

The Diagnosis and Management of Occupational Asthma and Work-Aggravated Asthma

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Work-related Asthma -2004

Overview, definitions
Types of work-related asthma
 Immune-mediated (sensitizer) asthma
 RADS - irritant
 Work-aggravated asthma
Epidemiology
Pathophysiology
Cases
 Diagnosis
 Natural history
 Management

Work-related Asthma - Definitions

Occupational Asthma (OA)

New asthma *caused* by specific exposure / process at work
- Immune-mediated (sensitizer) asthma
- RADS - irritant

Work-aggravated asthma (some include as type OA)

Pre-existing asthma that exacerbated by exposures at work

Patient with work-related asthma symptoms

Primary causation vs exacerbation can be very difficult
differentiate (only clear cut in text books)

Epidemiology

Immune-mediated vs RADS vs Work-aggravated

Work-aggravated likely most common

RADS least common (< 5-20%)

Ontario - WC claims

50% we claims work-aggrav

10% RADS

40% sensitizer

(Tarlo 2000)

OEM clinic US - 30% work-aggrav

25% RADS

40% sensitizer

(Barnhardt 1999)

Work-Related Asthma in New York State

Alicia Fletcher, MPH
NYS Dept Health

Analysis NY Occupational Health Clinic Network data base

454 Patients with MD diagnosis work-related asthma

93 % met NIOSH diagnosis (MD diagnosis, work-related symptoms)

Work-Related Asthma in NY (N= 421 patients)

50% female

>75% white

Work-aggravated

15%

RADS

40%

Known inducer (antigen)

50%

Industries

services industries (health care, education)

manufacturing

agriculture

transportation

Agents

dust, indoor air, mold, solvents, cleaning products, paints

Work-Related Asthma

If 10-25 % adult asthmatics have work-related asthma, why aren't more cases recognized and diagnosed ?

Problems with Diagnosis Work-related Asthma

Difficult differentiate from ordinary asthma -
 especially later in course disease
 No single simple diagnostic test
 Host factors unclear
 Focus on treatment rather than etiology /prevention
 Lack of information about workplace /exposures
 Concern about /avoidance worker's comp / legal

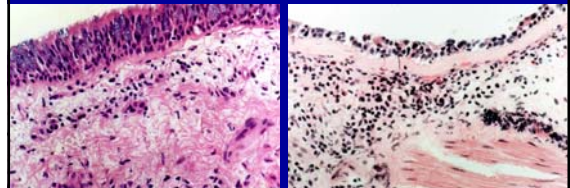
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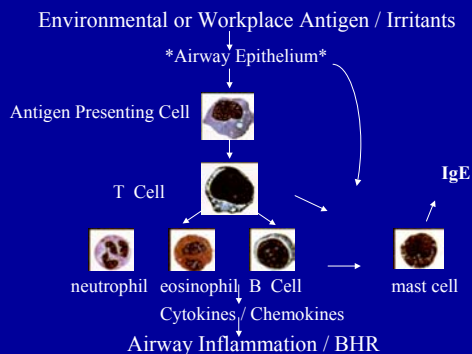
Airway Pathology in Occupational Asthma

Normal

Isocyanate (any) Asthmatic



Mechanisms in Asthma



Case

A 34-year-old man presents with a 6 month history of worsening cough and dyspnea. He 'outgrew' childhood asthma. He has worked for past 4 years at an auto body shop repairing, sanding, and painting cars. He notes improvement on weekends. Spirometry is normal.

Case

Is the problem Asthma ?

Is the problem work-related ?

Differential diagnosis Work-related Asthma

- A) upper respiratory tract irritation
- B) hypersensitivity pneumonitis
- B) vocal cord dysfunction
- C) sinusitis / rhinitis
- D) ordinary asthma

Diagnosis Asthma

Need to confirm reversible airflow obstruction

Spirometry: changes, BD response

Methacholine challenge (off meds)

Physician or patient diagnosis of asthma, reported wheezes on exam, use of asthma medications not sufficient

More is at stake if potentially work-related

Case - Additional History

Auto body shop worker for 4 years

Onset symptoms past 4-6 months

No known changes, new products at work

Cough, wheeze in evening at home, not at work

Evening symptoms worse when paints / primes

No one else at work symptoms

No allergies, pets, recent URIs

No carpeting, mold at home

Positive methacholine challenge

Is it Work-Related Asthma?

