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Archives of Environmental & Occupational Health

ISSN: (Print) (Online) Journal homepage: https://www.tandfonline.com/loi/vaeh20

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To cite this article: Rafael E. de la Hoz & Michael R. Shohet (2023) World Trade Center Health Program best practices for diagnosing and treating chronic rhinosinusitis, Archives of Environmental & Occupational Health, 78:4, 212-215, DOI: 10.1080/19338244.2023.2169655

To link to this article: https://doi.org/10.1080/19338244.2023.2169655

ARCHIVES OF ENVIRONMENTAL & OCCUPATIONAL HEALTH
2023, VOL. 78, NO. 4, 212–215
https://doi.org/10.1080/19338244.2023.2169655

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BRIEF REPORT

World Trade Center Health Program best practices for diagnosing and treating chronic rhinosinusitis

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ABSTRACT

The most frequent adverse physical health effect among World Trade Center Health Program (WTCHP) members is chronic rhinosinusitis (CRS), with some evidence supporting its association with the exposures to dust, gases, and toxicants. We selected the International Consensus Statement on Allergy and Rhinology: Rhinosinusitis (ICARS-RS-2021) as a comprehensive evidence-based guide on best practices for CRS diagnosis and treatment for the WTCHP.

ARTICLE HISTORY
Received 26 September 2022
Accepted 12 January 2023

KEYWORDS
Chronic rhinitis; chronic sinusitis; occupational medicine; smoke inhalation injury; World Trade Center attack, 2001

Background

Chronic rhinosinusitis (CRS) is a broad public health issue with a prevalence of 12.5%, a yearly estimated cost of $8.6 billion in the United States,[1] and an adverse impact on quality of life that matches those of major chronic medical conditions.[2] Occupational and environmental exposures to both allergenic and nonallergenic air toxicants and pollutants can contribute to the development and worsening of CRS,[2–5] but methodological limitations have precluded an estimation of the population attributable risk.[2,6] Multiple studies have shown that occupational CRS, and other upper airway diseases, very frequently precede and/or coexist with occupational lower airway diseases (LAD).[3] Furthermore, occupational CRS incidence substantially exceeds that of its LAD counterpart. The latter, however, receive considerably more attention in the medical literature. CRS is the most commonly reported adverse health effect in WTC exposed individuals,[7] it was very often associated with disease at the pharyngeal and laryngeal level,[8,9] probably accounted for most of the reported "WTC cough,"[7,10] and contributes to comorbid disease (such as lower airway diseases and obstructive sleep apnea) symptom worsening. As of December 2021, 30% of all WTCHP members were certified for this condition, with some evidence supporting its association with WTC occupational exposures.[11–14] Notably, the prevalence of atopy in the WTC exposed workers does not seem to exceed that of the general U.S. population.[15,16]

Contemporary medical terminology has suggested for several years[17] the use of "rhinosinusitis" (RS) to emphasize the continuum of inflammatory findings throughout the sinonasal mucosa from the anterior nares to the nasopharynx and including the paranasal sinuses and acknowledge the frequently overlapping symptoms.[2,18,19] "Rhinosinusitis" is preferred to "rhinitis" or "sinusitis," which imply confinement of the inflammatory process to the nasal cavity, or the paranasal sinus(es), respectively. Although the consensus terminology has favored this choice for several years,[20] some specialists may prefer to separate chronic rhinitis from chronic sinusitis or CRS, and in some cases a clinician may conclude after careful evaluation and follow up that an individual patient has compartmentalized disease.

The recently published International Consensus Statement on Allergy and Rhinology: Rhinosinusitis (ICARS-RS-2021)2 provides the most comprehensive evidence-based guidance on CRS diagnosis and treatment and met the quality requirements for this best evidence-based clinical practice brief communication series.[21] Given its length (over 500 pages, 2500

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references), this document and the flowchart below (Figure 1, adapted from[8] and consistent with ICARS-RS-2021 guidance) highlight key recommendations and illustrate a diagnostic and treatment pathway.

