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An Update of Rhinosinusitis.

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Abstract: Rhinosinusitis is one of most common disease worldwide, affecting the quality of life of the person afflicted by it. Its causes and pathophysiology has been well delineated so far, but its treatment is changing fast due to change in organisms causing it and also emergence of resistance to antimicrobial agents, thus making the management challenging. This article highlights the changing microbial pattern, classification of the disease, its socio-economic burden, medical treatment and ever evolving surgical treatment, with its indications.

Keywords: Rhinosinusitis, Acute Bacterial Rhinosinusitis, Chronic Rhino sinusitis, Paranasal sinus, URTI.

INTRODUCTION: - Rhinosinusitis (RS) is such a common entity although given the wide range of clinical presentation. The multiple potential types & varied etiological & associated factors. This diagnosis often includes several patho – physiologic conditions. In order to better define the incidence & prevalence, the more specific criteria should be applied. The prevalence rate has also been found to very substantially depending on the type of practitioner & method of diagnosis used. Prevalence in children in Primary Care setting with symptoms of on acute URTI is 9to 17% in various studies. The number increases 30% for patients presenting to a general medical clinic & this further increases to 83% of patients presenting with acute URTI having ABRS in an otolaryngology clinic[1,2]. The improper diagnosis & management of RS has given way to emergence of

Antimicrobial resistance & thus the overall increase in prevalence of RS. Up to 50% resistance rates to macrolides & betalactams have been noted. Quality of life with RS is really poor. It is one of the most common reasons for an individual seeking medical care. Recent efforts to evaluate the impact of disease on Quality Of Life (QOL) & the outcome of disease have clarified the importance of such impacts. Glicklich et.al [3] have shown that RS has a significant QOL impact. Even in comparison to chronic debilitating disease such as diabetes mellitus and congestive heart failure. The RS disability index is validated instrument that measures the physical, functional and emotional impact of RS on persons QOL [4,5].

RHINOSINUSITIS:

Sinusitis is defined as group of disorders characterized by inflammation of mucosa of paranasal sinuses due to various etiologies. Since inflammation of sinuses nearly always also involves the nose & nasal mucosa. 'Rhinosinusitis' is now generally preferred term for this condition. A fairly big number of factors, including both host & environment, play a role in development of rhino sinusitis. The most common types of rhinosinusitis are of allergic and viral etiology. Because of complexity of factors associated with rhino sinusitis, the classification & / or definitions of RS is significantly debatable topic. A widely accepted classification & definition was developed by Rhinosinusitis Task Force of the American Academy of Otolaryngology – Head & Neck Surgery [6] and reported by Lanza & Kennedy[7] [table1]. These criteria are based on temporal time frames. The distinctions between ARS, RARS, SRS, CRS & AECRS are based on the temporal differences in the presentation & in some cases, on clinical presentations. Being a common & vexing clinical entity, most of the discussion here will be focused on community – acquired acute bacterial rhino sinusitis. Viral rhinosinusitis has been identified by CT scan in approximately 90% of patients with a viral upper respiratory infection. A small percentage of these patients may develop secondary bacterial infection.

Since URTI symptoms may mimic those of an episode of rhinosinusitis , a minimal duration of symptoms is recommended prior to determination of an acute bacterial rhinosinusitis, typically 5-7 days (10days to 4weeks). A typical viral URTI improves within 5-7 days & usually largely resolves by 10 to 14 days. Bacterial super infection or ABRS can be considered when symptoms persist beyond 10 days or worsen after 5-7 days. Recurrent acute infections are defined by 4 or more episodes per year , with each lasting greater than 7-10 days & absence of signs & symptoms that would suggest chronic rhinosinusitis. Subacute Bacterial RS is defined when duration of symptoms range between 4 weeks to 12 weeks & those of greater than 12 weeks are considered as CRS. Table 1[7].

Table :1

TYPE	DURATION
ARS (Acute RS)	7 Days $\leq$ 4 weeks
RARS (Recurrent Acute RS)	$\geq$ 4 episodes of ARS per year
SABRS (Sub Acute Bacterial RS)	4 – 12weeks
CRS (Chronic RS)	>12wks
AECRS (Acute Exacerbation of Chronic RS)	Sudden worsening of CRS with return to baseline after treatment.

Even though inflammation of mucosal linings is present in all cases of RS, the focus is on those patients who develop the symptoms of inflammation. Table No.2. depicts group of symptoms to be applied for clinical diagnosis[6].

Table 2: Rhino sinusitis symptoms and signs (requires two major factors or one major and two minor).