Need information on
type work, exposures
amount /dose

Need confirmation of the association between asthma
and workplace

□

Exposure Information: What and How Much?

Patient (description of job, process, products used, respiratory / skin protection, ventilation)

MSDS

Type of industry / job / process

Employer

Work site visit

OSHA

Union

IH data



Auto body shops

- Isocyanates (paints / primers)
- Solvents
- Plastic resins
- Grinding / sanding metals, plastics, fiberglass
- Welding fumes
- Particulates / dusts

You suspect work-related asthma.

What is the best way for you to “confirm the diagnosis” ?

Association between Asthma and Work

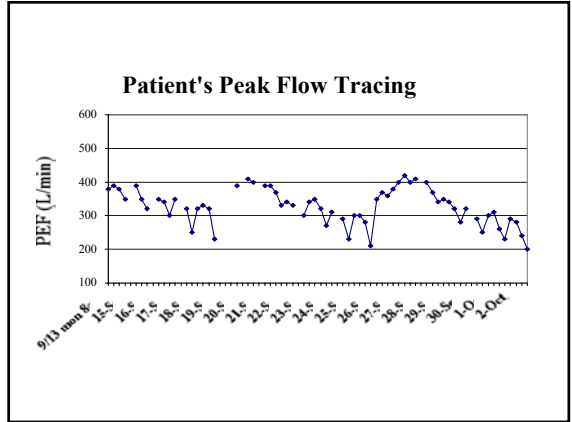
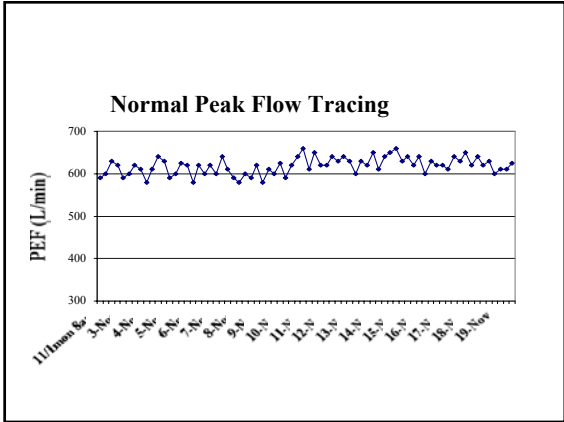
Occupational history
(symptoms, onset, timing, association with work)

Physiology
Peak flows
Spirometry
Specific challenge

Immunology

Physiological Documentation of Work Association

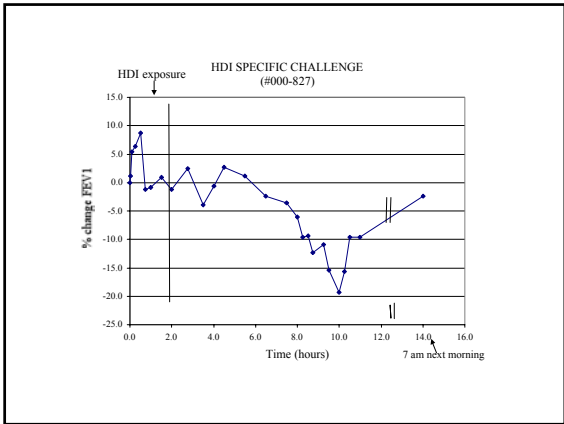
Peak flows vs
Spirometry vs
Specific challenge



PEFs

PEF more sensitive (70-80%) and specific (80-100%) than spirometry (< 50%).
 use 4-6 times / day for at least 2-3 weeks, off work 2 days - 2 weeks. □

Limitations:
 Patient has to be at work, exposed
 Patient cooperation
 No clear criteria to interpret - 'expert opinion'
 May need more than 1-2 days away from work for improvement
 Time consuming
 Use medications, respiratory protection can confound



Specific Inhalation Challenge

'Gold Standard' for sensitizer OA, but *not so gold*
 False positives and negatives
 Need identify and safely deliver specific agent in correct form (aerosol, vapor, particle size)
 Not available in US
 Way more involved / complicated / impractical than appears
 Not good for RADS, irritant-induced asthma