Diagnostic considerations

The diagnosis of CRS requires both symptoms and objective findings, based on one or a combination of physical, endoscopic, and/or radiologic examination findings. While assessment of occupational and environmental exposures is important for correct diagnosis and successful management of CRS, it is unfortunately rarely considered in general medical practice. RS is typically classified based on disease time course into acute (ARS) and chronic subtypes, and on triggers into allergic, nonallergic, or mixed. CRS is further divided based on the presence (CRSwNP) or absence (CRSsNP) of sinonasal polyps. Individualized treatment, particularly in complex or difficult to treat cases leads in clinical practice to phenotypic characterization with as many qualifiers as needed to guide effective treatment. More generalizable and precise disease phenotyping is likely to evolve further in the near future, as therapeutic options increase, and active research proceeds.[18]

ARS is preceded by infection (e.g., viral) or acute exposures, and is histopathologically characterized by a neutrophilic inflammatory infiltrate. ARS in adults is defined as sinonasal inflammation lasting less than 4 weeks and associated with the sudden onset of nasal airway obstruction and/or facial pain/pressure/fullness AND purulent nasal drainage. A clear distinction should be made between ARS and acute exacerbations of CRS. As acute exposures to WTC toxicants are not a possibility today, ARS is unlikely to be relevant to the objectives of the WTCHP best practices series. CRS is more complex in nature and causation, it implies symptom persistence exceeding 12 weeks,[2,17] and its clinical course can be marked by acute exacerbations that need to be distinguished from acute RS.2

The diagnostic criteria for CRS require duration for at least 12 weeks of at least two of the following symptoms: rhinorrhea or posterior nasal discharge, nasal airway obstruction or congestion, hyposmia or anosmia, facial pain, or pressure, along with one or more of the following objective findings: endoscopic or radiographic evidence of sinonasal inflammation or polyps, OR evidence of mucopurulence draining from paranasal sinuses or outflow tracts. CRS is associated with several risk factors, including genetics, comorbidities, and occupational or environmental exposures. Those patients with acute irritant exposures may present with very few findings on CT and nasal endoscopy. With repeated irritant exposures, there is resulting mucosal damage and progression of the inflammatory process. CRS may result from or worsen the clinical course of other diseases, and clinicians need to evaluate those comorbidities carefully.[17,19]

Treatment considerations

Once the diagnosis of RS has been established and upon distinguishing acute from chronic symptoms, CRS symptoms can usually be managed very effectively. Alarm symptoms such as bleeding, coincident ocular symptoms, severe facial pain or headaches, symptom unilaterality, or disequilibrium may imply a complication of active infection or underlying neoplastic process. Such symptoms warrant more aggressive diagnostic investigations such as early imaging and/or endoscopic examination,[8] which are helpful to assess the extent of the inflammatory changes, and to identify any structural factors that may interfere with medical treatment efficacy and/or provide potentially correctible surgical targets to improve symptom control (Figure 1).

In the absence of alarm symptoms, a trial therapy of saline nasal irrigation (performed one to four times daily) to limit the interface between any underlying irritants and the sinonasal mucosa is remarkably effective in both reducing airway irritant exposures and improving mucociliary clearance of such irritants. Greater severity of symptoms or findings, such as mucosal edema or excessive mucus production, may merit the addition of a topical corticosteroid.

Exposure history is critical at this point. History consistent with CRS symptom triggering by seasonal or perennial exposure to aeroallergens may warrant allergy testing and treatment, accordingly, more so if there is comorbid lower airway disease. Further management of underlying allergies may take the form of reasonable avoidance, topical or systemic antihistamines, leukotriene modifiers, and consideration of immunotherapy or biological agents. Documentation of potential irritants in the workplace is also very helpful in considering best management options. Circumstances in which symptoms are significantly more prominent while at the workplace or improved while away from the workplace for an extended period may indicate the presence of a workplace irritant, allergen, or other toxicant, and warrant further investigation and potential mitigation.

Acute bacterial and nonbacterial exacerbations of chronic symptoms are quite common and may be

