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Diagnosis and Management of Reactive Airways Dysfunction Syndrome (RADS)

An APCCMPD Ambulatory Care Curriculum Teaching Script

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Diagnosis and Management of Reactive Airways Dysfunction Syndrome (RADS)

Scenario 1:

Ms. H is a 45-year-old female with a past medical history of obesity, hypertension, and diabetes mellitus type 2 who worked as a housekeeper in a hotel several months ago. During that time, she reports exposure to cleaning chemicals for a few hours each day that she worked. After a period of time with no exposures, she developed dyspnea with even mild exertion and was hospitalized with a presumptive diagnosis of chemical pneumonitis. She returned to work several weeks later with strict advice to avoid the known chemical exposures but developed similar symptoms a few days after restarting as a housekeeper and was hospitalized again. Following her second hospitalization, she utilized her short-term disability coverage and did not return to work, finding that her symptoms had improved. After a few months out of work, she was able to climb at least two flights of stairs without dyspnea.

Scenario 2:

Ms. B is a 37-year-old female presenting with daily headaches after 20-60 minutes of being at work. She was recently moved to the basement of her employer's building but then moved back to her original office with resolution of her symptoms. Several months later, she moved back to the basement and experienced progressive dyspnea on exertion, sore throat, cough, nasal congestion, and sneezing with return of her headaches. These symptoms progressed over 7 months before she moved back to her original office though these symptoms continued. She transitioned to working from home and only has symptoms when she returns to the office for meetings. She no longer has respiratory symptoms outside of work. Despite her excellent metabolic capacity and regular exercise regimen, she expresses concern about the long term or delayed effects of any exposures on her future health.

Question 1: How would you characterize these exposure histories?

Question 2: Ms. H and Ms. B each ask about the possible cause of their symptoms. What is the differential diagnosis for their work-related symptoms?

Question 3: Both individuals ask, "What is RADS?" How would you explain this diagnosis to them?

Question 4:
What is your diagnostic evaluation? What are the typical clinical symptoms?

Question 5:
What is the treatment for RADS? Will they get better?

Question 6:
Ms. B is seeking worker's compensation and asks if you will testify on her behalf. Can you be her expert witness? What forms can you expect to see? What ethical conflicts does this pose?

Diagnosis and Management of Reactive Airways Dysfunction Syndrome (RADS)

Educational Objectives:

1. Review the definition, diagnostic approach, and management of RADS
2. Understand the relevant differential diagnosis of RADS among occupational lung diseases and the importance of concomitant medical diseases and treatments
3. Begin to understand the different approaches to disability and legal evaluations

Scenario 1:

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Scenario 2:

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Question 1: How would you characterize these exposure histories?

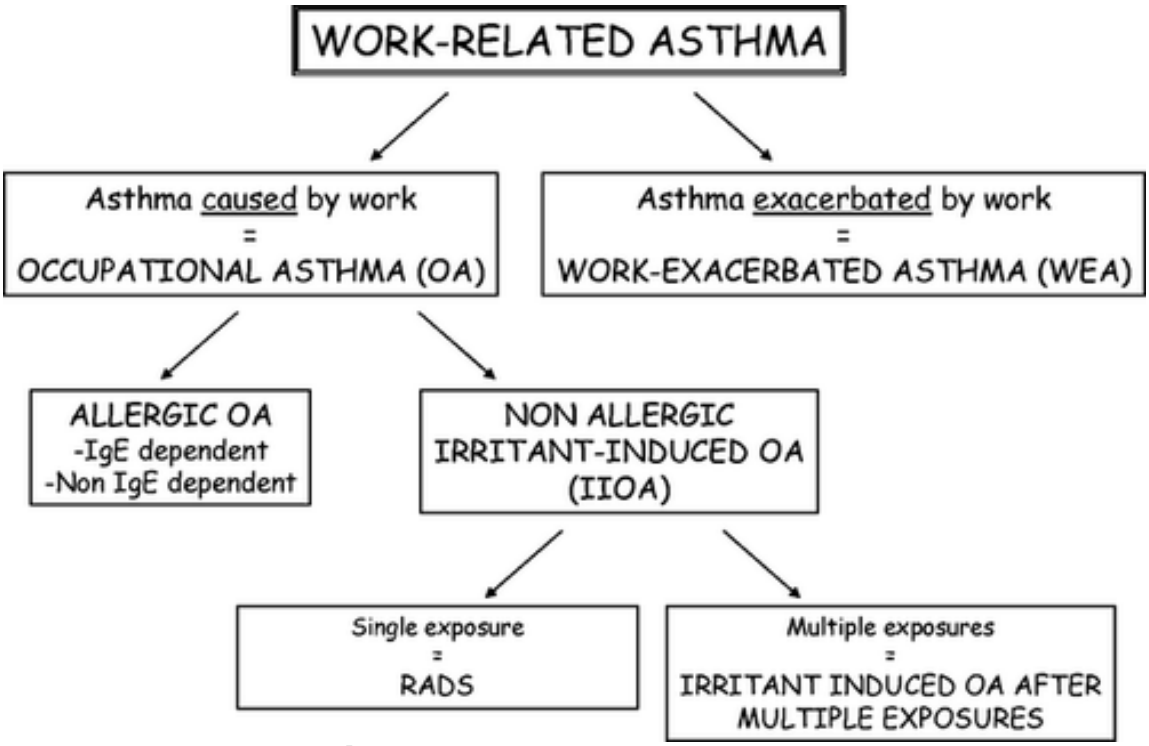
Short-term and transient exposures in the workplace. There is typically no progression of symptoms over a work week. Symptoms persist for prolonged periods of time after initial exposure. Symptoms eventually resolve after the patient has removed themselves from their initial work environment.

