Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

Treatment of pediatric sinusitis

Ari J. Goldsmith, MD[a,b,*],
Richard M. Rosenfeld, MD, MPH[a,b]

[a]*Pediatric Otolaryngology, SUNY Health Sciences Center at Brooklyn, 450 Clarkson Ave., Box 126, Brooklyn, NY, USA*
[b]*Division of Pediatric Otolaryngology, Long Island College Hospital and University Hospital of Brooklyn, 450 Clarkson Avenue, Box 126, Brooklyn, NY 11203, USA*

Pediatric sinusitis has a significant adverse effect on health-related quality of life in children [1]. Despite widespread acceptance of this principle, the proper management of sinusitis in children is controversial. One school of thought maintains that the disease is typically self-limited and therefore should be treated rarely with antibiotics, and almost never with surgery. Others maintain that these infections should be treated with aggressive medical therapy and surgical treatment when medical therapy fails. The correct answer lies somewhere between these extremes; therefore this article strives to is to provide a reasonable and effective strategy for the treatment of sinusitis in children, balancing the self-limited nature of the disease with the potential for detrimental effects on quality of life.

Definitions

Rhinosinusitis (RS) is the more correct term for sinusitis in children, because most infections begin with or have a concomitant rhinitis. In addition, the pathophysiology of the disease is thought to be more than just an abscess cavity of the sinus, and probably involves changes in the mucosa of both the nose and sinus cavities. Several varieties of RS can be categorized as follows:

1. A. *Acute RS (ARS)* most commonly presents as an acute upper respiratory infection (URI) that persists (without improvement) beyond 10 days, with the key signs and symptoms being nasal discharge, cough, and bad breath. The 10-day mark is somewhat subjective, because viral URIs can

---

* Corresponding author. Long Island College Hospital and University Hospital of Brooklyn, 450 Clarkson Avenue, Box 126, Brooklyn, NY 11203.

E-mail address: pedsent@usa.net (A.J. Goldsmith).

persist beyond 10 days. A more accurate predictor of ARS would be the worsening of symptoms at 7 to 10 days, whereas with viral URIs, some improvement typically would be noted.

B. A less common presentation of ARS is that of a "severe" URI, with fever of 102°F plus concurrent purulent nasal discharge for 3 to 4 consecutive days. Conversely, acute rhinitis that is preceded by fever (not concurrent with fever) usually is viral in origin.

2. *Chronic RS (CRS)* is a more indolent infection that lasts beyond 3 months, with the typical symptoms being nasal congestion, cough, and bad breath. Other signs and symptoms include behavioral problems, headache, and nasal discharge. Exacerbations are common.

3. *Recurrent ARS (RARS)* implies recurrent infections with relatively healthy intervals in between, although some element of CRS usually is present between acute infections.

Diagnosis

Although this article focuses on treatment of RS, proper diagnosis is critical in establishing the need for treatment. Appropriate diagnosis will ensure appropriate treatment, which will decrease the chances of subsequent development of bacterial resistance in both the patient and the overall community. The proper paradigm in this age of increasing bacterial resistance needs to be:

Correct diagnosis of RS → tailored patient treatment
   → successful patient resolution → decreased recurrence
   → decreased community bacterial resistance

The common diagnostic problem is differentiating a viral URI from a sinus infection. In general, the diagnosis of ARS and CRS depends on clinical presentation alone, correlated with physical findings. Although occasionally not seen in CRS, the finding of purulent secretions in the region of the middle meatus is highly suggestive of sinusitis. Radiographic imaging is reserved for children with refractory RS who are being considered for surgery, or for suspected complications such as orbital or cranial infection. CT scanning, which is considered the gold standard for the diagnosis of sinusitis [2], will demonstrate sinus opacification in most children who have a nasal discharge that is self-limited in nature and who will never require antibiotics [3]. When the clinical presentations of ARS and CRS described above are correlated with findings on anterior rhinoscopy—which is best performed looking through an otoscope while gently pushing up on the nares—the diagnosis of RS is quite accurate. When rigid diagnostic criteria are adhered to, relatively few children with nasal congestion actually will be diagnosed as having RS [4]; this fact reinforces the need for careful diagnosis. Studies suggest that decreased measured levels of nitric oxide in exhaled nasal air may distinguish RS from URI [5], though this procedure is experimental at the present time.

Causative factors

When considering treatment options, it is critical to be aware of those forces that contribute to the development of RS. Knowledge of these "causative factors" will allow for treatment and prevention of disease by more appropriate medical or nonmedical means. For example, sinusitis caused by allergy is best prevented by knowledge and avoidance of offending allergens, as opposed to chronic antibiotics. More importantly, if these factors are neglected, treatment ultimately will not be successful. Although some of the causative factors listed below are uncommon, they still must be considered if suggested by the clinical scenario.

