facial nerve paralysis and petrous apicitis (Gradenigo's syndrome).[6]

Intracranial complications

Meningitis is the most common intracranial complication of acute and chronic otitis media. Signs that should increase the suspicion of an intracranial complication include persistent or intermittent fever, nausea and vomiting, irritability, lethargy, or persistent headache. Ominous signs virtually diagnostic of an intracranial process include visual changes, new-onset seizures, nuchal rigidity, ataxia, or decreased mental status. If any of these suspicious or ominous signs occur, immediate treatment and further work-up are critical. Broad-spectrum antibiotics, such as third-generation cephalosporins, should be administered while diagnostic tests are ordered and arranged. A contrasted CT scan or an MRI will show characteristic meningeal enhancement and rule out additional intracranial complications known to occur in up to 50% of these cases. In the absence of a significant mass effect on imaging, a lumbar puncture should be performed to confirm the diagnosis and to allow for culture and sensitivity testing.[6]

Other intracranial complications are brain and epidural abscesses, lateral sinus thrombosis and otitic hydrocephalus.[6]

References available at www.cmej.org.za

Management of acute bacterial rhinosinusitis

T Daniller, MB ChB, MMed ORL, FCORL

Medical Specialist, ENT Department, Universitas Hospital, Bloemfontein

Correspondence to: T Daniller (danillert@ufs.ac.za)

Rhinosinusitis is one of the most common conditions presenting to clinicians worldwide, and can potentially have an enormous and a devastating socioeconomic impact.[1-7] The majority of infections are viral in origin, and acute bacterial infection occurs in only 0.5 - 2% of cases.[1-5] The dilemma and diagnostic challenge are therefore to distinguish acute viral rhinosinusitis (AVRS) from acute bacterial rhinosinusitis (ABRS).

Definition

Acute rhinosinusitis (ARS) is defined as symptomatic inflammation of the nasal cavity and paranasal sinuses of less than four weeks' duration. Inflammation of the paranasal sinuses rarely occurs without associated inflammation of the nasal mucosa, and the preferred term is rhinosinusitis.[1-7] As the focus of this review is ABRS, please see the reference list – an excellent source for definitions of subacute, chronic and recurrent ARS and any related inquiry.

Pathophysiology[1-8]

Whatever the insult, the underlying problem is sinus ostial obstruction. This is usually due to a preceding viral infection. However, a number of host and environmental factors may predispose an individual to the development of ABRS (Table 1).

AVRS occurs via direct contact with the nasal mucosa or conjunctiva, with symptom onset within approximately 24 hours. Most commonly, rhinovirus, influenza and parainfluenza viruses are implicated. Thereafter, infection spreads contiguously or systemically to the paranasal sinuses. Positive intranasal pressures, as generated during nose blowing, are believed to play a role.

Inflammation ensues that results in nasal hypersecretion, mucosal oedema, increased vascular permeability and impaired mucociliary clearance with transudation of fluid into the sinuses and nasal cavity. This in turn leads to impaired drainage and

Table 1. Factors that may predispose to the development of ABRS

Host:	Immotile cilia syndrome/ciliary dyskinesia Cystic fibrosis Immunodeficiency (congenital/acquired) Allergy Anatomical abnormalities, e.g. severe septal deviation/spurs, nasal polyps, neoplasms, concha bullosa, paradoxically bent turbinates
Environmental:	Infectious agents (viral/bacterial/fungal) Irritants: tobacco smoke, noxious chemicals
Iatrogenic/traumatic:	Nasal packing Surgery Nasogastric tube Barotrauma Medications Foreign bodies

ventilation of the paranasal sinuses due to obstruction of the sinus ostia. The ostiomeatal complex – the common drainage pathway for the frontal, anterior ethmoidal and maxillary sinuses – is particularly sensitive to this and affected most commonly. Retained, thickened secretions in concert with ciliary dyskinesia, obstructed ostia as well as the antigravitational placement of the ostia, especially of the maxillary antrum, perpetuate the disease process. This leads to the establishment of a favourable milieu for secondary bacterial colonisation and infection.

The normal nasal flora include: coagulase-negative staphylococci, corynebacteria and *Staphylococcus aureus*. The organisms (aerobic bacteria) most commonly associated with acute sinusitis are: *Streptococcus pneumonia*, *Haemophilus influenzae* and *Moraxella catarrhalis*. In odontogenic infections, or more chronic cases, microaerophilic organisms and anaerobes may be encountered.

Clinical manifestations and diagnosis[1-4]

Purulent rhinorrhoea, nasal congestion and facial pain or pressure are highly predictive of acute sinusitis, but the distinction between AVRS and ABRS is often difficult. Secondary symptoms such as anosmia, ear fullness, headache and cough may support the diagnosis. The diagnosis of ABRS is made when symptoms or signs of ABRS are present 10 days or more after the onset of upper respiratory symptoms, or with a worsening of symptoms and signs within 10 days (usually after 5 days) after initial improvement (double worsening). ARS is defined by EPOS[3,4] as symptoms lasting less than 12 weeks with complete resolution, and the BSACI[4] guidelines incorporate a clinical, endoscopic and radiological diagnosis. The RI guidelines[4] use major and minor criteria