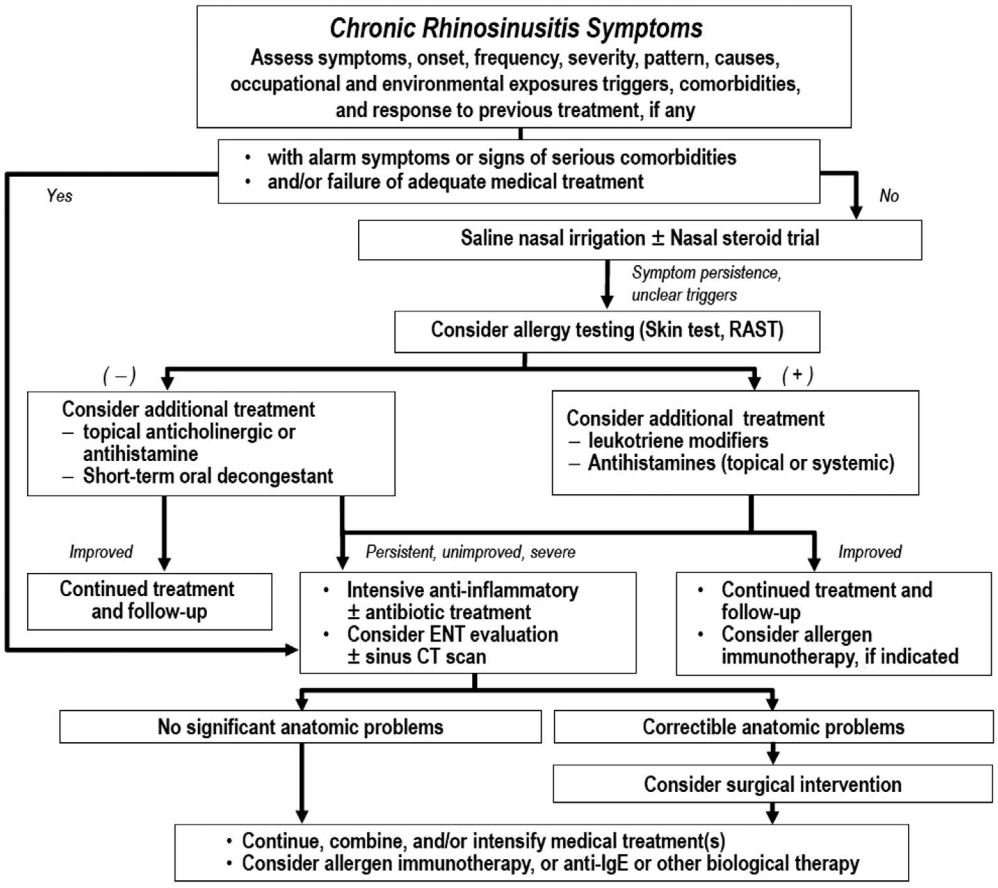

Figure 1. Flowchart illustrating best practices for chronic rhinosinusitis diagnosis and treatment (updated from[8] and consistent with guidance in[2]).

precipitated by more extensive irritant or allergen exposures or by infectious agents. Depending on the underlying factors, such exacerbations may require management with more aggressive short-term medical therapy such as systemic antihistamine and/or decongestants, antimicrobials, and potentially anti-inflammatory management with topical or systemic corticosteroids. Frequent acute exacerbations of CRS also warrant a more aggressive diagnostic approach such as CT imaging and/or endoscopy.

The pathogenesis and management of the different phenotypes of CRS, namely CRSwNP and CRSsNP, are often complicated by the divergence of pathogenetic factors, many of which remain to be investigated.[18] Their management may therefore require different and specialized medical and/or surgical approaches such as more extensive biologic therapy to modulate IgE- or eosinophil-mediated inflammatory pathways and continued close multidisciplinary follow up.

Program coverage

CRS diagnosis and treatment services can be covered by the WTC Health Program. For treatment to be covered, the WTC Health Program member's CRS must be administratively certified. To receive certification, a Clinical Center of Excellence (CCE) or Nationwide Provider Network (NPN) needs to submit a WTC-3 form (https://www.cdc.gov/wtc/pdfs/Appendix-WTC3.pdf). Among other things, on the WTC-3 form that CCE/NPN physician must attest that WTC site exposures were substantially likely to have been a significant factor in aggravating, contributing to, or causing the enrolled WTC member's CRS.

Acknowledgments

The authors acknowledge the comments from the clinicians of the WTC HP Clinical Centers. This review did not involve human subjects research and was thus deemed exempt from institutional review board review.

Disclosure statement

The authors have no other relevant financial conflict of interest.

Funding

This work was funded in part by grant U01 OH010401 (RED, PI) from the Centers for Disease Control/National

Institute for Occupational Safety and Health (CDC/NIOSH). The contents of this article are the sole responsibility of the authors and do not necessarily represent the official views of, nor an endorsement by, the National Institute for Occupational Safety and Health (NIOSH), the Centers for Disease Control and Prevention of the U.S. Department of Health and Human Services (CDC/HHS), or the U.S. Government.

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