Question 2: Ms. H and Ms. B each ask about the possible cause of their symptoms. What is the differential diagnosis for their work-related symptoms?

- Occupational Asthma (Figure 1)
- Pneumoconiosis
    - Coal workers' lung
    - Silicosis
    - Hard metal lung disease
    - Berylliosis
    - Giant cell interstitial pneumonitis
    - Hypersensitivity pneumonitis
    - Asbestosis
    - Innumerable others (byssinosis, red cedar, etc.)
- Organizing pneumonia
- Non-asthmatic eosinophilic bronchitis
- Fungal and mold exposures
    - Aspergillus
        - Allergic bronchopulmonary aspergillosis (ABPA)
        - Aspergillomas
    - Allergic bronchopulmonary mycosis (ABPM)
    - Stachybotrys
    - Cladosporium
    - Fusarium
    - Penicillium
    - Other (coccidiomycosis, histoplasmosis)
- Reactive Airways Dysfunction Syndrome (RADS) or Acute-onset irritant induced asthma (IIA)[1]
    - Common irritants: acetic acid, ammonia, chlorine, isocyanates, mustard gas, fire/smoke
- Sub-acute IIA or IIA with latency[1]
- Low Intensity Chronic Exposure Dysfunction Syndrome (LICEDS)
- Inducible laryngeal obstruction
- Constrictive bronchiolitis[2]
- Bronchiolitis obliterans[3]
- Sick building syndrome[4]
- Multiple chemical sensitivity syndrome
- Sporadic, community acquired concurrent illnesses
    - Bronchitis, pneumonia, sinusitis
- Exacerbation (or simply detection) of pre-existing diseases
    - Asthma/Chronic Obstructive Pulmonary Disease (COPD)
    - Congestive Heart Failure (CHF)
    - Obesity and/or deconditioning
    - Gastroesophageal Reflux Disease (GERD)
    - Chronic rhinosinusitis

- Upper airway cough syndrome
- Side effects of anti-hypertensive and cardiac medications:
    1. ACE (Angiotensin-Converting Enzyme) inhibitors and beta blockers
        a. Cough (ACE>ARB (Angiotensin-Receptor Blocker)
        b. Beta blockers may worsen airway obstruction
        c. Beta- and calcium-channel blockers can cause chronotropic limitation
- Anxiety and/or depression with somatization

Figure 1. Classification of Work-Related Asthma[5]



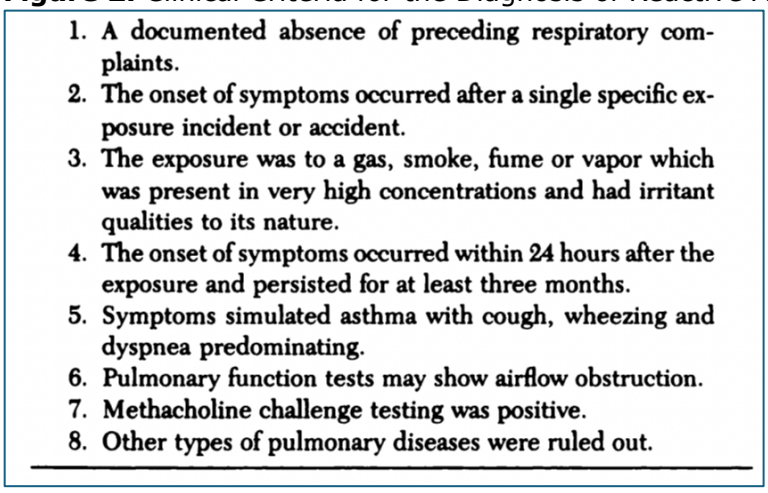
Reproduced from Moscato et al.[5]

Question 3: Both individuals ask, "What is RADS?" How would you explain this diagnosis to them?

RADS or acute-onset irritant induced asthma (IIA) is characterized by[6,7,8,9]

- Absence of a pre-existing respiratory disease or a history of asthma in remission and exclusion of conditions that can mimic asthma.
- Short duration, high intensity exposure to a known respiratory irritant resulting in asthma-like symptoms (cough, wheezing, dyspnea) that most often occur within 24 hours of the exposure and persist for at least 3 months.
- Figure 2 describes the Brooks' criteria for RADS diagnosis.[6]

Figure 2. Clinical Criteria for the Diagnosis of Reactive Airways Dysfunction Syndrome (RADS)[6]



1. A documented absence of preceding respiratory complaints.
2. The onset of symptoms occurred after a single specific exposure incident or accident.
3. The exposure was to a gas, smoke, fume or vapor which was present in very high concentrations and had irritant qualities to its nature.
4. The onset of symptoms occurred within 24 hours after the exposure and persisted for at least three months.
5. Symptoms simulated asthma with cough, wheezing and dyspnea predominating.
6. Pulmonary function tests may show airflow obstruction.
7. Methacholine challenge testing was positive.
8. Other types of pulmonary diseases were ruled out.

