Hypersensitivity Pneumonitis: Pigeons, Farmers and Hot Tubs, Oh My

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Case Presentation

• 50y F never smoker presents to PCP with progressive DOE, nonproductive cough and fatigue over the last 3 months

• History reveals she has been working in her husband’s parakeet shop more since he became ill several months ago

• Exam: bilateral diffuse fine crackles

• Labs: normal

• PFTs: FVC 64% predicted, DLCO 65% predicted

• CXR: bilateral interstitial and patchy opacities
Case Presentation

HRCT: patchy GGO, mid lung zone predominance, mosaic attenuation and air trapping
Hypersensitivity Pneumonitis

- Airway centered inflammation induced by an exaggerated immune response to an inhaled foreign substance
- Syndrome results from repeated exposure to variety of organic particles
- Up to 30% have no identifiable exposure
- 2 hit hypothesis (antigen exposure + genetic predisposition)

Selman, Pardo, King, AJRCCM 2012

Journal of Allergy and Clinical Immunology 2019 1431295-1301DOI: (10.1016/j.jaci.2018.09.040)
Hypersensitivity Pneumonitis: Categories

Historical Categories

- **Acute**
  - hours

- **Subacute**
  - weeks

- **Chronic**
  - months

ATS Guidelines 2020

- **Nonfibrotic**
- **Fibrotic**

Presence of fibrosis is the primary determinant of prognosis.
Exposure to Environmental Antigens

- Immune mediated reaction in susceptible and sensitized people to a large variety of inhaled environmental antigens
- Exposure can occur at work, home, hobbies
- Large list of HP inducers, 3 main groups
  - Animals (mostly birds)
  - Microbes (fungi, mold, bacteria)
  - Chemicals (solvents, drugs)
- Identification of source is crucial for management

Vasakova et al. AJRCCM 2019
# Examples of HP Inducers

<table>
<thead>
<tr>
<th>Antigen</th>
<th>Antigen source</th>
<th>HP variant</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Bacteria</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Thermophilic actinomycetes</td>
<td>Mouldy hay and straw</td>
<td>Farmer’s lung</td>
</tr>
<tr>
<td>Klebsiella oxytoca</td>
<td>Humidifiers</td>
<td>Humidifier’s lung</td>
</tr>
<tr>
<td>Thermophilic actinomycetes</td>
<td>Sugar cane dust</td>
<td>Bagassosis</td>
</tr>
<tr>
<td><strong>Mycobacteria</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mycobacterium avium complex</td>
<td>Outdoor hot tubs</td>
<td>Hot-tub lung</td>
</tr>
<tr>
<td>Mycobacterium immunogenenum</td>
<td>Metal-working fluid</td>
<td>Machine operator’s lung</td>
</tr>
<tr>
<td><strong>Fungi</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Absidia corymbifera</td>
<td>Mouldy hay and straw</td>
<td>Farmer’s lung</td>
</tr>
<tr>
<td>Trichosporon cutaneum</td>
<td>Indoor households</td>
<td>Summer-type HP</td>
</tr>
<tr>
<td>Penicillium roqueforti</td>
<td>Cheese washing and/or industrial source</td>
<td>Cheese-worker’s lung</td>
</tr>
<tr>
<td><strong>Animal proteins</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Feathers and excrements</td>
<td>Birds</td>
<td>Bird breeder’s or fancier’s lung</td>
</tr>
<tr>
<td>Serum and urine</td>
<td>Rats</td>
<td>Rat protein alveolitis</td>
</tr>
<tr>
<td><strong>Plant proteins</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nut dust</td>
<td>Processing of tiger nuts</td>
<td>Tiger nut alveolitis</td>
</tr>
<tr>
<td>Soy dust</td>
<td>Soy foods</td>
<td>Soy dust alveolitis</td>
</tr>
<tr>
<td><strong>Enzymes</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Phytase</td>
<td>Animal feed</td>
<td>Phytase alveolitis</td>
</tr>
<tr>
<td>Enzymes from Bacillus subtilis</td>
<td>Detergent industry and/or cleaning products</td>
<td>Detergent worker’s lung</td>
</tr>
<tr>
<td><strong>Chemicals</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Toluene diisocyanate, methylene diphenyl isocyanate and hexamethylene diisocyanate</td>
<td>Paint and/or varnish</td>
<td>Isocyanate lung</td>
</tr>
<tr>
<td>Acid anhydrides</td>
<td>Plastic industry</td>
<td>Acid anhydride alveolitis</td>
</tr>
<tr>
<td><strong>Metals</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Zinc vapour</td>
<td>Zinc welding</td>
<td>Zinc vapour alveolitis</td>
</tr>
<tr>
<td>Zirconium</td>
<td>Ceramic tile work</td>
<td>Zirconium silicate alveolitis</td>
</tr>
</tbody>
</table>
Why do only some people develop HP?

