onset of symptoms. Visual fields deficits are indicative of intracranial injury, but in most instances will be difficult to elicit; more than 50% of optic nerve injuries are associated with a loss of consciousness.[50,77]

Any deficits in vision should raise suspicion of an optic nerve injury. Again, an afferent pupillary defect is diagnostic of a posterior segment injury and requires immediate ophthalmologic evaluation. Delayed loss of vision can be described as a lucid interval before normal vision rapidly fades. Often it is associated with compression from an expanding hematoma; in most cases, full visual acuity is returned with prompt surgical decompression. This rapid deterioration of vision has a good prognosis, while delayed regressions during a 1-2 week period are associated with axonal atrophy and suggest more permanent visual deficits.

The physical exam should focus on reversible causes of optic nerve injury—specifically signs of retrobulbar hemorrhage such as a rigid globe and diplopia. Ophthalmoscopic examination will vary depending upon the location of the injury. Anterior injuries involving the intraocular portion of the nerve will demonstrate an edematous retina with a pale optic disc if the central retinal artery is involved. Otherwise, an avulsed intraocular optic nerve will produce a hemorrhagic ring on the fundus, with the optic disc appearing as a deep round pit. Posterior injuries involving the intracanalicular portion of the nerve will appear normal on initial presentation. Only after 3-6 weeks will the disc appear pale and atrophic. In more subtle cases, be aware of the close association of optic nerve injury with midface fractures (2.5% incidence). All patients with suspected ocular nerve injuries should receive a CT scan to evaluate for fractures, as well as, pathologic nerve sheath hematomas or retrobulbar hemorrhages.[16,77,84,85]

In 1998, The International Optic Nerve Trauma Study[86] attempted to evaluate the current management recommendations for optic nerve trauma, high-dose steroids vs. surgical decompression. Researchers concluded that neither improved visual outcomes, but this study was retrospective and grossly lacked power (n=133). There has never been a prospective, randomized trial to determine efficacy in the management of these injuries; one of the reasons is that the incidence of the disorder is too low to generate statistical significance within a given community. Thus, high-dose steroids commonly are used in some centers. Surgical decompression also is employed, but due to a lack of a universal protocol, the use of these therapies will vary by region, therefore, a consulting ophthalmologist should be notified prior to initiating any treatments.[77,79,81]

Prognosis and Conclusion

In general, post-traumatic eye injuries with poor visual acuity on initial presentation have a lower probability of regaining baseline vision. Pieramici et al demonstrated that 88% of patients with post-traumatic visual acuity of 20/40 or better retained that degree of acuity on follow-up visits. In contrast, 79% of patients with a post-traumatic inability to perceive light on initial evalua-

tion required enucleation of the affected eye.[87] Much of this prognostic data related to eye trauma focused on open-globe injuries in recent years; other initial indicators of poor visual outcome in the setting of these injuries include the length of the eye-wall defect, presence of an afferent pupillary defect, prolapse of intraocular contents, and finally, the presence of a hyphema.[88,89]

Pitfalls in assessing and managing eye trauma are related to the rarity of true vision-threatening injuries. The majority of the eye injuries evaluated by emergency health care providers will be benign, but complacency can be overcome by maintaining a systematic approach to all eye injuries, while being mindful of the hallmarks of severe injuries such as rigid proptosis and the absence of a pupillary response. As a final precaution, all eye injuries, regardless of their triviality, should be referred for follow-up ophthalmology evaluation.

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CE/CME Objectives

Upon completing this program, the participants will be able to:

a.) recognize or increase suspicion for traumatic injuries that present to the emergency department;

b) describe the various modalities used to identify different traumatic conditions covered in the newsletter;

c) describe how to correctly and quickly stabilize, and then to manage patients with the particular condition covered in the newsletter; and

d) identify both likely and rare complications that may occur with traumatic injuries.

CE/CME Instructions

Physicians and nurses participate in this continuing medical education/continuing education program by reading the article, using the provided references for further research, and studying the questions at the end of the article. Participants should select what they believe to be the correct answers, then refer to the list of correct answers to test their knowledge. To clarify confusion surrounding any questions answered incorrectly, please consult the source material. **After completing this activity, you must complete the evaluation form provided and return it in the reply envelope provided in order to receive a certificate of completion.** When your evaluation is received, a certificate will be mailed to you.

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CE/CME Questions

1. Which of the following conditions is *not* characterized as an open-globe injury?
   A. Intraocular foreign body
   B. Corneal abrasion
   C. Corneal laceration
   D. Scleral rupture

2. Which of the following conditions is *not* associated with diplopia?
   A. Orbital wall fractures
   B. Lens subluxation
   C. Retrobulbar hemorrhage
   D. Retinal detachment

3. A rigid rock-hard proptotic eye is characteristic of what post-traumatic eye injury?
   A. Retrobulbar hemorrhage
   B. Traumatic hyphema
   C. Scleral rupture
   D. Orbital wall fracture

4. Which of the following conditions is *not* associated with elevated intraocular pressures?
   A. Traumatic hyphema
   B. Vitreous hemorrhage
   C. Anterior lens dislocation
   D. Posterior lens dislocation

5. Which of the following eye injuries is the most specific for shaken baby syndrome?
   A. Retinal hemorrhage
   B. Retinal detachment
   C. Traumatic optic neuropathy
   D. Vitreous hemorrhage

6. Which of the following is *not* part of the anterior segment of the eye?
   A. Lens
   B. Iris
   C. Retina
   D. Cornea

7. Which of the following groupings has the highest incidence of eye trauma?
   A. Male, ages 10-15
   B. Female, ages 10-15
   C. Male, ages 25-30
   D. Female, ages 25 -30

8. Which of the following agents account for the majority of corneal burns?
   A. Acidic chemical agents
   B. Alkali chemical agents
   C. Thermal agents
   D. Ultraviolet light

9. What are the two most common bones injured in orbital wall fractures?
   A. Maxilla and sphenoid
   B. Maxilla and frontal
   C. Maxilla and ethmoid
   D. Maxilla and zygoma

10. Where is the most common anatomic site of optic nerve injury from blunt trauma?
    A. Intraocular (optic disc)
    B. Intraorbital
    C. Intracanalicular
    D. Intracranial (optic chiasm)

Answer Key: 1.B; 2.D; 3.A; 4.D; 5.A; 6.C; 7. A; 8.B; 9.C; 10.C

In Future Issues:

Management of the Burned Patient