Reproduced from Brooks et al.[6]

Question 4: What is your diagnostic evaluation? What are the typical clinical symptoms?

- Compatible history is key to making the diagnosis
- Clinical symptoms[10,11]
    - The most common symptoms are dyspnea (71% of cases) and cough (65% of cases) along with wheezing, chest tightness, and often immediate nasal and throat burning. Symptoms are rapid onset, almost always within 24 hours of exposure, usually with a very clear date/time of onset. Some important questions to ask would include:
        - Was the onset of symptoms related to one memorable event?
        - Were others in the vicinity also affected?
        - Was there any repeat gas, fume, or dust exposure? Is there ongoing exposure?
- Pulmonary function tests[12]
    - Peak flow monitoring, with exposure diary (at least 4x/day for at least 2 weeks at work and 2 weeks off work)
    - Serial spirometry: May see airflow obstruction with a significant bronchodilator response
    - Methacholine challenge testing is typically positive and should be checked when IIA is suspected, and spirometry is normal
        - Remember up to 15% of normal (lifelong nonsmokers with no respiratory or atopic history) can have borderline bronchial hyperresponsiveness
        - Prior tobacco use can produce false positive results
    - Cardiopulmonary exercise testing can be considered if the cause of symptoms remains unclear
- Imaging
    - Chest radiographs (PA and lateral) usually normal
    - Chest CT scan may show mosaicism/air trapping
- Laboratory studies
    - Hypersensitivity pneumonitis panel
    - Immunoglobulin E level
    - Radioallergosorbent testing (RAST)

    - Complete Blood Count (CBC) with differential (to assess for eosinophilia)
- Airborne testing: is workplace less or greater than ambient air?
    - Mold spore counts
    - Particulates (dust)
    - Volatile Organic Compounds (VOCs)
- Custom testing
    - Specific inhalation challenges (SICs): exposure to low doses of occupational agents at a specialized center or laboratory to measure dose response reaction[14]
- Safety Data Sheets, formerly (until c.2012) known as Material Safety Data Sheets (MSDS), available for known exposures (e.g., cleaning agents)[13]

Question 5: What is the treatment for RADS? Will they get better?

Typically, we follow an approach in treatment that mirrors the stepwise approach described in the National Asthma Education and Prevention Program (NAEPP) and Global Initiative for Asthma (GINA) guidelines, although there have not been any studies formally assessing its efficacy in this population. Often affected individuals can return to work after appropriate asthma management, but if symptoms worsen at work, they may require removal from that work setting. Recommendations are based on expert opinions, case reports, and case series.[14]

- Tincture of time
    - Removal of exposure, symptoms can abate after 12-18 months
    - Outcomes vary, with some patients still having pathologic and clinical evidence of pulmonary dysfunction many years out[17,18]
- Corticosteroids
    - Oral: for acute onset of symptoms. Consider taper rather than short burst given risk of rebound
    - Inhaled corticosteroid/bronchodilator combination
- Anticholinergics and leukotriene antagonists
    - Can be added if patients remain symptomatic despite above therapies
- Investigate for and treat other related disorders as well, which may include chronic sinus issues, inducible laryngeal obstruction, and post-traumatic stress disorder
- Prognosis
    - Recent research shows that patients with IIA had stable lung function after a median of 11 years since their initial exposure.[16]

Question 6: Ms. B is seeking worker's compensation and asks if you will testify on her behalf. Can you be her expert witness? What forms can you expect to see? What ethical conflicts does this pose?

- Independent medical examination (IME)[19-21]: This is completed by a physician at the request of employer, insurer, lawyer, or other entity. There is no establishment of a physician-patient relationship.
    - Obligation to requestor, NOT TO PATIENT!
    - Cannot divulge/report your opinion to the patient, but can alert them to incidental findings noted during the evaluation
    - Patient entitled to learn the IME results through appropriate due process
    - The physician can serve as an expert witness
- Treating physician: A physician-patient relationship is established.
    - Obligation to patient
    - No obligation to employer or lawyer on either side
    - Can testify as a fact witness
    - Remember, your notes are read!
    - ***MAY*** be precluded from testifying as an expert
- Physician's Affidavit of Recovery
- Return To Work Physical Capabilities Form
- Role of the physician – am I treating the patient or adjudicating a claim?
    - The incidental finding in an IME (e.g., lung cancer or unstable angina)
    - Empiric steroids or a surgical lung biopsy for hypersensitivity pneumonitis?
    - Do they have a disability from the exposure?

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