LUNGS AT WORK
www.lungsatwork.org.uk

13 January 2021
StRs training day

Acute inhalation injuries

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Definition

“An acute exposure to a potentially toxic agent that could cause respiratory illness”

Gas

Vapour: gaseous form of a substance that is in liquid or solid form at normal T and P

Aerosol: mix of potential states (liquid droplets/fine particulates dispersed in a gas); smoke is subset resulting from incomplete combustion

Fume: solid material, often metals, of small particle size [most <1.0μm] suspended in a gaseous medium that has condensed from a vaporized state

Questions you might want to ask

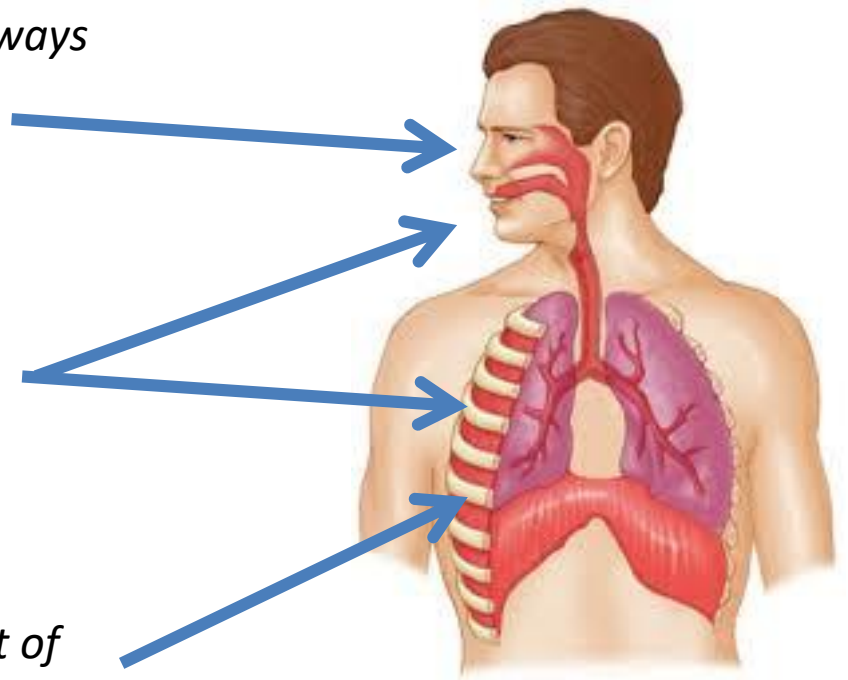
Agent factors:
known or unknown?
solubility
quantity inhaled
duration of exposure
temperature of agent (thermal injury esp if particles in the inhaled material)
density of gas (chlorine in WW1 trenches *vs* displacement of oxygen if lighter than air)

Environment:
poorly ventilated or enclosed areas
elevation (low and high)
others affected

Host factors:
use of RPE
pre-existing respiratory disease

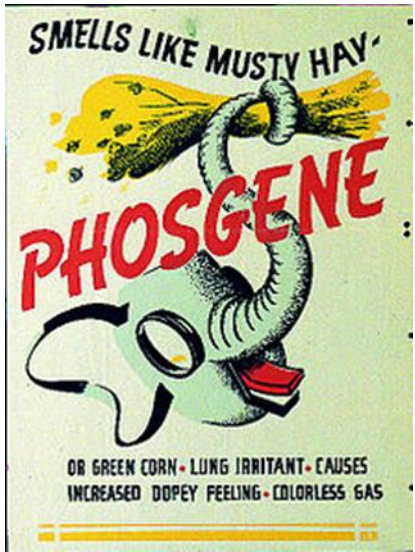
Water solubility

Highly water soluble agents:
immediate irritation to the eyes and upper airways
ammonia
hydrogen chloride
sulphur dioxide



Intermediately soluble:
upper and lower airway inflammation
chlorine

Low water solubility/insoluble: *delayed onset of lower airway inflammation (24-48 h): pneumonitis, pulmonary oedema, small airways obstruction*
oxides of nitrogen
Phosgene (carbonyl chloride)
ozone
zinc oxide



Acute management (severe cases): some principles [agent may be unknown]

Management is largely supportive
Recognise possibility of delayed onset of Sx (up to 48h)

Radiological changes rare; suggest significant exposure

Bronchorrhoea and airway oedema ------ distal airway injury with diffuse alveolar damage

Standard ARDS mx:

- preemptive intubation and ventilation
- avoid high O2 conc.
- protective ventilator strategies…..

No evidence for corticosteroids acutely (although usually given); esp. in smoke inhalation

Measurement of spirometry if possible

Inhaled bronchodilators and ICS if airflow obstruction or ongoing dyspnoea

Avoid prolonged polypharmacy without objective assessment

Deutsch et al Burns 44 2018

Prognosis

Majority with acute inhalation injury have no long-term adverse health outcomes (1)

Minority develop longer-term complications:

- case reports; little information on exposure measurement or pre-existing lung fn

- upper airways symptoms e.g. chronic rhinitis and VCD/ILO
- airflow obstruction / non-specific airway hyper-responsiveness (irritant-induced asthma)
- Breathing pattern disorder; PTSD (regardless of exposure and consequence)

- small airways disease e.g. Union Carbide explosion in pesticide factory (Bhopal 1984)
- “interstitial lung diseases”
- ARDS with assoc. complications of obliterative bronchiolitis and bronchiectasis

(1) Sallie and McDonald, 1996

51M Engineer in a leisure centre

Exposed to sodium hypochlorite and trichloro-s-triazinetrione
Undefined period of exposure 23rd and 24th August 2017 (whilst carrying out checks)
c.one hour on the 24th August at the time of the heaviest exposure (lid lifted on a tank)
Pulled colleague to safety

Productive cough with haemoptysis, dyspnoea, pleuritic chest pain, wheeze;
+ loss of sensation in his finger tips, brittle nails, significant weight loss (BMI 17) , poor memory, unsteadiness on his feet, yellow cataract, metallic taste in his mouth…..