Major	Minor
1. Facial Pain / pressure	Headache/fatigue
2. Facial congestion	Fever (non acute)
3. Nasal blockage/obstruction	Halitosis
4. Nasal Discharge/ purulence/ discolored post nasal drip	Dental Pain
5. Hyposmia / anosmia	Cough
6. Fever (Acute RS only)	Ear pain / Fullness

DIFFERENTIAL DIAGNOSIS OF RHINO SINUSITIS:

- Infectious Rhinitis
    1. Viral URTI.
    2. Bacterial RS.
- Allergic rhinitis (seasonal or perennial)
- Nonallergic rhinitis (idiopathic rhinitis) which includes
    1. Vasomotor rhinitis.
    2. Eosinophilic nonallergic rhinitis(NARES).
    3. Endocrine rhinitis i:e hormonal, pregnancy and hypothyroid states.
    4. Granulomatous rhinitis (Wegner's).
- Rhinitis medicamentosa.

- Anatomic deformities.
- Nasal septal obstruction.
- Nasal turbinate hypertrophy.
- Choncha bullosa.
- Tumors.

ANATOMY & PATHOPHYSIOLOGY OF RHINOSINUSITIS: -

The sinuses develop from nasal chambers. Only maxillary and ethmoid sinuses can be identified at birth. For the first 6 years of life the growth of sinuses is slow & the ostium of sinuses is small. The shapes of sinuses become irregular after age of 6-7 years because of distorting effect by developing adjacent structures, including other sinuses. The adult size and shape of sinuses is achieved by 12 – 14 years of age. The maxillary, frontal & sphenoidal sinuses are larger & paired bony chambers, while anterior & posterior ethmoid sinuses consist of labyrinth of small bony cells.

Maxillary and Frontal sinuses & anterior ethmoid air cells drain into middle meatus. The posterior ethmoid air cells & sphenoid sinus open/drain into superior meatus & sphenoethmoidal recess.

Normally the sinuses have sterile environment. New researches show that the Para Nasal Sinuses (PNS) are a large reservoir for Nitric Oxide(NO) which improves mucocilliary function. NO is also supposed to have bacteriostatic function as well as antiviral function. NO production increased in acute & chronic inflammation of nose & PNS.

Typically ABRS develops following acute viral URTI. Inflammatory response is expected sequel of this infections process. This inflammatory response leads to mucosal swelling with occlusion or obstruction of sinus ostia. A reduction in oxygen tension occurs which reduces mucociliary transport & transduction of fluid into sinuses[8]. These changes in the nasal – sinus environment lead to mucostasis & bacterial colonization, and hence infection [9]. The role of allergies has been strongly suggested but not proven. Many cells & proteins that are involved with inflammatory response have been implicated and are being investigated to their roles in rhinosinusitis. These inclued eosinophils, neutrophils, mast cells, T & B cells, immunoglobulins, interleukins, TNF, Major Basic Protiens & number of other mediators. Presence of specific inflammatory mediators very based on inciting factor in inflammation[8,10,11].

Histopathologically ARS shows predominance of neutrophils, along with exudative process, visible areas of necrosis, ulceration & hemorrhage are seen[7,12]. In CRS, a proliferative process is present with lymphocytes, plasma cells & eosinophils being present. Evidences of fibrosis of lamina propria are also seen [7,8,10,11,31].

Most common bacteria associated with ABRS in adults are Streptococcus Pneumoniae (20%-45%) & H influenzae (22%-35%) & M. catarrhalis (3%-10%). In children Strept. Pneumoniae (30%-43%), H Influenzae (20%-28%). & M catarrhalis

(20%-28%) are most commonly associated bacteria. Out of these M catarrhalis is largely a self limited pathogen[7,13,14].

As far as Pathogenesis of CRS is concerned it is thought to be an inflammatory disease & it may or may not involve pathogenic microbes. In those patients with CRS, who do have potential pathogenic bacteria, the most common organisms are staph species (55%), staph aureus (20%)[7,14]. Prevalence of Enterobacteriaceae organisms [15,16]. Anaerobes [17] & fungi [11] also been seen.