Immunologic Tests

Skin prick

Serologic

Total IgE

Antigen-specific IgE

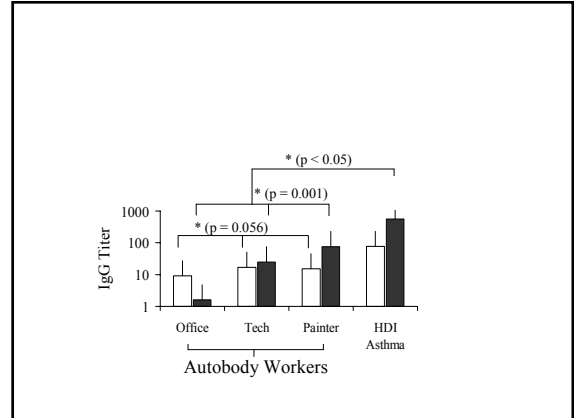
More helpful with large MW agents

(confirms exposure not disease)

Small MW agents

(frequently neg, usually not available)

Not useful for RADS, irritant-induced asthma



Isocyanate Asthma

Most common cause immune-mediated occupational asthma

5 - 25 % exposed workers

Latency

Early, late, dual reactions - late common

Host risk factors ?

Once sensitized re-exposure to low levels can cause asthma

Diagnosis, prevention difficult

Immune-Mediated Occupational Asthma

High MW agents

Cereals (flour, grain dust)

Animal-derived allergens (rodent, egg, seafood proteins)

Enzymes (papain, pepsin a-amylase)

Gums

Latex

Low MW agents

Isocyanates (paints, polyurethanes)

Wood dusts (plicatic acid)

Anhydrides (plastics, epoxy resins)

Dyes

Chloramine, formaldehyde, glutaraldehyde

Metals, fluxes

Natural History Immune-Mediated OA

Persistence disease away from exposure

Most some improvement

25 - 80 % persistent asthma

Can worsen away from exposure

Better prognosis with

early recognition and removal

less NSBR, symptoms for fewer years

Socioeconomic outcomes poor - unemployed, lower income, worse QOL

Case

Final diagnosis - Isocyanate asthma

Based on:

Positive methacholine challenge (confirm asthma)

Good story (sxs, timing etc)

Known isocyanate exposure

PEFRs - decline associated with work

Positive specific challenge

Do I need physiologic confirmation to diagnose occupational asthma?

- A) Yes
- B) No
- C) Only if worker's compensation case
- D) Only if planning to sue the employer

Do I need to know the specific / agent cause?

- A) Yes
- B) No
- C) Only if worker's compensation case
- D) Only if planning to sue the employer

How certain do I have to be of my diagnosis?

- A) > 95 %
- B) > 90 %
- C) > 75 %
- D) > 50 %

Level of Diagnostic Certainty

In US (worker's comp) - more probable than not = > 50 %

Diagnosis occupational asthma is not a criminal case (burden of proof 'beyond a reasonable doubt')

Rarely > 70 - 80 % sure

Case

A previously healthy 38 y/o policeman comes to your office because of a cough of three months' duration. He first developed a cough several hours after directing traffic around a large chemical spill and fire. The cough is worse in the cold. He is a nonsmoker. He has a history of seasonal allergies but no prior history of asthma. Pre-employment spirometry was normal.

Physical exam is unremarkable. No wheezing.

FEV₁/FVC is 68%, with BD response.

Chest radiograph is normal.

Reactive Airways Dysfunction Syndrome (RADS)

New onset asthma within 24 hours after single exposure to high concentration of inhaled irritant (gas, smoke, fume, vapor)

Asthma symptoms (cough, wheeze, dyspnea)

Symptoms continue for > 3 months

Presence reversible airflow obstruction or increased NSBHR

Other disease ruled out

Brooks et al. 1985
Alberts 1996

Irritants Cause RADS

Chlorine
Phosphoric acid
Nitrogen oxides
Sulfur dioxide
Ammonia
Smoke
Welding fumes
Acetic acid
Diisocyanates
WTC cough

5 - 20 % cases of OA



FDNY Firefighters - Cough

Cough - (with dysnea, GERD, nasal sx)
More common if high exposure

PFTs: Pre WTC - 1% FEV1 < 65%
Post WTC - 14% FEV1 < 65%
63 % positive BD
35 % BHR - associated with exposure