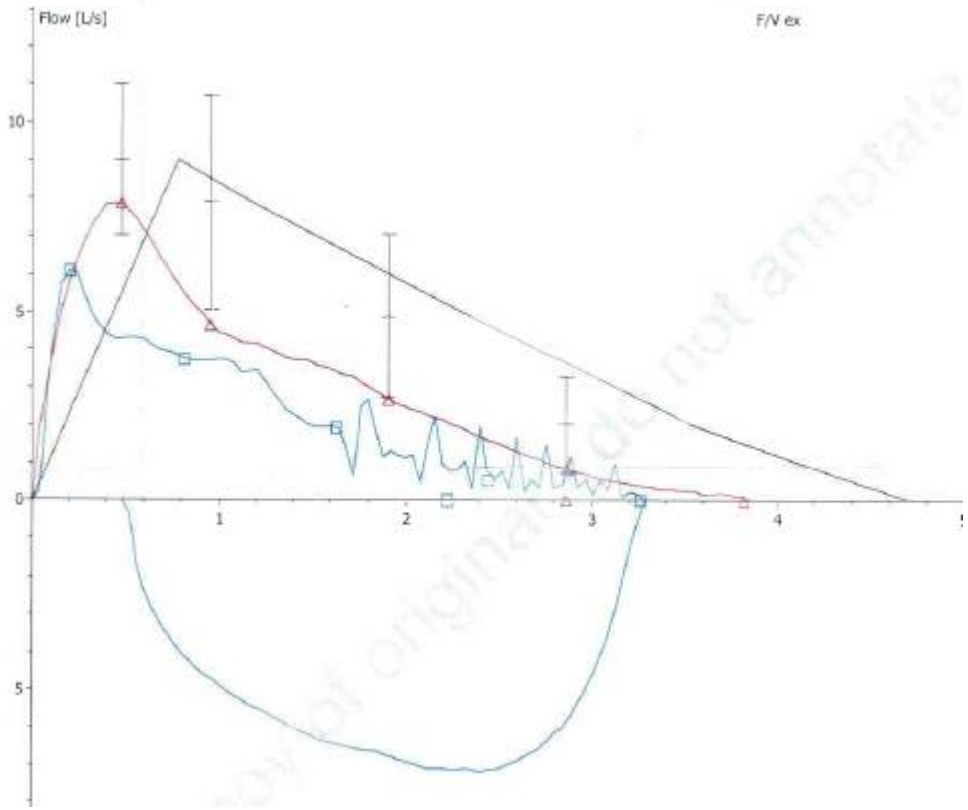
Has not been able to return to work; solicitor involved; angry +++

PMH: IHD, DM; no respiratory disease

DH: Ventolin x12/24h, Fostair 200/6 two puffs bd, Becotide, sertraline, analgesia

SH: smoker of cigarettes and cannabis

	Pred	Pre Test 1	% PRED	SR	POST	% PRED	% CHANGE
Date		28/12/17			28/12/17		
FEV 1	3.77	2.22	59.0	-3.03	2.86	75.8	28.5
FVC	4.70	3.26	69.3	-2.37	3.82	81.2	17.1
VC MAX	4.90	3.26	66.5	-2.93	3.82	77.9	17.1
FEV1%M	78.03	68.27	87.5	-1.36	74.90	96.0	9.7
PEF	9.01	6.09	67.6	-2.42	7.87	87.3	29.2
MEF 25	2.03	0.55	27.1	-1.90	0.84	41.2	52.4
MEF 50	4.89	1.94	39.6	-2.24	2.67	54.6	37.9
MEF 75	7.88	3.74	47.5	-2.42	4.65	59.0	24.3
FET		4.70			6.42		36.5
PIF		7.17					
TLC-SB	7.30	5.57	76.3	-2.48			
RV-SB	2.25	2.41	106.9	0.38			
RV%TLC	33.85	43.20	127.6	1.71			
FRC	3.58	3.68	102.7	0.16			
DLCOc	10.60	6.75	63.6	-2.72			
VA	7.15	5.44	76.1				
KCO	1.45	1.24	85.4	-0.89			
KCOc	1.45	1.24	85.4	-0.89			
VIN	4.90	3.16	64.5	-3.11			
Hb		14.60					



Bloods: normal

Histamine challenge: not possible

HRCT: normal

Q1: The most likely diagnosis is

A COPD due to cannabis smoking

B toxic pneumonitis from chlorine exposure

C **irritant induced asthma**

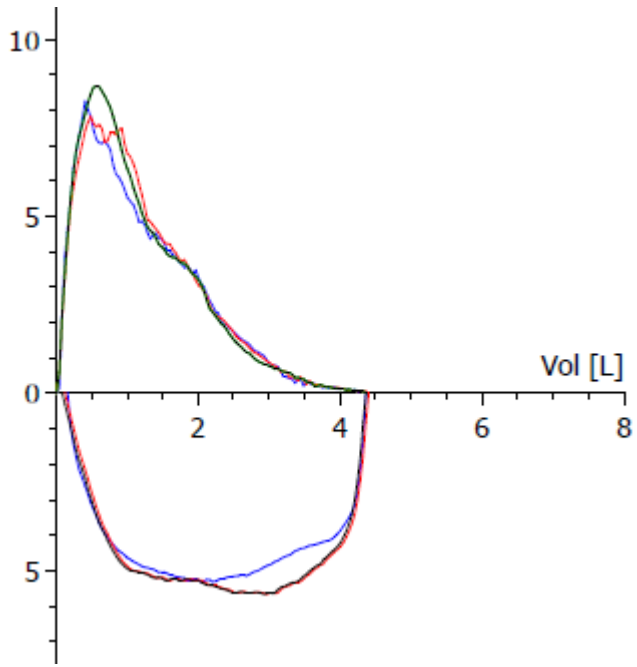
D inducible laryngeal obstruction

E breathing pattern disorder (due to PTSD)

What happened next?

Mx plan: analgesia, physiotherapy

	Pred LI	Pred UI	Test1	% Pred	S.R.
Date			14/05/18		
FEV 1	2.78	4.45	3.00	83.0	-1.21
FVC	3.51	5.51	4.37	97.0	-0.22
VC MAX	3.78	5.62	4.37	93.1	-0.58
FEV1%M	66.05	89.65	68.61	88.1	-1.28
PEF	6.80	10.77	8.65	98.5	-0.11
MEF 75	4.88	10.50	5.58	72.5	-1.24
MEF 50	2.58	6.92	2.29	48.3	-1.86
MEF 25	1.93	1.93	0.58	29.9	
FET			8.94		
PIF	6.59	6.59	5.66	85.9	



Irritant induced asthma (‘RADS’)
Histamine challenge now normal
Slow withdrawal of medication
IIDB
Pursuing personal injury claim

Reactive airways dysfunction syndrome (RADS)

original criteria

- documented absence of prior respiratory symptoms
- onset after a single, toxic exposure
- onset within 24 hours
- symptoms consistent with asthma
- **NSBHR**
- +/- airflow obstruction
- other pulmonary disease excluded (CXR normal)

Brooks. Chest 1985;88:376-384

Irritant induced asthma ('RADS')

Far less common than first thought

Very high levels of exposure required

Hard to sustain diagnosis without evidence of BHR

Even more difficult to diagnose with pre-existing asthma as already evidence of BHR

No hypersensitivity so can tolerate same agent (in contrast with occupational asthma)

Often referred with a label and a legal case
Diagnosis may or may not be made appropriately
Consider if it is helpful to undo diagnosis

Fear is often a reason not to return to work

52M Self employed sub-contractor for larger company

January 2014 A+E
reported exposure to “Lithofin StainStop” (LSS)
laying tiles in a bathroom and kitchen refurbishment
a small room with no ventilation in the area
operatives from a larger company used spraying equipment to apply LSS throughout the area he was working in.