1. *URI*: Most sinus infections in children develop following a viral URI, most commonly rhinovirus, coronavirus, and influenza virus. Children average between six to eight colds a year (and even more in day-care settings), of which 5% to 10% are complicated by RS. Inflammation of sinus ostia causes stasis of secretions and poor ventilation of the affected sinus. This leads to absorption of oxygen and the development of a relative negative pressure or vacuum within the sinus, causing movement of intranasal contents and nasopharyngeal bacteria into the sinus cavity. Viruses also can have a direct inhibitory effect on ciliary function. Finally, there is increased bacterial growth in the presence of viral infection.

2. *Bacterial pathogens*: As in purulent otitis media, most ARS is caused by *Streptococcus pneumoniae*, *Haemophilus influenzae* (nontypable), and *Moraxella catarrhalis*. In addition to these bacteria, CRS also can be caused by anaerobic organisms and *Staphylococcus aureus*. Resistance patterns are important in predicting response to antimicrobial therapy: *H. influenzae* and *M. catarrhalis* can be resistant to beta-lactam antibiotics such as ampicillin due to beta-lactamase production. In contrast, *S. pneumonia* and *S. aureus* can be resistant to the penicillins and most other antibiotics by a genetic alteration in penicillin-binding proteins. This form of resistance is much more significant because it is not treated successfully by the typical "second-line" agents such as cephalosporins, macrolides, and amoxicillin/clavulanate. Oral antibiotic options include high-dose amoxicillin and clindamycin.

3. *Allergy*: The relationship between allergy and RS in children is well known. More than 80% of children with RS have a family history of allergy, as opposed to a general population allergy frequency of 15% to 20% [6]. More than half of sinusitis is closely associated with asthma, a recognized allergic-mediated disease [7]. Finally, when various allergy tests are employed, more than half of the children with chronic sinusitis will have some element of allergy [8]. Allergy can contribute to sinusitis by either nasal congestion and subsequent ostia obstruction, or direct allergic effects on sinus lining cells. Although not IgE mediated, cow's milk protein allergy may be present in very young children with a history of rashes or colic, and can be a contributing factor to RS in these children.

4. *Adenoid vegetations*: The adenoid is a lymphoid organ located in the nasopharynx, similar in form and function to the pharyngeal tonsils. The adenoid contributes to RS in children by serving as a bacterial reservoir, similar to otitis media. Cultures of the adenoid core demonstrate organisms similar to those seen in RS [9], and studies have shown that removal of the adenoid will improve RS for 70% to 80% of children [10]. As in otitis media, the size of the adenoid pad is unrelated to the ability to harbor pathogens.

5. *Airway pollutants* can have direct irritant effects on the nasal and sinus mucosa. The most significant irritant in RS is environmental tobacco smoke, which has been shown to cause otitis media in children, and has been suggested to similarly contribute to RS.

6. *Structural anomalies of the sinus and nasal cavity* are rare causes of sinusitis, and include septal deviation, lateral nasal wall anomalies (paradoxical middle turbinate, concha bullosa, Haller cells), and maxillary sinus hypoplasia [11].

7. *Gastroesophageal reflux disease (GERD)*: Many opinions exist as to the relationship between GERD and RS in children, although most pediatric otolaryngologists agree that some RS is due to, or exacerbated by, GERD. GERD is prevalent in children with CRS [12], while double-lumen pH probe testing has shown that esophageal reflux can extend to the area of the nasopharynx [12–14]. A recent retrospective study [15] suggested that GERD therapy could prevent sinus surgery in almost 90% of children with refractory CRS.

8. *Immunologic defects*: All young children have a relative immune-deficient state due to a slow rise in immunoglobulin production, which may be a more important causative factor than is allergy in younger children. This is demonstrated by a decrease in RS prevalence in older children, despite an increase in the prevalence of allergy [16]. In contrast to this self-limited phenomenon, pathologic immune deficiency can cause severe and refractory RS in affected children. Humoral immunodeficiency is the most common disorder in these children, with IgG subclass deficiency being the most common subtype [8]. In general, children with immune deficiency will present with multiple upper and lower respiratory infections in addition to RS, including pneumonia, bronchitis, and otitis media.

9. *Primary ciliary dyskinesia (PCD)*: PCD is a rare disorder of ciliary structure or function that can occur alone, or can be a part of the more global Kartagener's syndrome. Structural abnormalities can be demonstrated by electron microscopy of nasal or tracheal biopsies, and include deviation of the normal 9 + 2 microtubule structure or a more subtle decrease in dyenin arm count [17]. Functional abnormalities can occur despite normal ciliary architecture, and can be demonstrated by a decrease in ciliary beat frequency [18], although this test is mainly of research interest and rarely is used in clinical settings. As with children who have

immunologic defects, children with PCD will have other upper and lower respiratory infections in addition to RS.