FIGURE 1 – Ranges of Prevalence of the Major Pathogens Associated With Acute Bacterial Rhinosinusitis in Adults

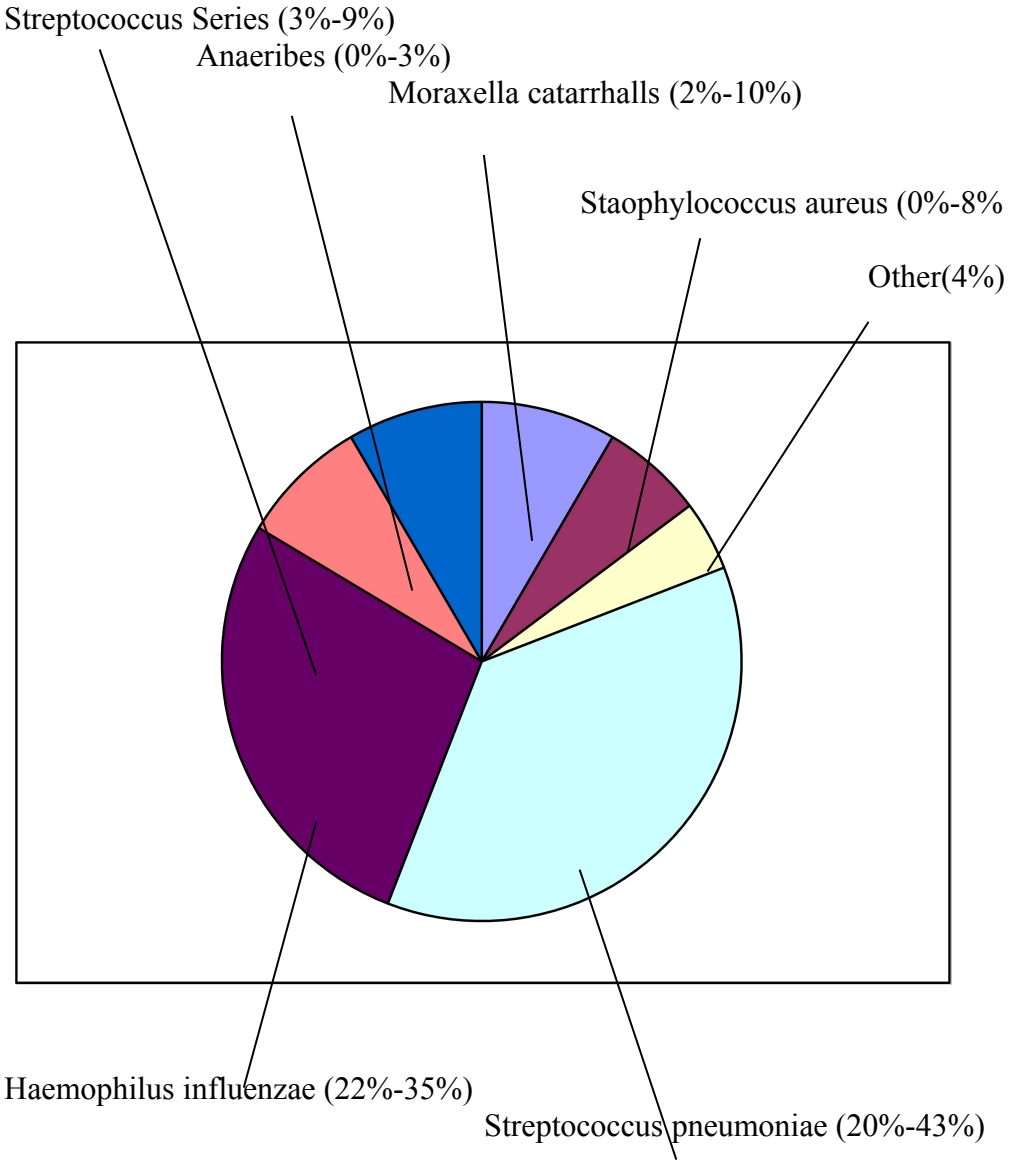
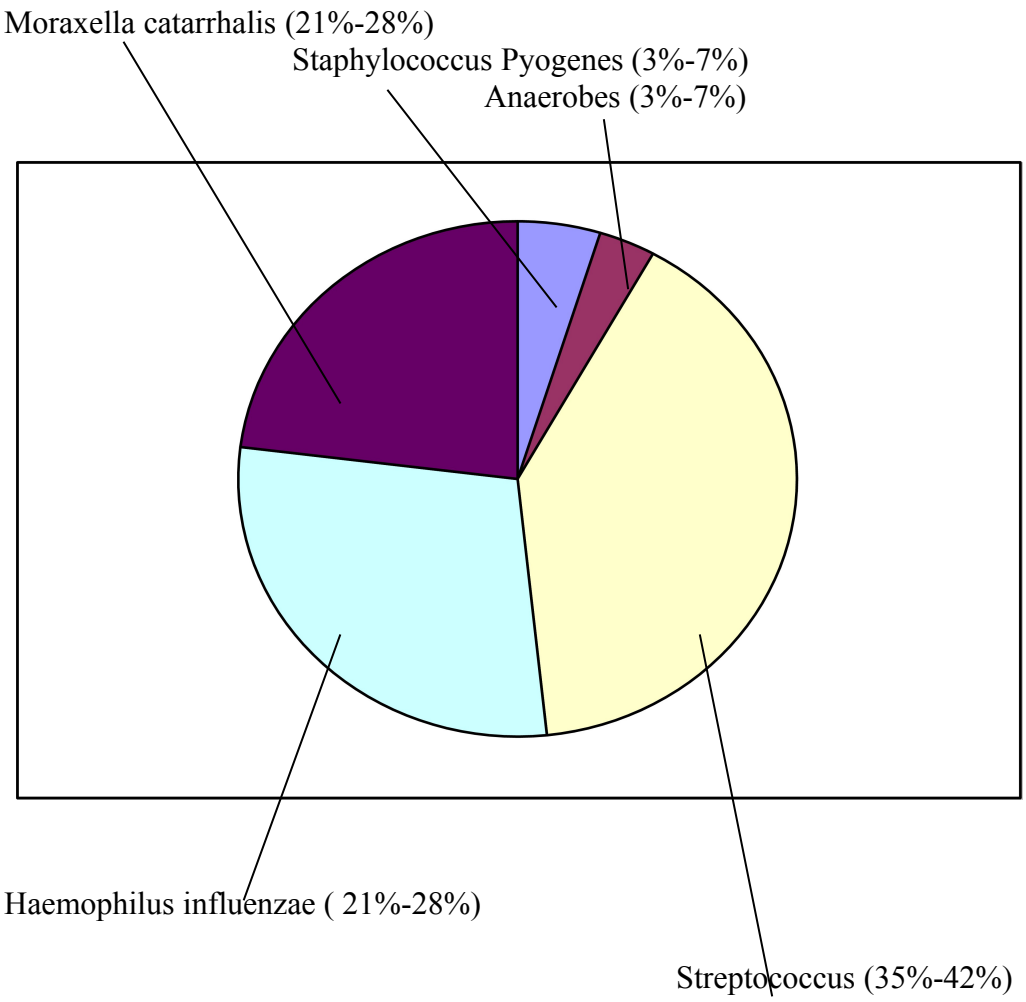