“He has, he told me, previously used this product himself and reports that the data sheets state that it should be applied by cloth and not to be sprayed, as if inhaled can cause symptoms. He recalls that he became acutely unwell.”

Cough, choking, vomiting ++, dizziness, headache

2/52 inpatient stay, 24h HDU
high dose steroids
lost 30 kg (previously weighed 123kg)

Feb 2015 RBH clinic
off steroids for some months
9 months to regain his fitness enough to return to work (lost business)
SOBOE on minimal exertion (previously fit)
“prone to chest infections”

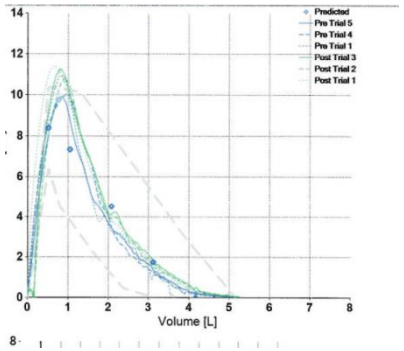
Clinical examination normal. Oxygen saturations 98% on air.

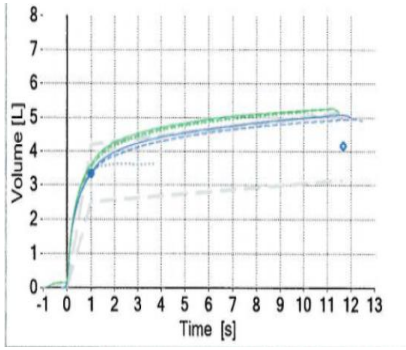
06/14 (local)

FEV_1 2.81L (83%)
FVC 4.71L (112%)
Ratio 60%
Gas transfer 65% predicted

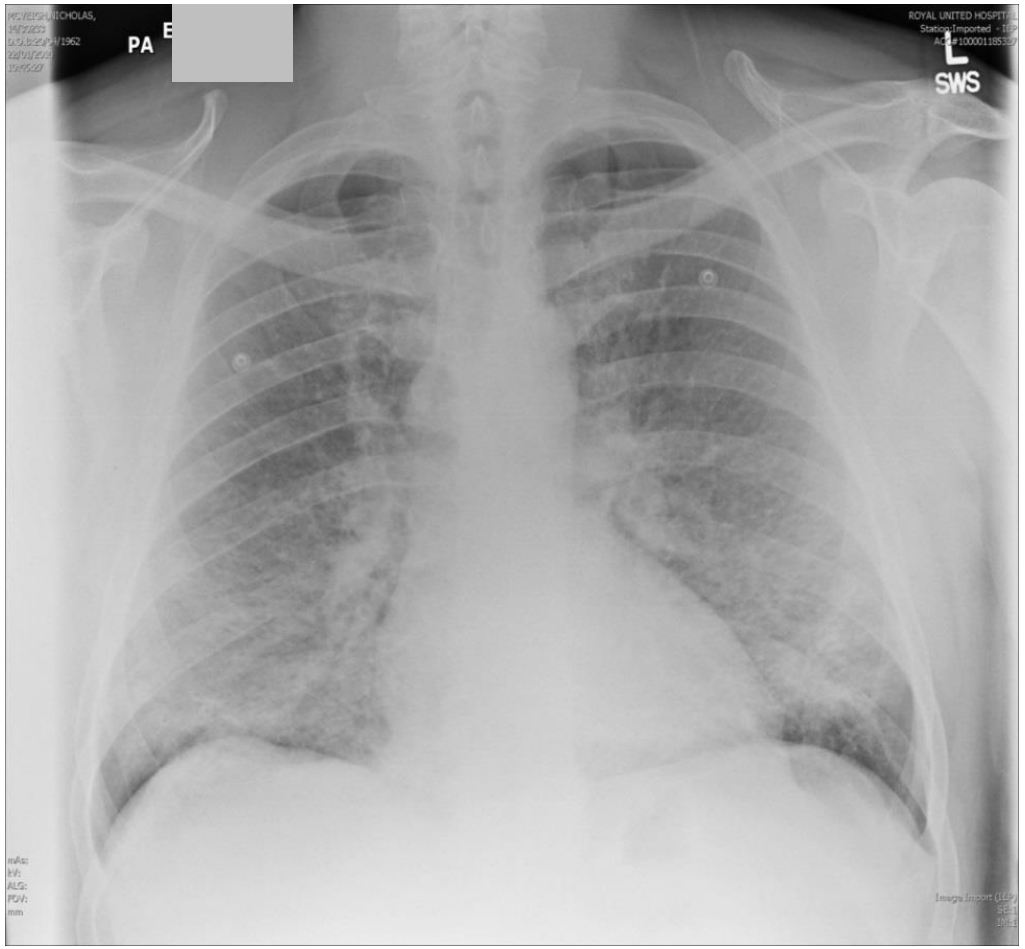
Lung function 02/15

FEV_1 3.38L (101%)
FVC 5.08L (122%)
Ratio 66%
low FEFs, scalloping (ex-smoker)
No reversibility