10. *Cystic fibrosis (CF)*: CF occurs in 1 in 3200 white newborns in the United States, and is the most common life-limiting recessive genetic disorder in whites [19]. Affected children present with chronic upper and lower respiratory infections—most commonly pneumonia and severe refractory RS—often with nasal polyp formation. Other manifestations include malnutrition, intestinal obstruction, and pancreatic insufficiency [19]. Although elevated sweat chloride concentrations are the hallmark of CF diagnosis, the ability to detect specific genetic mutations recently has been shown to be a much more reliable and accurate method of diagnosis, especially in less severely affected children [20,21]. These children can have milder upper and lower respiratory disease including RS, and may have normal sweat chloride concentrations.

Treatment overview

The treatment of bacterial infections traditionally involves presumptive diagnosis followed by antimicrobial treatment. This approach, although often successful in ARS, will not prevent recurring acute infections or adequately treat CRS. This can lead to antibiotic overuse, unnecessary surgery, and decreased patient and family quality of life. In addition, RS can rarely lead to suppurative complications including osteomyelitis, orbital infection, meningitis, and brain abscess, emphasizing the importance of appropriate treatment in more severely affected children.

The treatment of both acute and chronic sinusitis in children must consider the following principles:

1. Proper diagnosis.
2. Ascertaining disease burden.
3. Defining reasonable caregiver expectations.
4. Identification and treatment of causative factors.
5. Appropriate antimicrobial management.
6. Appropriate ancillary medical treatment.
7. Appropriate surgical therapy.
8. Vaccine administration.

The importance of proper diagnosis was discussed above, and must consider the accepted definitions of ARS, CRS, and RAS to prevent "mixing apples with oranges" when planning and evaluating treatment approaches.

Disease burden can be ascertained by directed discussions with the child, family, and primary care practitioner [22]. Factors include frequency of infections, duration and severity of symptoms, frequency of physician visits, medication use, baseline state between infections, and impact on behavior and school performance [22].

Caregiver expectations must be assessed, because unreasonable expectations always will lead to unhappy patients and family. The goals of therapy should be discussed with the parents before beginning treatment.

Identification and treatment of causative factors

Causative factors were detailed previously, and must be included in the treatment approach. They are particularly important in the prevention and treatment of recurrent or chronic infections. A summary of causative factors as they pertain to treatment approach is given below; adenoid and structural anomalies are addressed in the section on surgical therapy.

1. Although URIs are an inevitable part of childhood, certain steps can decrease their incidence and severity. Day-care centers should be as small and clean as possible, and older siblings should be encouraged to wash up as soon as they get home from school or day care.
2. Knowledge of local bacterial prevalence and resistance patterns are available from hospital microbiology labs. In addition, appropriate use of antibiotics for bacterial URIs will discourage the growth of resistant bacteria in both the child and the community.
3. Allergy should be considered in all children with the following: a history of allergic signs and symptoms (watery rhinorrhea, pruritis, sneezing, transverse nasal crease, allergic shiners, frequent rashes), seasonal patterns of infection, specific allergen reactions (dust, pet dander, particular foods), and a strong family history of allergy or asthma. Whereas milder seasonal allergies can be treated with antihistamines and inhaled nasal steroids, more severe cases are best evaluated and treated by a pediatric allergist who can identify the allergen and prescribe avoidance techniques, targeted drug therapy, and immunotherapy when necessary. The topic of cow's milk protein allergy in young children is controversial, although milk should be avoided in very young children with frequent or chronic RS who also have a history of neonatal colic or rashes.
4. Exposure to passive smoke must be eliminated due to its potential effects on nasal and sinus mucosa. In addition, otitis media and asthma can be prevented by this reasonable intervention.
5. GERD should be considered in all children with difficult RS in addition to other suggestive features, including frequent vomiting; unexplained respiratory symptoms such as vocal cord spasms, early hoarseness, and atypical wheezing; and findings on physical exam such as cobblestone appearance of the pharynx. The evaluation of suspected GERD is best performed by a pediatric gastroenterologist who is experienced in the relationship between RS and GERD; this evaluation typically involves pH probe testing. Direct laryngoscopy with posterior laryngeal biopsy can help to diagnose upper airway GERD when there are findings of posterior laryngeal edema, mucosa cobblestoning, and biopsy with eosinophils in the

basement membrane [15]. Empiric therapy with an H2 blocker such as ranitidine may be considered in a young child with suspected reflux-induced rhinitis [23]. A positive response to this ''therapeutic trial'' strongly suggests GERD as an etiologic factor.

6. Immune deficiency, primary ciliary dyskinesia (PCD), and CF are relatively uncommon, although they should be suspected in children with difficult RS together with a history of frequent upper and lower respiratory infections such as pneumonia and bronchitis.

   a. Immune testing typically includes total immunoglobulin levels, IgG subclass levels, and postvaccination titers to pneumococcal and *H. influenzae* type b vaccines [24]. Immunoglobulin replacement and daily antibiotics are the basis of successful therapy [24,25], although gene therapy eventually may prove to be successful in these patients and is presently being evaluated in clinical trials [25].

   b. Evaluation of ciliary structure can be performed on specimens obtained from nasal brushings (posterior turbinate), although tracheal brushings performed via bronchoscopy have a higher yield of respiratory mucosa. Although there is no specific treatment for PCD, the chronic use of treatment and prophylactic antibiotics can then be rationalized better.

   c. CF testing traditionally has involved sweat sodium chloride levels, although genetic testing now is accepted as the more accurate method of diagnosis, especially in children with milder phenotypic variants of CF that may indeed have normal sweat tests. This disease is best treated by an experienced CF practitioner.