Antigen is not enough!

Type and quantity of antigen
Exposure dose

Environmental cofactors
Host genetic and immune factors

Selman et al. ARJCCM 2012
Genetic Susceptibility to HP

- Most polymorphisms involved in antigen presentation (MHC)

- Fibrotic HP shares some genetic polymorphisms with other ILDs
  - Telomere length mutations
  - MUC5B

Familial clusters reported even when affected family members live far apart
- Okamoto et al. Respiration 2013

<table>
<thead>
<tr>
<th>Genes and Genetic Variants</th>
<th>Population Race or Nationality</th>
<th>First Author (Year) (Reference)</th>
</tr>
</thead>
<tbody>
<tr>
<td>MHC II polymorphisms</td>
<td></td>
<td></td>
</tr>
<tr>
<td>HLA-DR3</td>
<td>White</td>
<td>Rittner (1983) (63)</td>
</tr>
<tr>
<td>HLA-DR7</td>
<td>Mexican</td>
<td>Selman (1987) (64)</td>
</tr>
<tr>
<td>HLA-DQ3</td>
<td>Japanese</td>
<td>Ando (1989) (65)</td>
</tr>
<tr>
<td>HLA-DRB1*04</td>
<td>Mexican</td>
<td>Falfán-Valencia (2014) (66)</td>
</tr>
<tr>
<td>MHC II haplotypes</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Increased DRB1<em>1205-DQB1</em>0301; decreased DRB1<em>0802-DQB1</em>0402</td>
<td>Mexican</td>
<td>Camarena (2001) (67)</td>
</tr>
<tr>
<td>Proteasome and transporter polymorphisms</td>
<td></td>
<td></td>
</tr>
<tr>
<td>PSMB3 KQ</td>
<td>Mexican</td>
<td>Camarena (2010) (68)</td>
</tr>
<tr>
<td>MHC5B</td>
<td>Mexican</td>
<td>Aquino-Galvez (2008) (69)</td>
</tr>
<tr>
<td>Mucin polymorphisms</td>
<td></td>
<td></td>
</tr>
<tr>
<td>MUC5B rs5705950</td>
<td>White</td>
<td>Ley (2017) (70)</td>
</tr>
<tr>
<td>Telomere length and mutations</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Telomere length &lt;10th percentile</td>
<td></td>
<td>White</td>
</tr>
<tr>
<td>Telomere-related gene mutations</td>
<td></td>
<td>White</td>
</tr>
<tr>
<td>Antiprotease polymorphisms</td>
<td></td>
<td></td>
</tr>
<tr>
<td>TIMP-3-915 TIMP-3-1296 (protective role)</td>
<td></td>
<td>White</td>
</tr>
</tbody>
</table>
Immunopathology of HP

Dysfunctional T Regs
Immunopathology of HP

- **Tregs** from HP patients have no functional suppressive activity

- A second hit may further promote inflammation
  
  - **Viruses** often found in distal airways
    - Dakhama et al. AJRCCM 1999
    - Animal models of virus + HP antigen enhances inflammatory response
      - Girard et al. ERJ 2009
  
  - **Pesticides** potential risk factor for development of farmer’s lung
    - Hoppin et al. Occup Env Med 2007