FIGURE 2– Microbiology of Acute Bacterial Rhinosinusitis in Children



DIAGNOSIS: - The best of diagnosis of ABRS can be made on clinical ground & criteria (table 2). The most cost effective methods of diagnosis differ at different prevalence rates. The diagnosis can be made on empiric basis & symptoms if prevalence rates are high such as in specialty practices. In low prevalence areas clinical criteria including symptoms & physical findings are preferred [1,2]. The diagnosis for research usually needs more objective information. To identify specific Pathogen for research a maxillary sinus tap with culture is recommended(*GOLD standard for diagnosis of ABRS*) [15,18]. The role of endoscopic guided middle meatus culture is under research. If one has to make diagnosis of bacterial rhino sinusitis, the current accepted reference standard

for culture is more than 1X 10^4 CFU/ ml in sinus aspirate[19]. Lower counts potentially represent early infection.

In CRS & SRS definitive methods for diagnosis has yet not been determined. The recommended time frame for greater than 12week for CRS & between 4-12 weeks for SRS have been commonly adopted. Thus appropriate history, proper physical examination & testing will confirm the diagnosis. A number of tests can be used to establish the diagnosis of rhinosinusitis but these are more important in chronic rather than acute rhinosinusitis Nasal endoscopy is an important tool to assess the nasal anatomy, confirm drainage & evaluate treatment response. X-rays have been used for all types of rhinosinusitis but are not cost effective in diagnosis of ABRS[1,2]. At present time the preferred radiological test is sinus CT scan but it cannot distinguish between inflammation & infection .CT scan are not recommended in ABRS as they are not cost effective for this indication[1,2,20]. But are good for evaluation of suspected impending complications such as orbital or intracranial involvement. CT scan is good for assessing the severity of disease or response to treatment in CRS. Lund & Mackay grading (1993) of CT scan PNS is most commonly recommended as staging system to asses severity of rhino sinusitis[21]. It has been recently recommended that either a CT scan or endoscopic evaluation of nose preferably with photo or video documentation should be part of any prospective clinical trial in CRS[10].

TREATMENT:

Medical management of RS can be simplified into three groups-antimicrobial, anti-inflammatory and mechanical. It is breakdown treatment from each group and combine them when appropriate into a comprehensive treatment plan. Also, it is important to consider the side effect of each therapy, and weigh them with the patient's symptoms severity and other medical conditions. In general, medical management of RS should include culture directed(or broad spectrum) antibiotics, a nasal steroid spray, saline irrigation and decongestant. Strong consideration should also be given for steam inhalation.