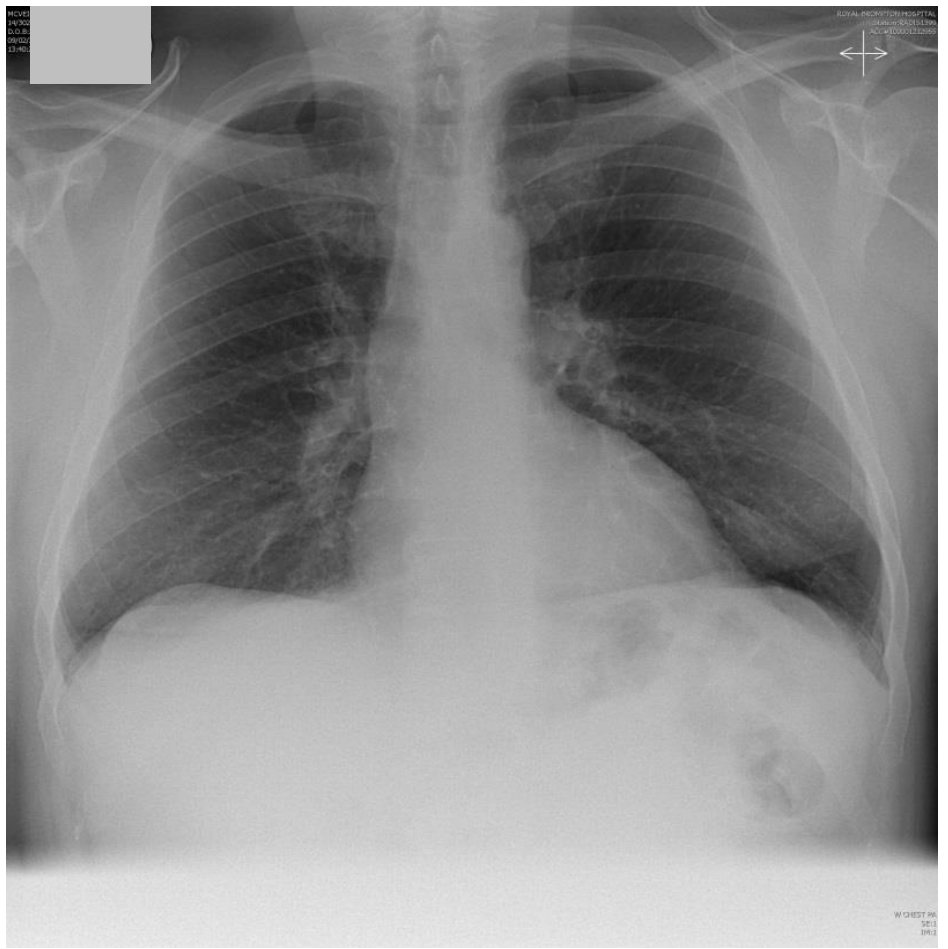




Jan 14



Feb 15



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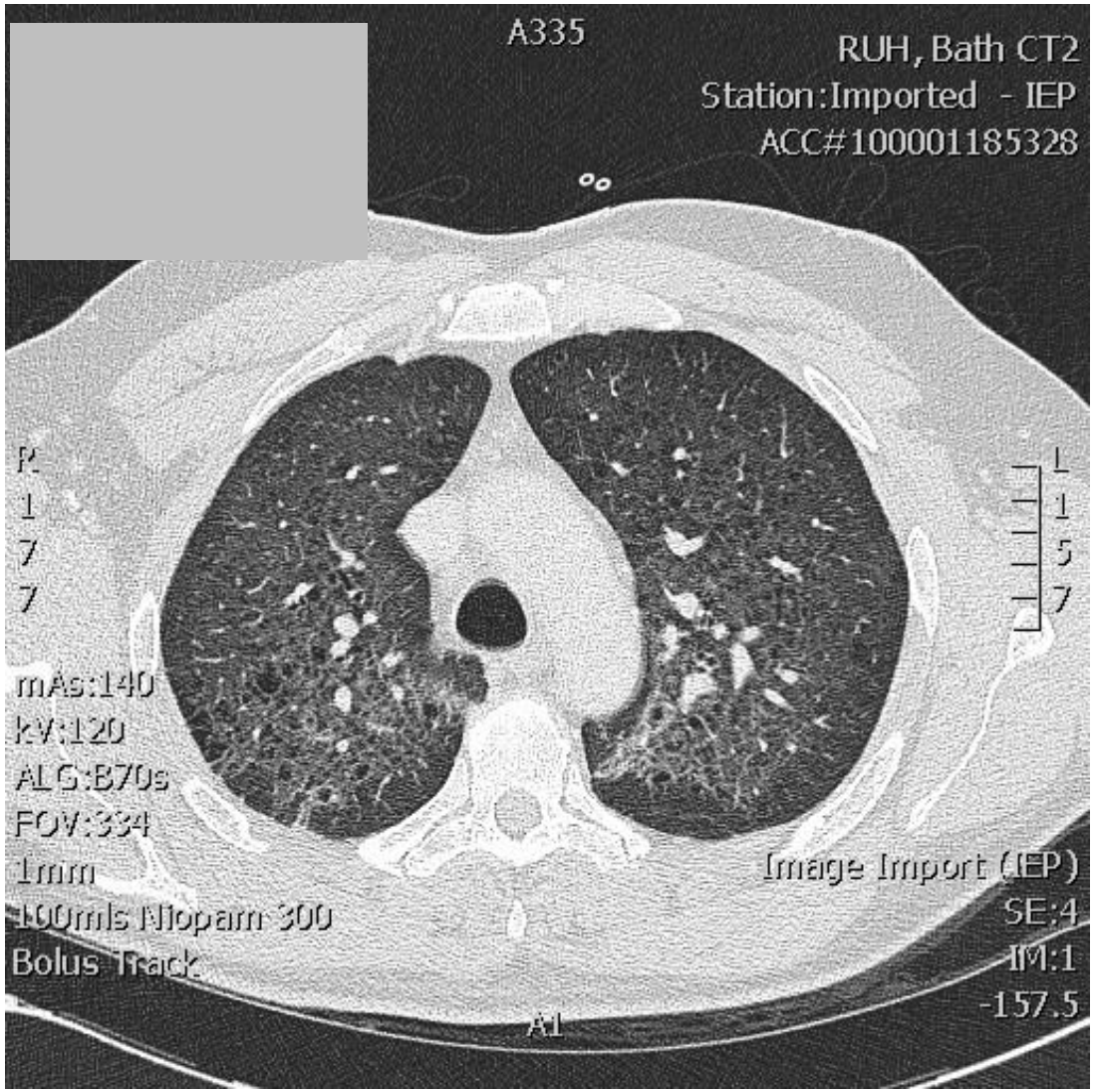
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Bolus Track
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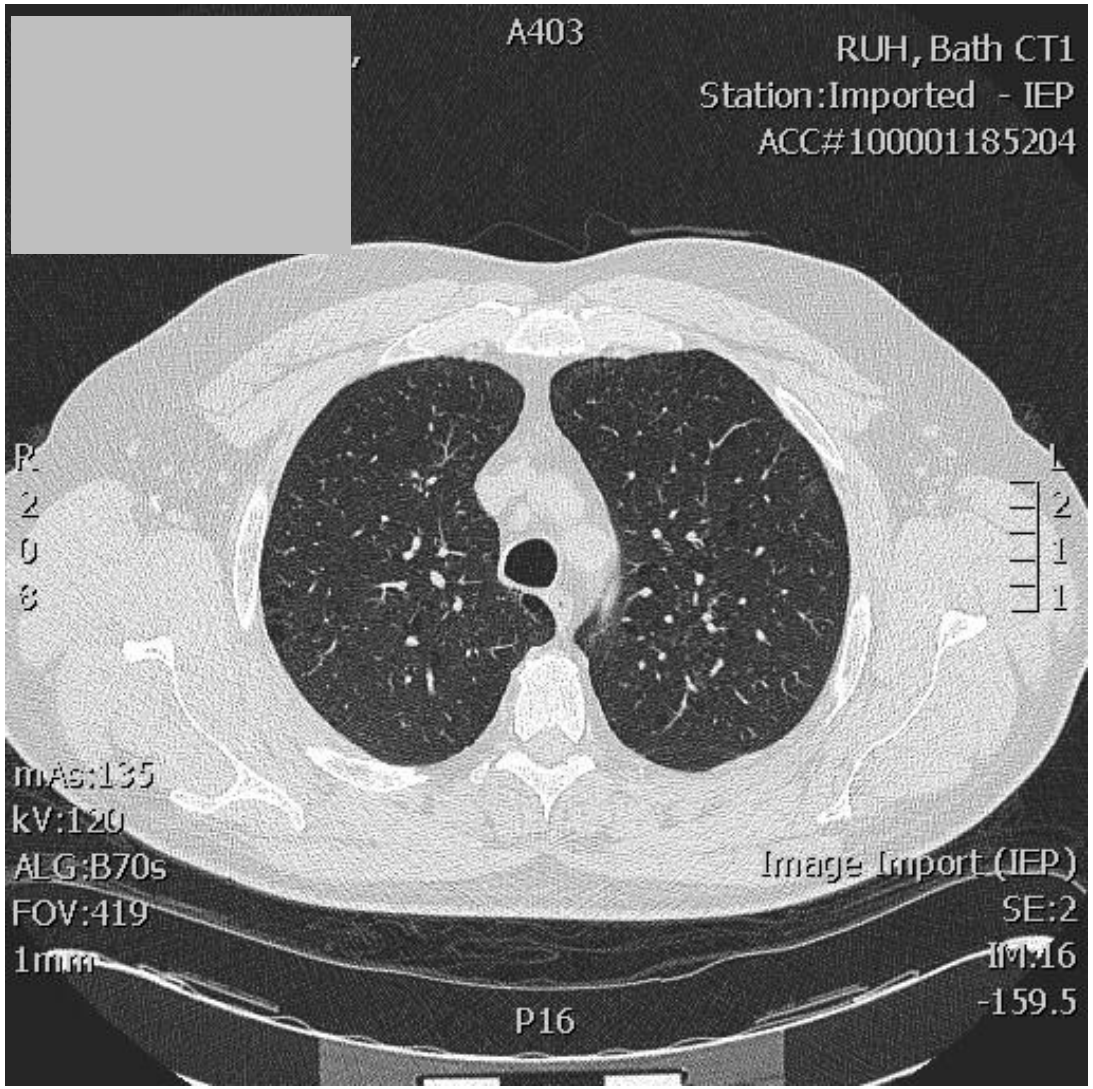
A1