CXR - unremarkable

HRCT scans - 50% airtrapping, 30% bronchial wall thickening

Persistent hyperreactivity and reactive
airway dysfunction in firefighters at the
WTC Banauch *AJRCCM* 2003; 168:54-62

BHR associated with exposure

25% high, 8% moderate exposure BHR

6 fold increased risk

BRH persisted in 55% for 6 months = RADS

Persistence BRH associated with early BHR

Diagnosis RADS

Consistent clinical and exposure history -

No prior history asthma

Acute high exposure - sx > 3 months

Document airflow obstruction or hyperresponsiveness

RADS / Irritant-induced Asthma Controversies

- Acute exposure(s) frequently poorly documented
- Can be difficult rule out pre-existing BHR
- Why only some affected?
 - Risk factors: atopy
- ? Previously asymptomatic /mild asthmatic - actually work-exacerbated asthma
- If one big exposure can cause it, what about several lower exposures 'low dose or chronic RADS' = irritant-induced asthma?

Case SR

43 year old female teacher presents with worsening cough, shortness of breath, fatigue. Non smoker. Asthma as child. Symptomatic again past 5 years. 3 hospital admissions for asthma in the past year. Home - cat for 10 years.

Further Occupational history:

Symptoms better over holidays, worse when teaching. Has missed numerous work days over past 2 years.

Further Exposure Information

School Evaluation

HVAC system not working, stagnant water, mold

Water damage - moldy carpet in her room

Work-Aggravated Asthma

Pre-existing asthma that is aggravated by exposures in the workplace

Factors Exacerbate Asthma

Environmental allergens

- cats
- dust mites, cockroaches
- molds, pollens

Viral infections

Cigarettes - ETS

Cold

Respiratory tract irritants

- Dusts / particulates

- Solvents / other chemicals

- Fumes / gases

- Metals

Drugs

Exposures in Indoor Air

Microbial agents

- Bacteria, fungi, molds

Volatile organic compounds

- Formaldehyde, solvents, glues, carpets

Dusts / fibers

- Fiberglass, construction dust

Animal proteins

- cats, birds, rodents, dust mites

Entrapped outdoor sources (vehicle exhaust)

Contaminants generated by human activity

- CO₂, perfume

Physical Factors

- Temperature, humidity, noise, lighting

Other

- Fuel combustion products, cleaning agents, pesticides
- ETS, Radon

Irritants Can Exacerbate Asthma

Higher exposures more likely exacerbate asthma

Exposures frequently non-industrial

Differences in host susceptibility

Interaction irritants/air pollution and allergens/sensitizers

- Animal studies - ovalbumin, irritants (ozone, sulphur dioxide)

- Human exposure studies - diesel exhaust, ozone

- Epidemiology studies - smoking, diesel exhaust risk factor

Active area research

Can Irritants Cause New Asthma? The Evidence - Probably and Growing

- Case reports - new carpets, cleaning agents, chem lab (Kipen 1994, Brooks 1990)
- Epidemiological studies
 - 1) Pulp mill workers - chlorine or ozone associated with meth responsiveness, sxs (Gautrin 1999; Bherer 1994; Toren 2002)
 - 2) Machinist apprentices - increased meth responsiveness at 2 yr follow-up (Kennedy 1999)
 - 2) Population-based studies - increased prevalence asthma in workers with irritant exposures (Kogevinas 1999, Blanc 1999, Toren 1999)
- Longitudinal follow-up new workers (inception cohort) difficult
- WTC cough

Work-related Asthma: Diagnosis

Consider in all adult onset or worsening asthmatics
Confirm diagnosis of asthma
Determine association between asthma and work history, physiology
Determine Immune-mediated vs RADS vs Work-aggravated - primarily by history, type of exposure
Level of diagnostic certainty - only > 50%

Work-related Asthma: Natural History / Prognosis

Literature mostly immune-mediated asthma

Persistent asthma symptoms away from causative agent(s) common
Earlier removal from exposure better outcome
Unemployment, reduced income, worse quality of life common

Work-related Asthma: Management

Depends (in part) on type work-related asthma
Important to recognize/ diagnose - but do not rush to remove from work
Modification work environment - Reduce exposure if sensitized - best to remove if can
Consider implications removal from work
Standard asthma meds - monitor
Desensitization - certain large MW antigens
Advise about disability / compensation
Consider public health issues
Early recognition critical

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