Medical treatment of ARS

Antibiotics

As in otitis media, the treatment of ARS in children must take into account the natural history and spontaneous resolution of the disease in one out of three to two out of three children [26,27]. Indeed, a recent, randomized placebo-controlled study of 188 children with ARS between 1 and 18 years [28] showed no benefit of antibiotics over placebo in sinus symptoms, relapse, or recurrence, although this study included older children and excluded sicker children. In contrast, most studies [29–32] have suggested that antibiotics allow for earlier resolution and may prevent recurrence, prompting the American Academy of Pediatrics (AAP) to recommend in their recent guidelines [33] that antibiotics be used in the treatment of ARS of suspected bacterial etiology. These recommendations were either evidence based or consensus based when no clear data existed.

• For young children with mild to moderate ARS, amoxicillin is recommended at the normal (45 mg/kg) or high dose (90 mg/kg).

- Patients with amoxicillin allergy should be treated with a cephalosporin such as cefdinir, cefuroxime, or cefpodoxime, whereas severely allergic patients should be treated with a macrolide such as clarithromycin or azithromycin.
- Children that do not respond to first-line therapy, children with more severe initial disease, and children who are considered high-risk for resistant *S. pneumoniae* (those who recently have used antibiotics or attend day care) should be treated with high-dose amoxicillin/clavulanate (90 mg/kg of amoxicillin component).
- Parenteral ceftriaxone should be used in those children who are vomiting and cannot take oral antibiotics.

In contrast to the AAP guidelines, the Sinus and Allergy Health Partnership in 2000 [30] devised their own guidelines for the treatment of acute sinusitis in adults and children, based on a mathematic model that included pathogen distribution, pathogen-specific spontaneous resolution rates, and in vitro susceptibility using pharmacodynamic principles. Based on these data, they recommended that children with mild sinus disease and no antecedent antibiotic use in 6 weeks receive amoxicillin/clavulanate (93.5% efficacy), low-dose or high-dose amoxicillin (91% efficacy), cefpodoxime (86.7% efficacy), or cefuroxime (83.7% efficacy) [30]. Children who are allergic to penicillins should receive erythromycin, clarithromycin, or azithromycin, despite lower predicted efficacy. More severely affected children should initially receive those drugs with the highest predicted efficacy. Optimum duration of therapy in ARS has never been proven in scientific trials, although recommendations typically include 10 to 21 days, or until symptoms resolve plus an additional 7 days [34].

Adjuvant therapies

The AAP guidelines do not specifically recommend any adjuvant therapies. One study [35] did suggest that topical steroid spray (budesonide) might improve symptoms during the second week of therapy when combined with appropriate antibiotics, although a larger multicenter trial in older children [36] showed no effect. Topical sympathomimetics often are used in ARS, although oxymetolazine actually has been shown to increase inflammation in a rabbit model, possibly due to decreased blood flow [37]. Saline spray is helpful in clearing out purulent secretions, although one in vitro study [38] suggested that only isotonic saline had no adverse effects on ciliary function, whereas isotonic and hypertonic saline actually decreased ciliary function. One study showed that antihistamine decongestants did not improve clinical cure in ARS [39], although this study included an abnormally low number of children with allergy, and the antihistamine preparation was a first-generation antihistamine that may have had anticholinergic drying effects.

In general, adjuvant therapies are not necessary in the treatment of uncomplicated ARS, although saline spray may make children feel better by clearing out

secretions, and the newer nonsedating antihistamines (loratidine and cetirizine) may be beneficial in those children with ARS where allergy is suspected as the causative factor.

Medical treatment of CRS

Successful treatment of CRS in children is critical, because failure usually will lead to surgical therapy. Despite this, CRS treatment has not been studied adequately, and a recent clinical practice guideline for CRS [40] needed to be devised by an outcome-based method as opposed to an evidence-based method, due to a lack of adequate supporting literature. This study suggested that children with CRS should be treated with a combination of an antibiotic and a nasal steroid spray, although no specific recommendations could be made about duration of therapy and choice of drug [40]. "Maximum medical therapy" is the term used to describe CRS treatment with adequate duration and choice of antibiotics, with or without a concomitant nasal steroid spray.

Antibiotics

Studies on short-term antibiotic use in CRS [41,42] have shown no benefit to short-term antibiotic therapy (7 to 10 days); thus, it is reasonable to suggest that antibiotics need to be used for a longer period of time—from 21 days to 6 weeks [27,43–45]—despite the absence of any supporting scientific studies.