Table 3[22] – Prevalence of Cross – Resistance Between Penicillin and Various Antibiotic Classes Among Strains of Penicillin – Nonsusceptible Strains of Streptococcus pneumonia

	% of Strains Resistant			
Class / Agent	**Pen – S**	**Pen – I**	**Pen – R**	**All**
Macrolides	6	49	76	32
Clindamycin	1	14	28	10
TMP/SMX	14	57	91	43
Doxycycline	4	25	55	22

Abbreviations: MIC, minimum inhibitory concentration ; Pen – I, penicillin – intermediate (penicillin MIC 0.12 mg/ mL to 1 mg/mL); Pen – R, Penicillin – resistant (penicillin MIC ≥2 mg/ mL); Pen – S, penicillin – susceptible (penicillin MIC ≤0.06 mg/mL); TMP/SMX, trimethoprim / sulfamethoxazole.

TABLE 4[23] – Antimicrobial Agents Stratified by Pharmacodynamic Profile Against Streptococcus pneumoniae and Haemophilus influenza

Antimicrobial Agnet	Achieves Pharmacodynamic Target*				
	S pneumoniae			H influenza	
	Penicillin-Susceptible	Penicillin-Intermediate	Penicillin-resistant	ß-Lactamase - negatic	ß-Lactamase – positive
ß – Lactams					
Amoxicillin †	√	√	√	√	
Amoxicillin / clavulanate*	√	√	√	√	√
Cefdinir	√	±		√	√
Cefpodoxime	√	√		√	√
Ceftrizxone	√	√	√	√	√
Cefuroxime	√	√		√	√
Folate Inhibitors					
TMP/SMX	√	±		√	√
Ketolides					
Telithromycin	√	√	√	±	±
Macrolides					
Azithromycin	√	±		±	±
Clarithromycin	√	±		±	±
Erythromycin	√	±			

Fluoroquinolones					
Gatifloxacin	√	√	√	√	√
Gemifloxacin	√	√	√	√	√
Levofloxacin	√	√	√	√	√
Maxifloxacin	√	√	√	√	√
Tetracyclines					
Doxycycline	√	√	±		
Abbreviations: AUC, area under the curve; MIC, minimum inhibitory concentration; TMP/SMX, trimethoprim/ sulfamethoxazole.					
Key: √, Adequate pharmacodynamic profile using conventional dosing in patients with normal renal and hepatic function; ±, borderline pharmacodynamic profile using conventional dosing in patients with normal renal and hepatic function.					
* ß – Lactams and macrolides: T > MIC > 40% of the dosing interval. Fluoroquinolones: 24 – h AUC/MIC ratio > 100 – 125 for H influenzae and > 30 – 50 for S pneumoniae. † High – dose amoxicillin					

TABLE 5[22] – Mechanisms of Action of Commonly Used Antimicrobials for Community – Acquired Respiratory Tract Infections	
Target	Examples
Cell – wall – active agents	ß – Lactams (penicillins, cephalosporins)
Protein synthesis inhibitors (ribosome)	Macrolides, Lincosamides (eg, clindamycin), tetracyclines, (eg, tetracycline, doxycycline), ketolides.
DNA replication inhibitors	Fluoroquinolones
Folic acid metabolism inhibitors	Trimethoprim / sulfamethoxazole