Aug 14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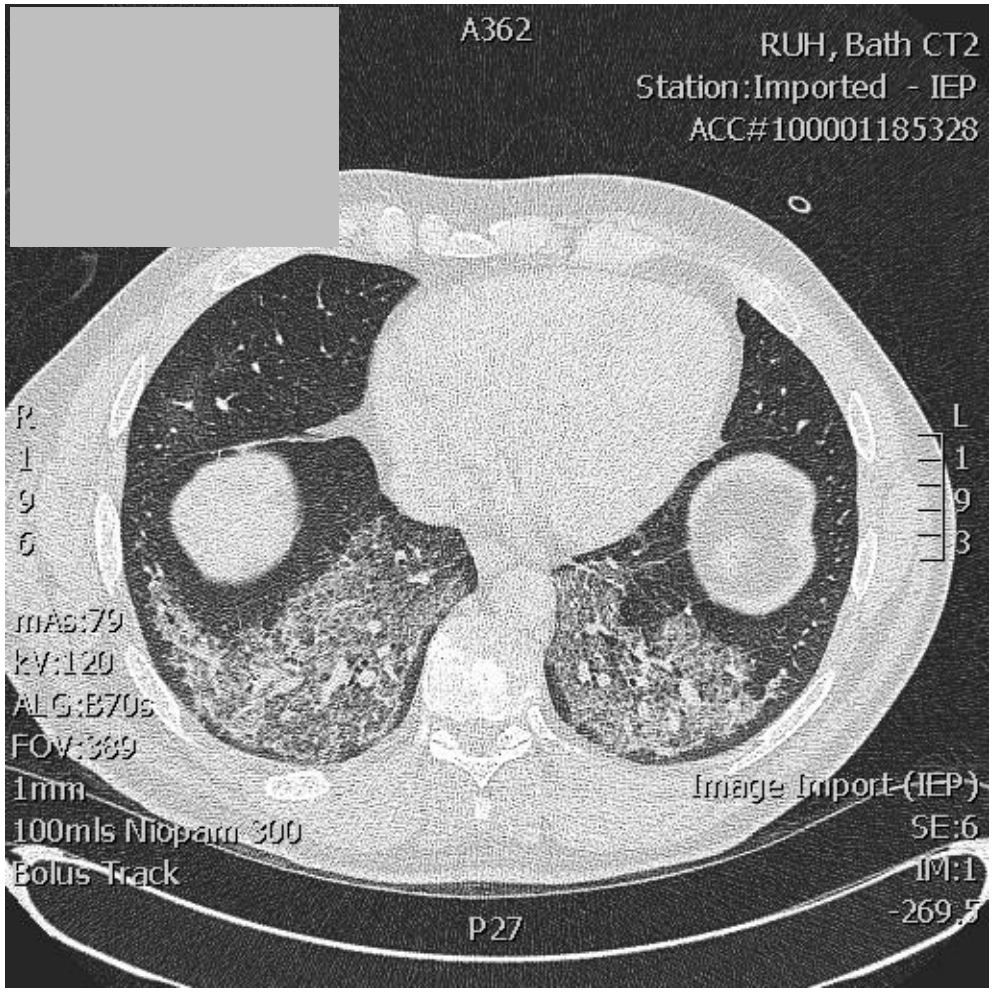
P16

Jan 14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R
L
mAs:79
kV:120
ALG:B70s
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100mls Niopam 300
Bolus Track
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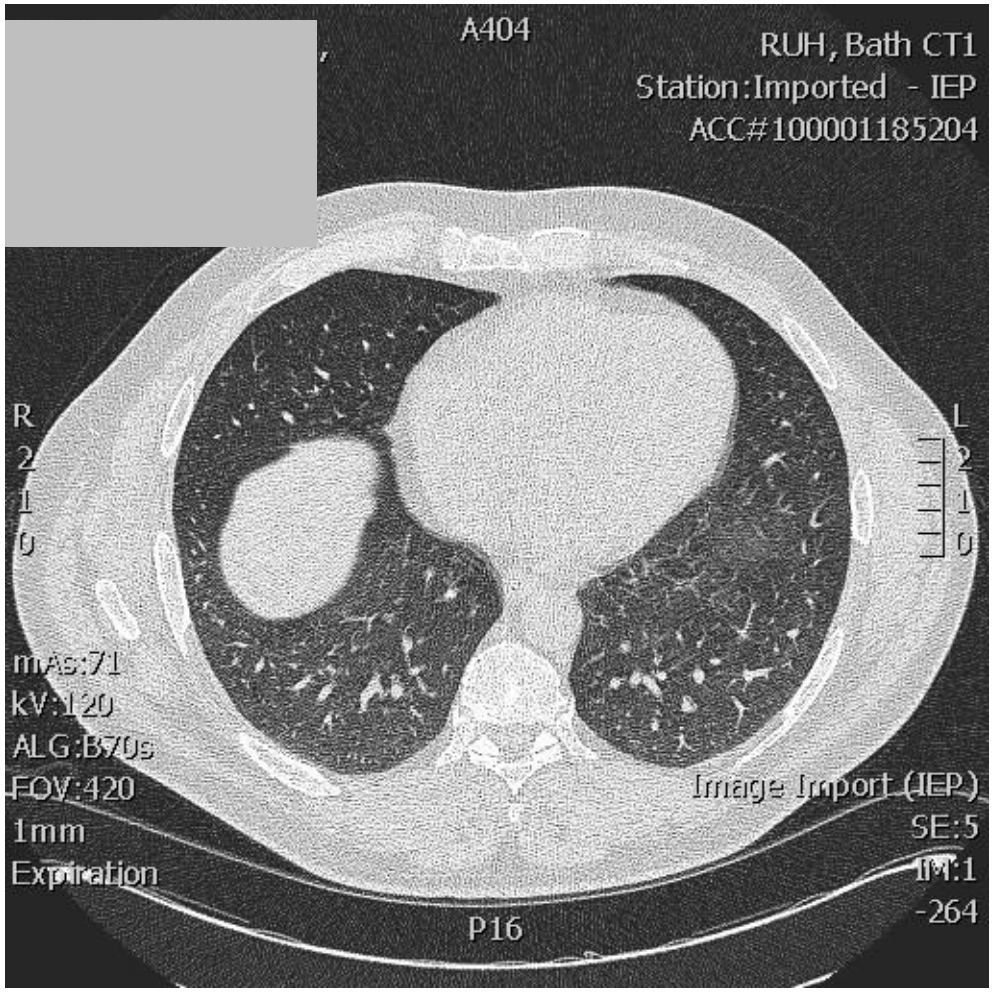
P27