  - **Air pollution** associated with increased HP cases in India, may reduce MCC and increase inflammation
    - Singh et al. ERJ 2019

  BAL Tregs + activated T cells
Inflammation Abundant in Nonfibrotic HP

- Lymphocytic alveolitis: expansion of T and B lymphocytes in lung tissue
- Formation of granulomas
- Activation of both humoral (Ig) and cellular (T cell) immune responses

Vasakova et al. AJRCCM 2019
Some Patients Develop Fibrotic HP

- Progressive pulmonary fibrosis can occur, which can appear similar to IPF
  - Include HP on differential for new ILD!

- Potential Mechanisms include:
  - Premature senescence
  - Transition from Th1 to Th2 immune response
  - Shortened telomeres / Aging
Diagnosis of HP

• In the context of respiratory sx, diagnosis based on combination of history, imaging and BAL/tissue data

1. Evidence of exposure to antigen (history, serum IgG antibodies)

2. Radiologic patterns consistent with HP

3. Lymphocytosis in BAL (>30%)

4. Histopathologic patterns consistent with HP

<table>
<thead>
<tr>
<th>Likelihood</th>
<th>Criteria</th>
<th>Next Step in Diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Confident</td>
<td>1,2,3 or 1,2</td>
<td>Lung biopsy not needed in most cases</td>
</tr>
<tr>
<td>Probable</td>
<td>1,3 but CT more c/w other ILD</td>
<td>Lung biopsy needed</td>
</tr>
<tr>
<td>Possible</td>
<td>1 but CT more c/w other ILD</td>
<td>Lung biopsy needed</td>
</tr>
<tr>
<td>Unlikely</td>
<td>none</td>
<td>Lung biopsy may be appropriate</td>
</tr>
</tbody>
</table>

Vasakova et al. AJRCCM 2017
Diagnosis: Evidence of Exposure

• Obtain detailed exposure history

  • Standardized questionnaire may be helpful, though none currently validated

  • Pets (especially birds)
  • Use of feather pillow, duvet, sleeping bag, jacket
  • Wind instrument player
  • History of water damage to home or office, carpet (even if cleaned)
  • Use of hot tub, jacuzzi, sauna, swimming pool
  • Use of air conditioning units, humidifier, air cooler
  • Workplace exposures (lab animals, vet work, barn, horse stable, farming, hay handling, mushroom growing, brewery, winery, metalwork, plastics or epoxy manufacturing, spray painting)
Diagnosis: Evidence of Exposure

- Antigen specific serum IgG antibodies
  - Only markers of sensitization and not disease
  - Can support diagnosis of HP in the right clinical setting
  - Can facilitate identification of responsible antigen
  - Many different commercial tests exist with limited antigens and different sensitivities and specificities

Lacasse et al. AJRCCM 2003
### Radiology of Nonfibrotic HP

<table>
<thead>
<tr>
<th>Confidence</th>
<th>Criteria</th>
</tr>
</thead>
<tbody>
<tr>
<td>Typical of HP</td>
<td>Requires diffuse evidence of: 1 pattern of infiltration (GGO, mosaic attenuation) and 1 pattern of small airway disease (centrilobular nodules, air trapping)</td>
</tr>
<tr>
<td>Compatible with HP</td>
<td>Nonspecific patterns that have been described in HP</td>
</tr>
<tr>
<td>Indeterminate for HP</td>
<td>N/A</td>
</tr>
</tbody>
</table>
Radiology of HP

3 density pattern “headcheese sign”

1. GGO (increased attenuation, inflammation/infiltration) yellow arrow
2. Lobules of decreased attenuation (small airways obstruction) white arrow
3. Normal appearing lung asterisk

- Highly specific pattern for HP

Costabel et al. Nat Rev 2020
Radiology of Fibrotic HP

• Diffuse reticulation, traction bronchiectasis, mid and lower lobe distribution, relative basal sparing, centrilobular nodules, mosaic attenuation