The choice of antibiotic must take into account the likely pathogens and known resistance patterns. The polymicrobial nature of CRS and the presumed higher levels of resistant bacteria make it prudent to use a drug that theoretically can cover all possible organisms, because directed culture is often difficult when purulent secretions are absent, and cultures often are negative because these children typically have been on many antibiotics. This is especially true for children in day care and children who recently have been on antibiotics [46].

A recent international consensus meeting [45] concluded that appropriate antibiotic agents for CRS include amoxicillin/clavulanate and the second-generation cephalosporins (excluding cefaclor). Although all of these drugs have good clinical and bacteriologic profiles, there is some concern with regard to spectrum, because none of these antibiotics has shown acceptable coverage against intermediate or highly resistant *S. pneumoniae*. In contrast, a recent, large, double-tap otitis media study [47] showed that high-dose amoxicillin/clavulanate (90 mg/kg) covered 94% of *H. influenzae* (including beta-lactamase producing strains), and 98% of *S. pneumoniae*, including more than 90% of the resistant *S. pneumoniae*. This bacteriologic profile makes high-dose amoxicillin/clavulanate an ideal drug for the treatment of CRS, although no scientific studies exist. In addition, as compared with the macrolides, beta-lactams are less likely to contribute to the growth of resistant bacteria in the nasopharynx [48]. High-

dose amoxicillin/clavulanate must be taken twice daily, however, and also has a higher incidence of gastrointestinal side effects than most other antibiotics, though the incidence is no greater than that with regular-dose amoxicillin/clavulanate [49].

Penicillin-allergic patients can be treated with the cephalosporins listed above, although highly allergic patients should be treated with a macrolide. There are, however, increasing concerns of an *H. influenzae* resistance to the macrolides [50]. Clindamycin may be used if *S. pneumoniae* is the suspected organism based on culture or if there is no response to other antibiotics.

Adjuvant therapies

Topical nasal steroids typically are used to treat CRS [45], although no scientific data exists with regard to their efficacy. Mometasone furoate is the only drug approved for children who are 2 years of age or older, based on long-term studies that showed no effect on growth [51] and no effect on the hypothalamic-pituitary-adrenal axis (HPA) axis [52]. Fluticasone propionate is approved for children who are 4 years of age, whereas most of the other topical nasal sprays, such as budesonide and triamcinolone, can be used in children older than 6 years. It is reasonable to continue the topical nasal steroid spray for the antibiotic duration.

As noted previously, saline spray may make children feel better by clearing out secretions, and the newer nonsedating antihistamines (loratidine and cetirizine) may be beneficial in children with CRS, the origin of which is a suspected underlying allergy. Mucolyitcs such as guaifenesin also may help thin secretions and may theoretically help the patient feel better sooner.

Surgical therapy

Perhaps the most difficult decision in the treatment of pediatric RS is deciding which child needs surgical therapy. A reasonable yet subjective approach is the following: surgery is indicated for children with CRS or RAS who have failed maximum medical therapy (see above); are unlikely to have untreated allergy or GERD; and who have no untreated contributory systemic disease such as immune deficiency, PCD, or CF. This approach will avoid unnecessary surgery [53], and improve the quality of life of those difficult cases that fail appropriate medical therapy [41].

When surgery is indicated, adenoidectomy should be the initial procedure if the quantity of adenoid tissue visualized on endoscopy or imaging is considered sufficient to serve as a reservoir of bacterial pathogens. Adenoidectomy is almost always the first-line surgical intervention for preschoolers, and often is appropriate in older children. The expected rate of improvement is 70% to 80% [9,10,44]. Adenoidectomy is safe, has minimal morbidity, and has no effect on long-term immune function; and children can usually return to school within 1 to 2 days. Controversy as to whether or not to perform adenoidectomy alone exists in older children or when the adenoid pad is small [9,26,54], although if the

pathophysiology is that of bacterial reservoir [9], adenoidectomy still is an appropriate first-line surgical option. Although no studies have shown maximum medical therapy to be effective, it (see above) may be continued for several weeks after adenoidectomy, in the hopes of completely sterilizing the sinus cavities following removal of the likely bacterial reservoir. The potential detrimental effects of increased bacterial resistance, however, must be considered in the decision for or against prolonged antibiotics.