Aug 14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L
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Expiration
Image Import (IEP)
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IM:1
-264
P16

Q2: The most likely diagnosis is

A irritant induced asthma

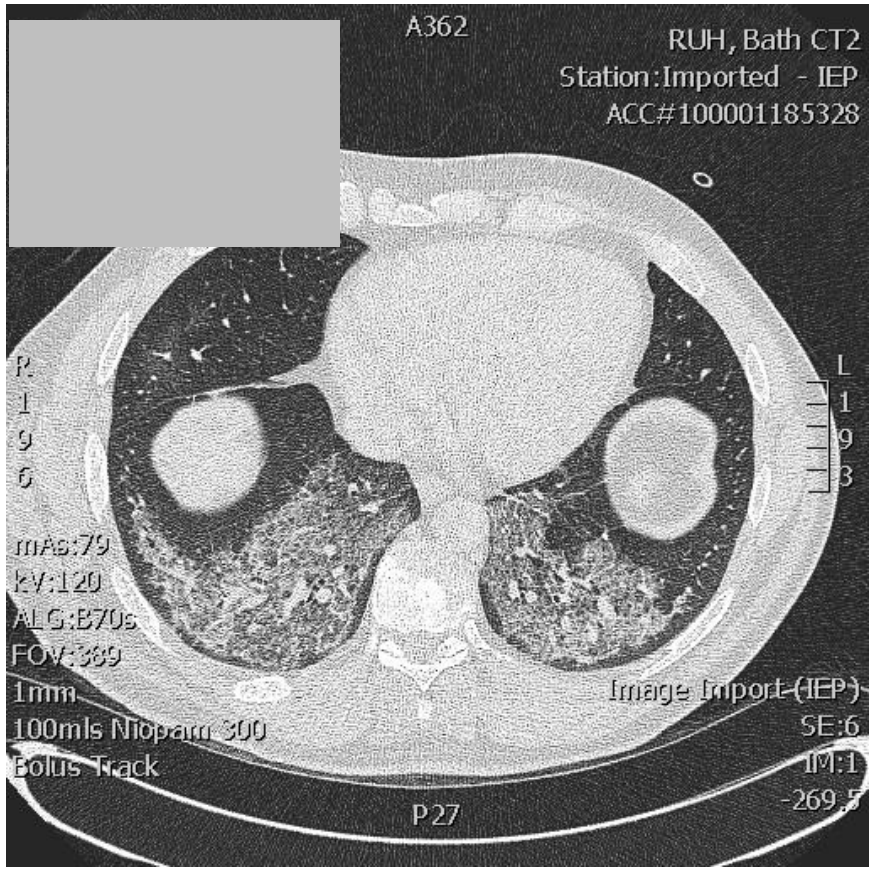
B **toxic pneumonitis**

C bronchiolitis obliterans

D organising pneumonia

E breathing pattern disorder (due to PTSD)

Jan 14



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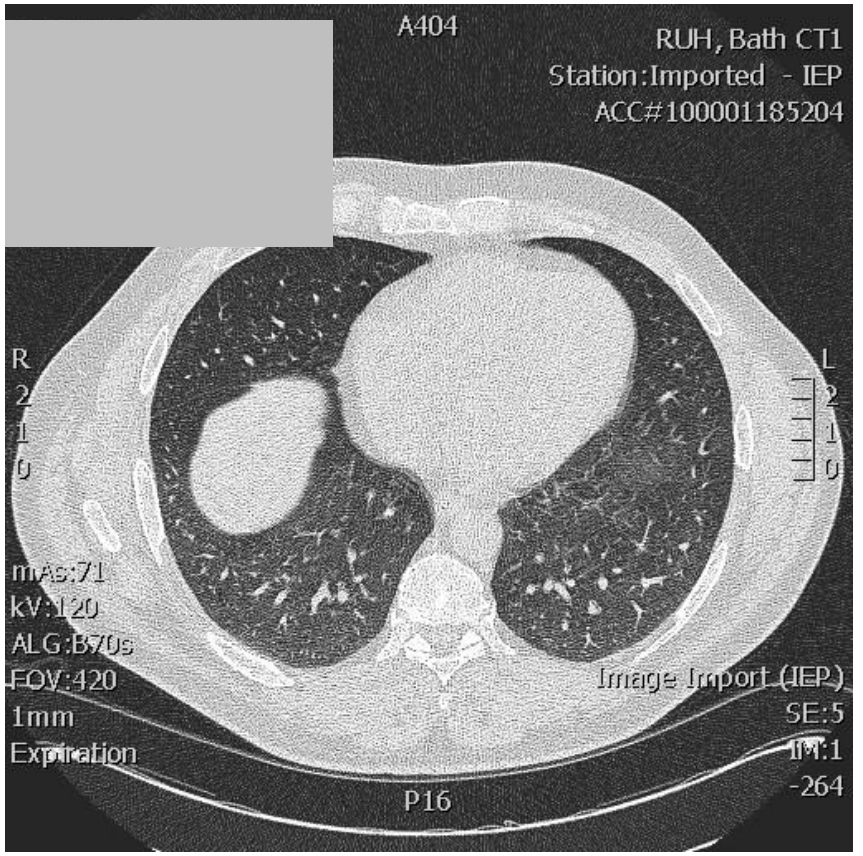


Imaging

His imported CT scans show an acute lung injury/diffuse alveolar damage, consistent with inhalation of toxic material

Changes resolved on the August scan and CXR today with no residual fibrosis.

material safety data sheet for Lithofin StainStop:

2.1. Classification of the substance or mixture

Classification according to 67/548/EEC or 1999/45/EC

R10
Xn; R65

R-phrases

10	Flammable.
65	Harmful: may cause lung damage if swallowed.

SECTION 7: Handling and storage

7.1. Precautions for safe handling

Advice on safe handling

While spraying wear respiratory protection.
Use only in thoroughly ventilated areas.
Use solvent-resistant equipment.
Take the usual precautions when handling with chemicals.