TABLE 6 – Antimicrobial Agents for Acute Bacterial Rhino sinusitis				
Generic Name	Trade Name	Typical Adult Dosage Regimen	Typical Pediatric Dosage Regimen	Available Dosage(mg)
ß – Lactams				
Amoxicillin	Amoxil Generics	875 mg bid x 10 d	> 3 mo: 45 mg/kg/d given q12h x10 d	250,500,875*
Amoxicillin / Clavulanate	Augmentin	500-2000mg bid x 10 d	≥ 12 wks: 45mg/kg given q 12h x 10 d	250/125,500/125, 875/125*
	Augmentin ES – 600	N/A	90 mg/kg/d divided q 12h x 10 d	600 mg/5mL
	Augmentin XR	2000/125 mg bid x 10 d	N/A	1000 / 62.5
Cefdinir	Omnicef	300 mg bid x 10 d or 600 mg qd x 10 d	6 mo – 12 yrs: 7 mg/kg q12h or 14 mg/kg q24h x 10 d	125,250,300*
Cefpodoxime	Vantin	200 mg bid x 10 d	2’ mo – 11 yrs: 5 mg/kg/d given q12h x 10 d	100, 200*
Ceftin	Cefuroxime	250 mg bid x 10 d	3 mo – 12 yrs; 15 mg/kg bid x 10 d	125,250,500*
Ceftriaxone	Rocephin	1-2 g 1M qd x 3-10 d	50 mg/kg IM x 3 d (minimum)	500,1 g, 2 g
Folate Inhibitors				
TMP/SMX	Bactrim, Septra	160/800 bid x 10 d	≥ mo: 4 mg/kg TMP and 20 mg/kg SMX given q12h x 10 d	80/400, 160/800 (DS)*
Macrolides / Azalides/ Ketolides				
Azithromycin	Zithromax	500mg qd x 3 d	≥6 mo: 10 mg/kg qd x 3 d	250,500*
Clarithromycin	Biaxin	500 mg q12h x 14 d	≥ mo: 7.5 mg/kg given q12 x 10 d	250,500*
	Biaxin XL	1000 mg qd x 14 d	N/A	500
Clindamycin	Cleocin	150 – 300 mg q 6h x 10 d	Birth – 16 yrs: 8 – 16 mg/kg/dx 10 d	75,150,300*
Telithromycin	Ketek	800 qd x 5 d	N/A	400
Respiratory Quinolones				
Gatifloxacin	Tequin	400mg qd x 10 d	N/A	200, 400
Gemifloxacin	Factive	320 mg qd x 10 d	N/A	320
Levofloxacin	Levaquin	500-750 mg qd x 10 d	N/A	500, 750
Moxifloxacin	Avelox	400 mg qd x 10 d	N/A	400
Tetracyclines				

Doxycycline	Vibramycin, others	100 mg qd given q12h x 10 d	≥8 yrs; ≤ 100 lbs; 1 mg/ lb bid on day 1, then 1 mg/lb or 0.5 mg/lb bid x 10 d	50,75,100*
Abbreviations DS, double strength; N/A, not approved for use; TMP / SMX, trimethoprim/ sulfamethoxazole.				
This table provides typical dosage regimen.				
* Some dosages are also available in suspension form.				

TABLE 7 – Recommended Antibiotic Therapy for Adults With ABRS

Initial Therapy	Calculated Clinical Efficacy (%)*	Calculated Bacteriologic Efficacy (%)*	Switch – Therapy Options (No Improvement or Worsening After 72 Hours)†
Mild disease † with no recent antimicrobioal use (past 4-6 weeks)$			
Amoxicillin / Clavulanate (1.75 - 4 g/250 mg/d)$II	90 – 91	97 - 99	Gatifloxacin, levofloxacin, Moxifloxacin Amoxicilli /clavulanate4g/250 mg Ceftriaxone Combination Therapy $
Amoxicillin (1.5-4 g/d)II	87 – 88	91 – 92	
Cefpodoxime proxetil	87	91	
Cefuroxime axetil	85	87	
Cefdinir	83	85	
ß – Lactam allergic#			
TMP / SMX	83	84	Gatifloxacin, levofloxacin, Moxifloxacin Rifampin plus clindamycin
Doxycycline	81	80	
Azithromycin, Clarithromycin, erythromycin	77	73	
Telithromycin	See footnote. **	73	
Mild disease# with recent antimicrobial use (past 4-6 weeks) or moderate disease $			
Gatifloxacin, Levofloxacin, moxifloxacin	92	100	

Amoxicilli /clavulanate (/250 mg)	91	99	Reevaluate patient [$$]
Ceftriaxone	91	99	
(Combination therapy)[$]	N/A	N/A	
ß – Lactam allergic [#]			
Gatifloxacin, levofloxacin, moxifloxacin	92	100	Reevaluate patient [$$]
Clindamycin and rifampin[††]	N/A	N/A	

Abbreviations : ABRS, acute bacterial rhinosinusitis; CT, computed tomography; N/A, not available; TMP/SMX, trimethoprim / sulfamethoxazole.

- Clinical and bacterial efficacy (ie, linical and microbiologic adequacy) is represented by the calculation from the poole Therapeutic Outcome Model using the mean values of two surveillance data sets: the US component of the Alexander project (1998 to 2001) and SENTRY surveillance data. These values to not guarantee clinical success or failure. The Poole Therapeutic outcome model has not been clinically validated.

† When a change in antibiotic therapy is made, the clinician should consider the limitations in coverage of the intitial antibiotic. The respiratory fluoroquinolones (gatifloxacin, Levofloxacin, and moxifloxacin), ceftriaxone, and amoxicillin/ clavulanate (4 g/250 mg) currently have the best coverage for both Streptococcus pneumoniae and Haemophilus influenzae. The terms mild and moderate are designed to aid in selecting antibiotic therapy.