Endoscopic sinus surgery (ESS) should be performed only when children have failed previous therapies [10,44]. In contrast to older traditional techniques of sinus surgery, ESS focuses on enlarging the natural ostia of the maxillary and ethmoid sinuses, while preserving most or all of the sinus mucosa. In properly selected children, the results are good, with an expected improvement of 80% to 100% [22,55,56]. Preoperative CT scan is essential in defining the specific diseased sinuses, and in looking for anatomic abnormalities that need to be addressed, including septal deviation, concha bullosa, and paradoxical middle turbinate. The typical procedure, however, is that of a "mini-ESS," consisting of uncinectomy plus limited anterior ethmoidectomy. The surgery should be performed only by an otolaryngologist experienced in pediatric ESS [22,55]; when properly performed, the incidence of major complications is less than 1% [55]. Theoretic concerns with adverse effects on facial growth [57] have not been substantiated in human trials [58,59]. Second-look endoscopy is unnecessary [60], although maximum medical therapy (see above) should be continued for several weeks after ESS. Reasonable caregiver expectations are essential—parents and children should expect an improvement, but not a cure.

A recent retrospective review [61] suggested that maxillary sinus aspiration and culture followed by culture-directed intravenous antibiotics might avoid ESS in children with persistent sinusitis after oral antibiotic therapy. Further controlled studies will need to address the feasibility of this option. The role of adenoidectomy could not be ascertained because adenoidectomy was performed at the discretion of the surgeon and not based on scientific reasoning.

Vaccines

Although no studies have addressed specifically the role of vaccines in the prevention of ARS and CRS, it is reasonable to suggest that severely affected children will benefit from vaccines such as the conjugated pneumococcal vaccine (Prevnar, Wyeth, Pharmaceuticals, Philadelphia, PA), due to reduced infections by this specific bacteria [62]. In addition, knowing that a child has received the vaccine makes the "observation option" for nonsevere ARS more palatable, because *Pneumococcus* is presumably the main cause of complications. Similar to otitis media, the patient is given a "safety net" prescription, but is told to wait several days before filling it, to give symptoms a chance to resolve on their own.

Summary

The successful management of RS in children relies on careful diagnosis, recognition of causative factors, and judicious yet adequate antibiotic usage. Refractory cases will require surgical therapy, with adenoidectomy as the first-line intervention and ESS reserved for those cases refractory to adenoidectomy. This overall approach will improve quality of life and prevent complications in children with RS.

References

[1] Cunningham MJ, Chiu EJ, Landgraf JM, Gliklich RE. The health impact of chronic rhinosinusitis in children. Arch Otolaryngol Head Neck Surg 2000;126:1363–8.

[2] Lazar RH, Younis RT, Parvey LS. Comparison of plain radiographs, coronal CT and intraoperative findings in children with chronic sinusitis. Otolaryngol Head Neck Surg 1992;107:29–34.

[3] Schwartz RH, Pitkaranta A, Winther B. Computed tomography imaging of the maxillary and ethmoid sinuses in children with short-duration purulent rhinorrhea. Otolaryngol Head Neck Surg 2001;124:160–3.

[4] Aitken M, Taylor JA. Prevalence of clinical sinusitis in young children followed up by primary care pediatricians. Arch Pediatr Adolesc Med 1998;152:244–8.

[5] Schoem SR, Zalzal GH. Sinusitis in children. Curr Opin Otolaryngol Head Neck Surg 1999;7:14–9.

[6] Shapiro GG, Rachelevsky GS. Introduction and definition of sinusitis. J Allergy Clin Immunol 1992;90:417–8.

[7] Nguyen KL, Corbett ML, Garcia DP. Chronic rhinosinusitis among pediatric patients with chronic respiratory complaints. J Allergy Clin Immunol 1993;92:824–30.

[8] Shapiro GG, Virant FS, Furukawa CT, Pierson WE, Bierman CW. Immunologic defects in patients with refractory sinusitis. Pediatrics 1991;87:311–6.

[9] Lee D, Rosenfeld RM. Sinonasal symptoms and adenoid bacteriology. Otolaryngol Head Neck Surg 1997;116:301–7.

[10] Vandenberg SJ, Heatley DG. Efficacy of adenoidectomy in relieving symptoms of chronic sinusitis in children. Arch Otolaryngol Head Neck Surg 1997;123:675–8.

[11] Walke M, Shankar L, Hawke M, Takeno S. Maxillary sinus hypoplasia, embryology, and radiology. Arch Otolaryngol Head Neck Surg 1993;119:1353–7.

[12] Phipps CD, Wood E, Gibson WS, Cochran WJ. Gastroesophageal reflux contributing to chronic sinus disease in children. Arch Otolaryngol Head Neck Surg 2000;126:831–6.

[13] Contencin P, Narcy P. Nasopharyngeal monitoring in infants and children with chronic rhinopharyngitis. Int J Pediatr Otorhinolaryngol 1991;22:249–56.

[14] Beste DJ, Conley SF, Brown CW. Gastroesophageal reflux complicating choanal atresia repair. Int J Pediatr Otorhinolaryngol 1994;29:51–8.

[15] Bothwell MR, Parsons DS, Talbot A, Barbero GJ, Wilder B. Outcome of reflux therapy on pediatric chronic sinusitis. Otolaryngol Head Neck Surg 1999;121:255–62.

[16] Lund VJ, Neijens HJ, Clement PA. The treatment of chronic sinusitis: a controversial issue. Int J Pediatr Otorhinolaryngol 1995;32(Suppl):521–32.