General protective measures

Avoid contact with eyes and skin
Do not inhale gases/vapours/aerosols.

8.2. Exposure controls

Respiratory protection

Breathing apparatus in the event of aerosol or mist formation.
Breathing apparatus in the event of high concentrations.
Short term: filter apparatus, filter A
Multi-purpose filter ABEK
(EN 14387, 133, 140, 149)

naphtha (petroleum), hydrotreated heavy

a cause of toxic pneumonitis by inhalation

data sheets inadequate

46F University Administrator

Breathlessness and chest tightness on exertion since pneumonia in 2014

Attacks of ‘brittle’ asthma:

- dyspnoea, chest tightness, throat closing, stridor and wheeze
- triggers include perfumes (Lynx), strong smells (students), bonfires
- “back-to-back” nebulisers and 3-5/7 prednisolone
- paramedics every fortnight +/- admission; sickness absence ++

Symbicort 400/12
Seretide 500
tiotropium 18mcg
salbutamol nebuliser

asthma treatment generally unhelpful

CT thorax , PFTs, histamine challenge all normal

Q3: The most likely diagnosis is

A occupational asthma due to perfume

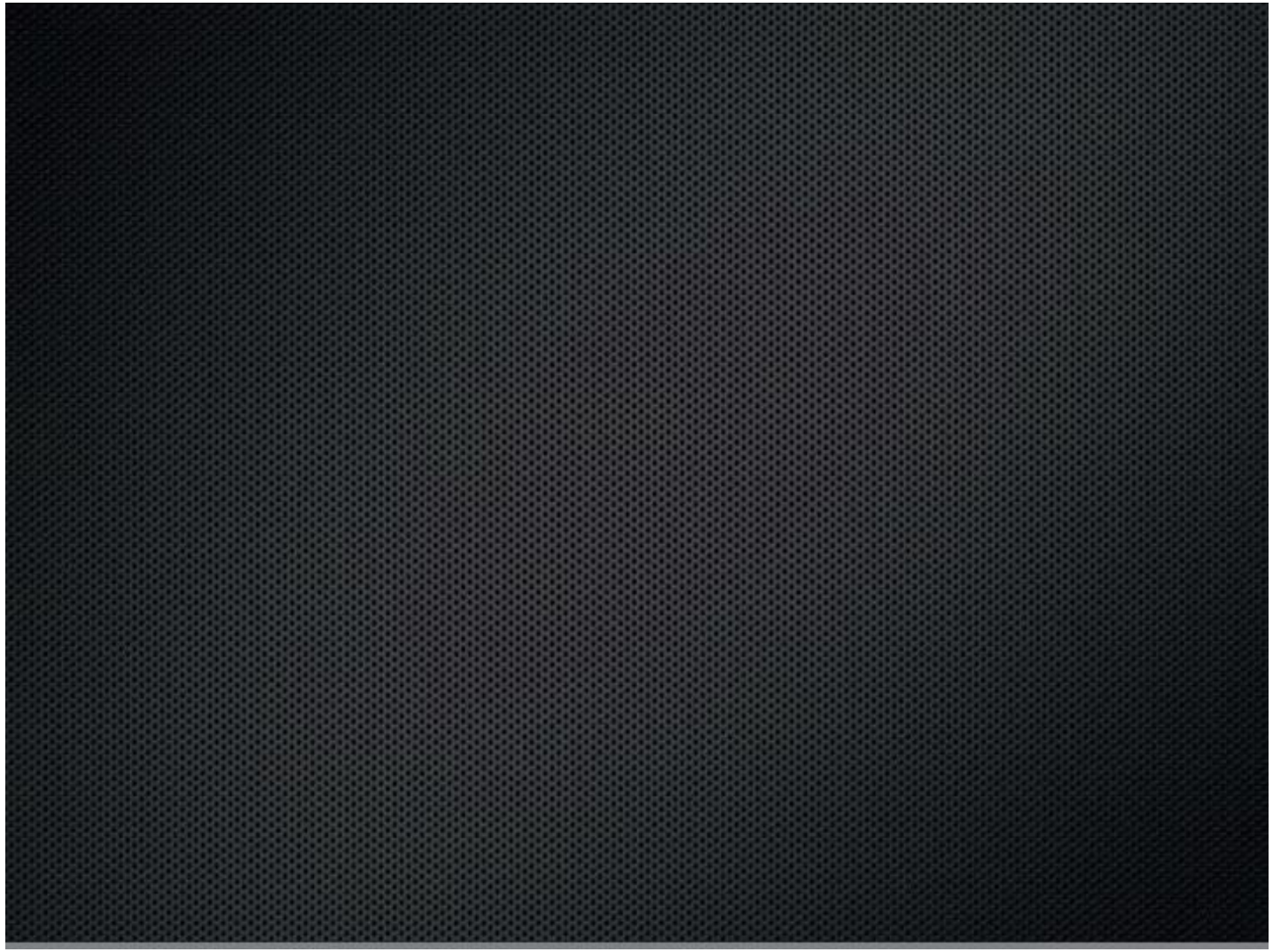
B irritant induced asthma

C severe (brittle) asthma

D **inducible laryngeal obstruction**

E breathing pattern disorder

Continuous laryngeal endoscopy with provocation; classical appearances



Courtesy of Dr J Hull, RBH

Inducible laryngeal obstruction (ILO): in the occupational setting

symptoms not typical for asthma (occupational or otherwise)

rapid onset of sx with exposure, often perfumes
variable (poor) response to asthma treatment
“dying”; anaphylaxis, allergy, brittle asthma, “RADS”

sickness absence, restricted working areas
significant disruption to general life

increasingly recognised
provocation challenge with real-time objective assessment to confirm diagnosis (RBHT)
NB: BPD, hyperventilation syndrome etc.

non-pharmaceutical management; “cure” is possible but support often needed

International consensus conference nomenclature on ILO. Christensen et al. European Resp Rev 2015 24 445-450

26M BBC journalist working in foreign news

Exposure on 22nd October 2016 to toxic fume in Northern Iraq

2 hours outside in ‘cloud’
6 hours inside in a tent with gas seeping through
US army wearing gas masks

Eyes and nose streaming;
Non-productive cough
Dyspnoea ++; chest tightness; wheeze

CXR: “cloudy”
Low oxygen levels
Not fit to fly for a week

Returned to London 28th October 2016
Ongoing chest tightness with cold air, exertion, talking