+ The difference in severity of disease does not imply the presence or absence of antimicrobial resistance. Rather, this terminology indicates the relative degree of acceptance of possible therapeutic failure and the likelihood of achieving spontaneous resolution of symptoms. The determination of disease severity lies with the clinician's evaluation of the patient's history and clinical presentation. Severe, life-threatening infection, with or without complications, is not addressed in these guidelines.

$ Prior antibiotic therapy within 4 to 6 weeks is a risk factor for infection with resistant organisms. Antibiotic choices should be based on this and other risk factors.

II The total daily dose of amoxicillin and the amoxicillin component of amoxicillin / clavulanate can vary from 1.5 to 4 g/day. Lower daily doses (1.5 g/day) are more appropriate in mild disease in patients with no risk factors for infection with a resistant pathogen (including recent antibiotic use). Higher daily doses (4 g/day) may be advantageous in areas with a high prevalence of penicillin – resistant S pneumoniae or drug – resistant S pneumoniae, for patients with moderate disease, for patients who may need better H influenzae coverage or for patients with risk factors for infection with a resistant pathogen. There is a greater potential for treatment failure or resistant pathogens is these patient groups.

II Based on in vitro spectrum of activity; combination therapy using appropriate gram – positive and gram – negative coverage may be appropriate. Examples of combination therapy regimens include high – dose amoxicillin (4 g/day) or clindamycin plus cefixime, or high –

dose amoxicillin (4 g/day) or clindamycin, plus rifampin. There is no clinical evidence at this time, however, of the safety or efficacy of these combinations.

Cephalosporins should be considered initially for patients with penicillin intolerance / non –Type I hypersensitivity reactions (eg, rash). TMP/SMX, doxycycline, macrolides, azalides, and ketolides are not recommended unless the patient is ß – lactam allergic. Their effectiveness against the major pathogens of ABRS is limited, and bacterial failure of 20% to 25% is possible. A respiratory fluoroquinolone (eg, gatifloxacin, lovofloxacin, moxifloxacin) is recommended for patients who have allergies to ß – lactams or who have recently failed other regimens.

** Further Pharmacokinetic/ pharmacodynamic data on this compound is needed.

†† Rifampin is a well – known inducer of several cytochrome P450 isoenzymes and therefore has a high potential for drug interactions.

++ Reevaluation is necessary because the antibiotics recommended for initial therapy provide excellent activity against the predominant ABRS pathogens, including S pneumoniae or H influenzae. Additional history, physical examination, cultures, and / or CT scan may be indicated, and the possibility of other less common pathogens considered.

Chronic Rhinosinusitis

Chronic rhinosinusitis (CRS) is an umbrella term for a number of pathologic conditions in which inflammation of the nose and paranasal sinuses is the final common pathway. These include:

- Allergy
- Immunodeficiency states
- Neoplasm's
- Virus
- Bacteria
- Fungi
- Genetic / congenital abnormalities
- Mucociliary dysfunction
- Noxious chemicals
- Pollutants
- Smoking.

Subgroups of these categories, such as super antigen formation, bacterial biofilms, and allergic fungal disease, have been identified.

The diagnosis of CRS has been defined by consensus in order to develop a framework of terminology that clinicians and researchers can utilize for standardized

communication (Table). CRS is when symptoms have been present for 12 weeks and there is physical evidence (usually by nasal endoscopy) of mucosal swelling, nasal discharge, or polyps. Mucosal abnormalities of the middle meatus or bulla are also concrete signs of inflammatory disease. Generalized or localized edema, erythema, or granulation tissue can be caused by other rhinologic diseases, such as allergic rhinitis, and therefore requires imaging confirmation.

TABLE 8 – Measures for Diagnosing Chronic Rhinosinusitis for Adult Clinical Care

- Duration of disease is qualified by continuous symptoms for > 12 consecutive weeks or > 12 weeks of physical findings.*
- One of these signs of inflammation must be present and identified in association with ongoing symptoms consistent with CRS.
  - Discolored nasal drainage arising from the nasal passage, nasal polyps, or polypoid swelling as identified on physical examination with anterior rhinoscopy or nasal endoscopy. Anterior rhinoscopy should be performed in the decongested state.
  - Edema or erythema of the middle meatus or ethmoid bulla as identified by nasal endoscopy.
  - Generalized or localized erythema, edema, or granulation tissue. If it does not involve the middle meatus or ethmoid bulla, radiologic imaging is required to confirm diagnosis #.
- Imaging modalities for confirming the diagnosis:
  - CT scan: demonstrating isolated or diffuse mucosal thickening, bone changes, air-fluid level.
  - Plain sinus radiograph: Water's view revealing mucous membrane thickening of >5 mm or complete opacification of one or more sinuses. An air – fluid level is more predictive of acute rhino sinusitis but may also be seen in CRS**.
  - MRI is not recommended as an alterative to CT for routine diagnosis of CRS because of its excessively high sensitivity and lack of specificity.