[17] Teknst N, Meton R, Chasse T, Balrcia G, Dickersin G. New developments in the diagnosis of Kartagener's syndrome. Otolaryngol Head Neck Surg 1997;116:68–74.

[18] Chapelin C, Coste A, Reinert P, Boucherat M, Millepied MC, Poron F, et al. Incidence of primary ciliary dyskinesia in children with recurrent respiratory diseases. Ann Otol Rhinol Laryngol 1997;106:854–8.

[19] Rosenstein BJ, Cutting GR. The diagnosis of cystic fibrosis. J Pediatr 1998;132:589–95.

[20] Strong TV, Smit LS, Turpin SV, Cole JL, Hon CT, Markiewicz D, et al. Cystic fibrosis gene mutation in two sisters with mild disease and normal sweat electrolyte levels. N Engl J Med 1994;325:1630–4.

[21] Augarten A, Kerem BS, Yahav Y, Nomain S, Rivlin Y, Tal A, et al. Mild cystic fibrosis and normal or borderline sweat test in patients with the 3849 + 10kb C→T mutation. Lancet 1993; 342:25–6.

[22] Rosenfeld RM. Sinusitis in children. In: Gates GA, editor. Current therapy in otolaryngology head and neck surgery. 6th edition. St. Louis (MO): Mosby; 1998. p. 354–8.

[23] Orenstein SR, Izadnia F, Khan S. Gastroesophageal reflux disease in children. Gastroenterol Clin North Am 1999;28(4):947–69.

[24] Polmar SH. The role of the immunologist in sinus disease. J Allergy Clin Immunol 1992;90: 511–5.

[25] Woroniecka M, Ballow M. Primary immune deficiencies: presentation, diagnosis and management. Pediatr Clin North Am 2000;47(6):1211–24.

[26] Madgy DN, Haupert MS. Management of pediatric rhinosinusitis. Curr Opin Otolaryngol Head Neck Surg 2000;8:469–76.

[27] Carron JD, Derkay CS. Pediatric rhinosinusitis: is it a surgical disease. Curr Opin Otolaryngol Head Neck Surg 2001;9:61–6.

[28] Garbutt JM, Goldstein M, Gellman E, Shannon W, Littenberg BL. A randomized, placebo-controlled trial of antimicrobial treatment for children with clinically diagnosed acute sinusitis. Pediatrics 2001;107:619–25.

[29] Wald ER, Chiponis D, Ledesma-Median J. Comparative effectiveness of amoxicillin and amoxicillin-clavulanate potassium in acute paranasal sinus infections in children. Pediatrics 1986;77: 795–800.

[30] Sinus and Allergy Health Partnership. Antimicrobial treatment guidelines for acute bacterial rhinosinusitis. Otolaryngol Head Neck Surg 2000;123(Suppl 1):S1–32.

[31] Agency for Health Care Policy and Research (AHCPR). Diagnosis and treatment of acute bacterial rhinosinusitis. Rockville (MD): AHPRC; 1999.

[32] Brook I, Gooch WM, Jenkins SG, Pichichero ME, Reiner S, Sher L, et al. Medical management of acute bacterial sinusitis: recommendations of a clinical advisory committee on pediatric and adult sinusitis. Ann Otol Rhinol Laryngol 2000;109:2–20.

[33] American Academy of Pediatrics Subcommittee on Management of Sinusitis and Committee on Quality Improvement. Clinical practice guideline: management of sinusitis. Pediatrics 2001; 108(3):798–808.

[34] Wald ER. Sinusitis. Pediatr Ann 1998;27:811–8.

[35] Barlan IB, Erkan E, Bakir M, Berrak S, Basaran M. Intranasal budesonide spray as an adjunct to oral antibiotic therapy for acute sinusitis in children. Ann Allergy Asthma Immunol 1997;78: 598–601.

[36] Meltzer EO, Orgel HA, Backhaus JW, Busse WW, Druce HM, Metzger WJ, et al. Intranasal flunisolide spray as an adjunct to oral antibiotic therapy for sinusitis. J Allergy Clin Immunol 1993;92:812–23.

[37] Bende M, Arfors KE, Stierna P, Fukami M, Mark J, Intaglietta M. Effect of oxymetolazine nose drops on acute sinusitis in the rabbit. Ann Otol Rhinol Laryngol 1996;105:222–5.

[38] Boek WM, Keles N, Graamans K, Huizing EH. Physiologic and hypertonic saline solutions impair ciliary activity in vitro. Laryngoscope 1999;109:396–9.

[39] McCormick DP, John SD, Swischuk LE, Uchida T. A double blind, placebo-controlled trial of decongestant-antihistamine for the treatment of sinusitis in children. Clin Pediatr 1996; 35(9):457–60.