WORLDPOST 10/22/2016 08:45 am ET | **Updated** Oct 22, 2016

ISIS Sets Sulfur Plant Ablaze In Northern Iraq, Choking The Air With Deadly Chemicals

At least two civilians have died and more than 100 sought medical attention over the attack, a local medical worker said.

n=c.1000 treated

Patrick Osgood
@PatrickOsgood

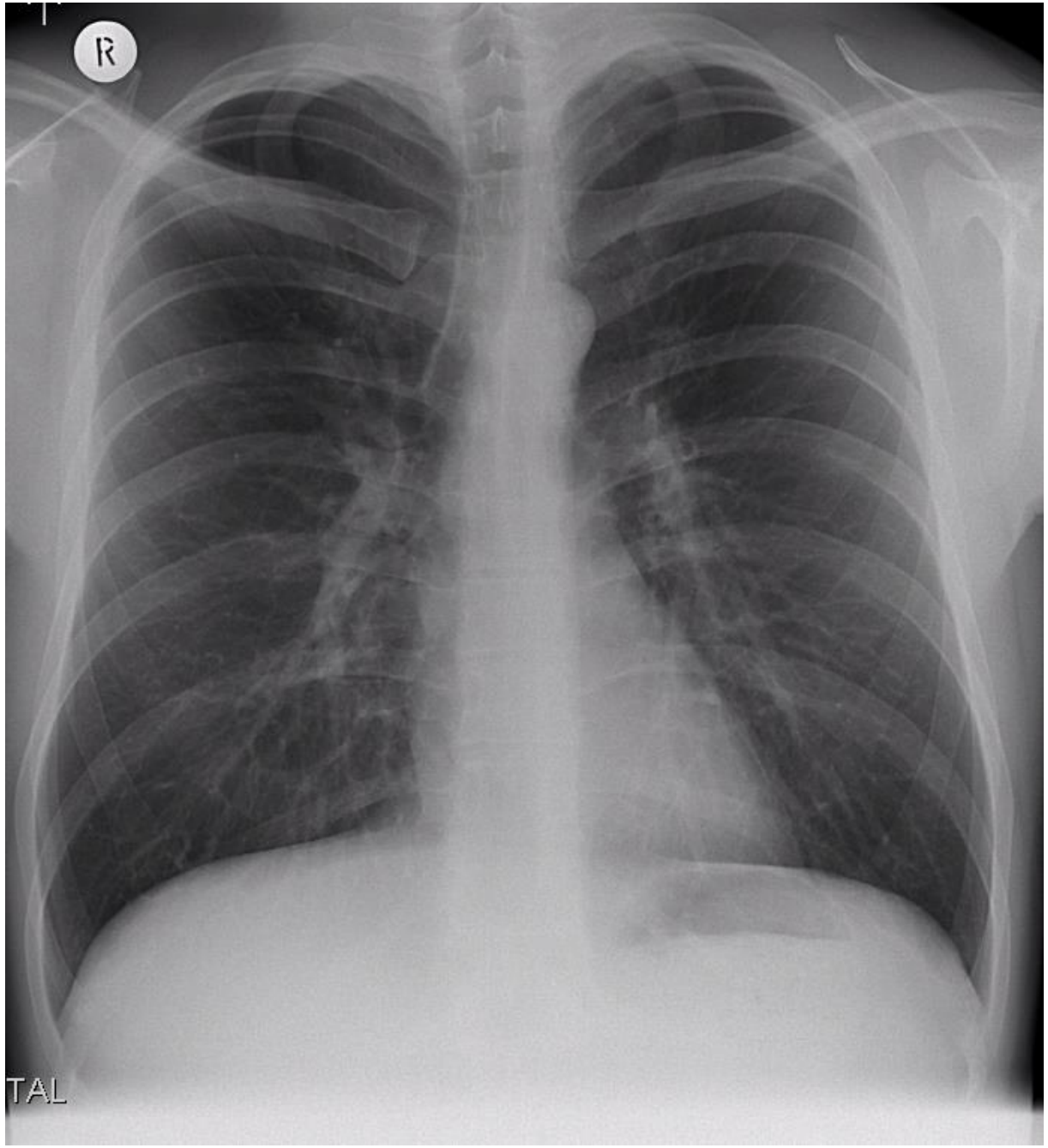
Follow

ISIS torched part of the Mishraq sulphur plant between Qayarah and Mosul this afternoon. Toxic fumes over area now much worse. (image: NASA)

CREDIT: THAIER AL-SUDANI/REUTERS

Smoke rises from a sulfur plant south of Mosul after ISIS militants set it on fire, releasing toxic smoke in the area Friday.

27th February 2017



	27/02/17	27/02/17 Post BD
FEV1	4.54 (97%)	5.39 (115%) *
FVC	6.55 (117%)	6.94 (124%)

*19% change but not reproducible

	27/02/17	**27/02/17 Post BD**	**20/03/17**
FEV1	4.54 (97%)	5.39 (115%) *	5.70 (124%)
FVC	6.55 (117%)	6.94 (124%)	7.17 (130%)

Unable to perform histamine challenge (spirometry not reproducible)

Q4: The most likely diagnosis is

A occupational asthma due to chlorine gas

B irritant induced asthma

C toxic pneumonitis from chlorine exposure

D inducible laryngeal obstruction

E **breathing pattern disorder (due to PTSD)**

Twelve months on……

Much better, but breathlessness and “feeling like can’t get air in”

- Middle East
- South Korea terrorist attack simulation
- Grenfell tower fire

Partly relieved by Becotide (?)
Still unable to perform reproducible spirometry

- Reassured++
- Stopped all inhalers
- Physiotherapy
- Intervention for PTSD (EMDR)

A general strategy in the clinic: “I’ve not been right since ……”

Establish disease

History:

- a long time may have elapsed since exposure; beware recall bias
- what is known and what is unknown
- new symptoms or exacerbation of pre-existing disease?
- feeling for the psychological impact (PTSD; legal involvement)

Objective tests:

- lung function, BHR, CPET, Provocation challenge, breathing pattern review

Consider attribution: extent of exposure and biological plausibility

- rarely possible (absence of comparator info); esp for common symptoms
- unless it is clear, avoid the issue as far as possible (‘we’ll never really know’)
- beware of attributions that you cannot robustly defend (leave that to others)

Management

- is it safe for this person to return to their normal activities?
- how can this best be ‘managed’ moving forward?

Summary

LUNGS AT WORK
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Solubility of agent useful in predicting effects

Majority with acute inhalation injury have no long-term adverse health outcomes

Minority develop longer-term complications

Management is largely supportive

Use objective tests to evaluate the present or absence of disease

Consider ILO for those with upper airway symptoms

Irritant induced asthma (“RADS”) is probably over-diagnosed

Avoid polypharmacy, especially prolonged steroids

Psychological factors may be important in recovery

Thank you!