Abbreviations: CRS, chronic rhino sinusitis; CT, computed tomography; MRI, magnetic resonance imaging.

*Signs consistent with CRS will support the symptom time duration
#Other chronic rhinologic conditions such as allergic rhinitis can result in such findings, and therefore they may not be associated with rhino sinusitis. It is recommended that a diagnosis of rhino sinusitis require radiologic confirmation under these circumstances
**A plain sinus radiograph without the equivocal signs listed here is not considered diagnostic. Aside from an air-fluid level, plain sinus radiographs have low sensitivity and specificity.

The best radiologic study for the evaluation of the sinuses is a computed tomography (CT) scan. Plain film sinus radiographs may be helpful in some instances for confirming the diagnosis of symptomatic patients with equivocal physical findings. Magnetic resonance imaging (MRI) scans may be too sensitive. Given the role of CT Scanning in identifying mucosal abnormalities in the sinuses, MRI scans are not currently recommended.

A treatment paradigm for CRS is difficult, since there are numerous underlying etiologies, since inflammation is present, anti – inflammatory therapy is key in the clinician's arsenal. Frequently, a "Shot-gun" approach is undertaken. One regimen might include antibiotic therapy for 10 to 21 days, systemic steroids, leukotriene modifiers, nasal irrigations (such as saline with antifungal, antibacterial), and nasal steroids. Repeat evaluation of the patient with or without CT scan is performed a number of weeks after this "maximal" therapy.

SURGICAL MANAGEMENT:

Surgery is a last resort and is reserved for patients who have not responded to therapy. Obviously, neoplasm's and certain other pathologies are absolute indications for surgical intervention. Surgery is usually performed via a minimally invasive endoscopic approach. The use of computer – aided surgical devices has revolutionized the manner in which endoscopic surgery is performed.

Despite the fact that maximal medical therapy is indicated in all cases of RS and many cases do respond to it, there still exists a number of patients who improve only after surgical management[25,26,27].

From surgical drainage of sinuses, being practiced even in preantibiotic era, and conventional open sinus surgeries to the presently practiced minimally invasive endoscopic sinus surgeries, sinus surgery has evolved a long way. Being a highly prevalent disease worldwide [28,29] that result in a huge financial and disease burden globally[28,30]. All over the world approximately 5 lakhs of surgical procedures are performed annually on RS patients [27].

TABLE 9: Procedures for CRS.

SINUS	*CONSERVATIVE*	*RADICAL*
Maxillary	Antral washout Intranasal antrostomy Middle meatal(endoscopic) Inferior meatal	Caldwell-Luc
Frontoethmosphenoid	Draf 1-3 endonasal drainage Trephination of frontal sinus or sphenoid washout Intranasal ethmoidectomy FESS Transantral ethmoidectomy(Jansen-Horgan)	External frontoethmosphenoidectomy(Lynch-Howrath, Patterson) Midline septectomy (Lothrop; rhinofrontal sinuseptotomy). Osteoplastic flap(with or without obliteration). Cranialisation of frontal sinus.

Table: 10: Indication for surgical management:

1. CRS not responding to optimum medical management.
2. Recurrent acute RS.
3. Allergic fungal RS.
4. Sinonasal polyposis.
5. Acute RS with complications.
6. Sinus mucoceles.
7. Antrochoanal polyp.

Table 11:

Contraindications for surgical treatment:

1. Patients with Extensive polyposis or allergic fungal sinusitis who are not likely to adhere to post operative prolonged medical treatment.
2. Patients chief complains of headache or midfacial pain which is unlikely to be of sinus origin even though CT scan shows sinus opacity.
3. Frail patient or other medical condition making surgery highly risky proposition.
4. Grossly hypoplastic sinuses and/or thick bones.(relative).

Conclusion: Rhinosinusitis is a complex condition with profound effect on patients quality of life and health care expenditure. The complexity of its etiopathogenesis presents a challenge for its management. While the antimicrobials and steroids from the

mainstay of treatment, topical therapy in form of improved delivery system is being preferred as of now. Potential development of microbial resistance remains a salutary concern in these patients due to repeated and prolonged use of antibiotics. Surgery continues to play an important role in management of recalcitrant disease, resulting in improved quality of life. Immune modulators, such as anti-IgE and anti-IL5 antibodies, are promising newer agents today. Surgical modalities like balloon sinuplasty and stenting are under research. The complex and diverse nature of rhinosinusitis requires an individualized approach to both medical and surgical management in a multidisciplinary setting.

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