[40] Chan KH, Winslow CP, Levin M, Abzug MJ, Shira JE, Liu AH, et al. Clinical practice guidelines for the management of chronic sinusitis in children. Otolaryngol Head Neck Surg 1999;120: 328–34.

[41] Otten HW, Antvelink JB, Ruyter De Wildt H, Rietema SJ, Siemelink RJG, et al. Is antibiotic treatment of chronic sinusitis effective in children? Clin Otolaryngol 1994;19:215–7.

[42] Otten FWA. Conservative treatment of chronic maxillary sinusitis in children. Acta Otorhinolaryngol Belg 1997;51:173–5.

[43] Parsons DS. Chronic sinusitis: a medical or surgical disease? Otolaryngol Clin North Am 1996; 29:1–9.

[44] Rosenfeld RM. Pilot study of outcomes in pediatric rhinosinusitis. Arch Otolaryngol Head Neck Surg 1995;121:729–36.

[45] Clement PAR, Bluestone CD, Gordts F, Lusk RP, Otten FWA, Goossens H, et al. Management of rhinosinusitis in children. Int J Pediatr Otorhinolaryngol 1999;49(Suppl 1):S95–100.

[46] Leiberman A, Dagan R, Leibovitz E, Yagupsky P, Fliss DM. The bacteriology of the nasopharynx in childhood. Int J Pediatr Otorhinolaryngol 1999;49(Suppl 1):S151–3.

[47] Dagan R, Hoberman A, Johnson C, Leibovitz EL, Arguedas A, Rose FV, et al. Bacteriologic and clinical efficacy of high dose amoxicillin/clavulanate in children with acute otitis media. Pediatrics 2001;20(9):829–37.

[48] Hyde TB, Gay K, Stephens DS, Vugia DJ, Pass M, Johnson S. Macrolide resistance among invasive Streptococcus pneumoniae isolates. JAMA 2001;286:1857–62.

[49] Bottenfield GW, Burch DJ, Hendrick JA, Schaten R, Rowinski CA, Davies JT. Safety and tolerability of a new formulation of amoxicillin/clavulanate in the empiric treatment of pediatric acute otitis media caused by drug-resistant Streptococcus pneumoniae. Pediatr Infect Dis J 1998;17(10):963–8.

[50] Berry V, Thorburn CE, Knott SJ, Woodnutt G. Bacteriological efficacies of three macrolides compared with those of amoxicillin-clavulanate against Streptococcus pneumoniae and Haemophilus influenzae. Antimicrob Agents Chemother 1998;42(12):193–9.

[51] Schenkel EJ, Skoner DP, Bronsky EA. Absence of growth retardation in children with perennial allergic rhinitis after one year treatment with mometasone furoate aqueous nasal spray. Pediatrics 2000;105:e22.

[52] Brannan MD, Herron JM, Affrime MB. Safety and tolerability of once-daily mometasone furoate aqueous nasal spray in children. Clin Ther 1997;19:1330–9.

[53] Poole MD. Pediatric sinusitis is not a surgical disease. Ear Nose Throat J 1992;71(12):622–3.

[54] Ramadan HH. Adenoidectomy vs. endoscopic sinus surgery for the treatment of pediatric sinusitis. Arch Otolaryngol Head Neck Surg 1999;125:1208–11.

[55] Hebert RL, Bent JP. Meta-analysis of outcomes of pediatric functional endoscopic sinus surgery. Laryngoscope 1998;108:796–9.

[56] Walner DL, Markey R, Jain V, Myer CM. Clinical outcome of pediatric endoscopic sinus surgery. Am J Rhinol 2002;16:151–4.

[57] Mair EA, Bolger WE, Breisch EA. Sinus and facial growth after pediatric endoscopic sinus surgery. Arch Otolaryngol Head Neck Surg 1995;121:547–52.

[58] Senior B, Wirtschafter A, Mai C, Becker C, Belenky W. Quantitative impact of pediatric sinus surgery on facial growth. Laryngoscope 2000;110:1866–70.

[59] Bothwell MR, Piccirillo JF, Lusk RP, Ridenour BD. Long-term outcome of facial growth after functional endoscopic sinus surgery. Otolaryngol Head Neck Surg 2002;126:628–34.

[60] Walner DL, Falciglia M, Willging JP, Myer CM. The role of second-look nasal endoscopy after pediatric functional endoscopic sinus surgery. Arch Otolaryngol Head Neck Surg 1998;124: 425–8.

[61] Buchman CA, Yellon RF, Bluestone CD. Alternative to endoscopic sinus surgery in the management of pediatric chronic rhinosinusits refractory to oral antimicrobial therapy. Otolaryngol Head Neck Surg 1999;120:219–24.

[62] Dagan R, Sikuler-Cohen M, Orly Z, Janco J, Givon-Lavi N, Fraser D. Effect of a conjugate pneumococcal vaccine on the occurrence of respiratory infections and antibiotic use in day-care center attendees. Pediatr Infect Dis J 2001;20(10):951–8.