Table 2. Rhinosinusitis initiative (RI) guidelines

Major symptoms	Minor symptoms
Purulent nasal discharge (anterior or posterior)	Headache
Nasal obstruction/blockage	Ear pain/pressure/fullness
Facial congestion/fullness	Halitosis
Facial pain/pressure/fullness	Dental pain
Hyposmia/anosmia	Cough
Fever (acute only)	Fever
	Fatigue (malaise)

Table 3. Treatment of AVRS and ABRS

AVRS	ABRS
• Analgesia (paracetamol/NSAIDs) • Mechanical irrigation with buffered hypertonic saline/normal saline • Topical nasal steroid sprays (mometasone furoate 200 mg bd, superior to placebo and amoxicillin) • Topical decongestants (oyxmetazoline) – not longer than 3 days • Oral decongestants (beware: cardiovascular disease/hypertension/ benign prostatic hypertrophy) • Ipratropium bromide (0.06%) • Antihistamines • Mucolytics (guaifenesin) • Zinc preparations not recommended (anosmia)	**1. Observation** Community acquired, uncomplicated (mild pain and temperature <38.3°C) – treat supportively for 7 days after diagnosis; if no improvement or if worsening, initiate antibiotics (age, general state of health and co-morbidities important) **2. Antimicrobials** (moderately severe and severe symptoms) **Adults** 1st-line: amoxicillin 1 g tds for 10 days Alternatives: amoxicillin-clavulanate 1g bd plus additional 500 mg amoxicillin bd or 2 g bd SR for 10 days Cefpodoxime proxetil 200 - 400 mg bd for 10 days Cefuroxime axetil 500 mg - 1g bd for 10 days **Children** Amoxicillin 90 mg/kg/d in 3 divided doses for 10 days Alternatives: amoxicillin-clavulanate 90 mg/kg/d total amoxicillin in 2 or 3 divided doses for 10 days Cefpodoxime proxetil 8 - 16 mg/kg/d bd for 10 days Cefuroxime axetil 15 - 30 mg/kg bd for 10 days **(Please see reference 9 for β-lactam allergy and failed initial therapy)** **3. Adjunctive treatment** Intranasal steroid sprays (topical) Systemic steroids not recommended as outpatient treatment Analgesics, nasal saline irrigation, decongestants

for a diagnosis of ARS, at least two major symptoms or one major symptom with two or more minor symptoms. (See Table 2, cardinal symptoms in bold.)

Physical examination may reveal diffuse mucosal oedema with narrowing of the middle meatus, inferior turbinate hypertrophy and copious rhinorrhoea or purulent discharge. Pain localised to the sinuses on bending forward is more reliable than pain provoked by percussion of the sinuses.

Blind nasal swabs are not recommended. Endoscopically guided middle meatal cultures correlate well with maxillary punctures, but are not indicated routinely. Culture may be helpful in treating complicated cases, cases not responding to empiric treatment, or where atypical pathogens are suspected.

Radiological examination is usually not indicated in the initial evaluation. In cases not responding to treatment, in recurrent infections or in complicated ABRS, a high-resolution CT scan is the preferred modality. Plain films have a low sensitivity and specificity. MRI is not indicated for routine evaluation and ultrasonography is of limited use. (Excellent summary of guidelines in Table 2.[4])

Treatment[1,8,9]

Supportive treatment is indicated in AVRS; however, it does not shorten the clinical course. ABRS may also be treated with antibiotics to eliminate infection and prevent complications, although in a large proportion of patients (40 - 60%) the condition will resolve spontaneously (Table 3).

Treatment failure is defined as progression of symptoms at any time during treatment or failure to improve after 7 days of therapy. Relapse after treatment occurs due to recurrence of symptoms within 2 weeks as a result of inadequate eradication of infection. Unusual pathogens are often the culprit in patients with nosocomial infections and in those who are immune compromised. These patients may benefit from culture-directed therapy. In the acute setting, surgery is indicated only for complicated cases of ABRS.

Complications/sequelae

Transient hyposmia commonly occurs, but permanent viral-induced anosmia is rare. Complicated ABRS arises due to local extension and spread to the CNS (meningitis, intracerebral/epidural abscesses), orbit (orbital cellulitis) and periorbita (periorbital abscess, osteitis).

ABRS may persist and become a chronic rhinosinusitis.

When to refer

Refer patients with:

- high fever, acute facial pain
- swelling and erythema
- abnormal vision (diplopia, blindness)
- altered mental status or meningism
- periorbital oedema and proptosis.

References available at www.cmej.org.za

SINGLE SUTURE

Imaging cameras detect severity of skin problems

High-tech cameras such as those that use thermal imaging can reveal anomalies on a landscape, such as patches of forest infected with a pathogen. Now they could be used to help identify skin problems.

Doctors usually diagnose psoriasis by visually assessing how much skin is covered in lesions, as well as how reddened, thickened and scaly it has become. Such observations are highly subjective, so Francisco Tausk, at the University of Rochester, teamed up with colleagues at the Rochester Institute of Technology, both in New York, to find out whether imaging technology might be more accurate.

In preliminary trials, thermal cameras proved adept at quantifying redness because the increased circulation underneath skin lesions makes them warmer. Photographing skin under ultraviolet light highlighted hard plaques, which the researchers say contain an amino acid that may fluoresce. Both approaches picked out areas of skin that looked normal to the eye, suggesting they may be able to predict where lesions will develop.

The team is now seeking funding for a clinical trial.

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