• UIP pattern in up to 1/3 of fibrotic HP patients; air trapping and diffuse distribution can be key discriminatory features

Walsh et al. Eur Radiol 2012
## Diagnosis: HRCT

<table>
<thead>
<tr>
<th>Acute HP and chronic non-fibrotic HP</th>
<th>Chronic fibrotic HP</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Features</strong></td>
<td></td>
</tr>
<tr>
<td>Ground-glass opacities</td>
<td>Reticular opacities, traction bronchiectasis and honeycombing</td>
</tr>
<tr>
<td>Centrilobular nodules of ground-glass attenuation that are small and poorly defined</td>
<td>Superimposed with findings of acute HP (for example, combination of ground-glass opacities, centrilobular nodules and mosaic pattern)</td>
</tr>
<tr>
<td>Areas of decreased attenuation represent a mosaic pattern secondary to air-trapping, corresponding to areas of bronchiolitis</td>
<td>Emphysema, alone or in combination with other features of chronic HP possible</td>
</tr>
<tr>
<td>Head-cheese sign (a combination of ground-glass opacities, mosaic pattern and normal lung tissue)</td>
<td>Thin-walled pulmonary cysts, few and not dominant (may also occur in patients with chronic non-fibrotic HP)</td>
</tr>
<tr>
<td><strong>Distribution</strong></td>
<td></td>
</tr>
<tr>
<td>Mostly diffuse, usually bilateral, sometimes patchy and predominantly in the lower lung areas</td>
<td>Mostly lower lung zone predominance, sometimes diffuse or in mid-to-upper lung zones, with a subpleural and peribronchovascular distribution; usually bilateral, with relative sparing of the lower lung zones</td>
</tr>
</tbody>
</table>

Costabel et al. Nat Rev 2020
Diagnosis: BAL Analysis

- BAL lymphocytosis is characteristic but not specific to HP
  - >20-50% depending on the study, ATS Guidelines recommend >30%

- Not sensitive
  - May be affected by concurrent infections, steroids, etc.

- Not specific
  - Can be seen in sensitized individuals who do not have disease

- Low CD4:CD8 ratio (in comparison to sarcoidosis)

- Large systematic review recently found % BAL lymphocytes higher in fibrotic and nonfibrotic HP compared to IPF and sarcoidosis
  - >20% BAL lymphocytes distinguished:
    - Fibrotic HP from IPF (sens 69%, spec 61%)
    - Nonfibrotic HP from IPF (sens 95%, spec 61%)
    - Fibrotic HP from sarcoidosis (sens 69%, spec 26%)
    - Nonfibrotic HP from sarcoidosis (sens 95%, spec 26%)

Patolia et al. Annals ATS 2020
Costabel et al. Nat Rev 2020
Diagnosis: Tissue Sampling

- **Transbronchial Biopsy**
  - ATS Guidelines recommend TBBx for diagnosing nonfibrotic HP > fibrotic HP based on higher yield with more diffuse disease
    - May be due to higher yield of granulomas in nonfibrotic HP
    - Diagnostic yield only ~50% but those pts were spared lung biopsy

- **Transbronchial Cryobiopsy**
  - Systematic review showed higher diagnostic yield as compared to TBBx
    - 11% bleeding, 11% PTX
      - Chami et al. Annals ATS 2021
    - ATS Guidelines recommend consideration in experienced centers

- **Surgical Lung Biopsy**
  - Higher morbidity and mortality but also higher diagnostic yield
  - Reasonable to consider once other diagnostic tests fail to make diagnosis

ATS Guidelines, AJRCCM 2020
Pathology of Nonfibrotic HP

Typical histopathogical features of HP:

1. Bronchiolocentric pattern

2. Cellular interstitial infiltrate (arrow)

3. Granulomatous inflammation:
   - Loosely formed granulomas (arrowhead)
   - Multinucleated giant cells (asterisk)

Confidence Criteria

<table>
<thead>
<tr>
<th>Confidence</th>
<th>Criteria</th>
</tr>
</thead>
<tbody>
<tr>
<td>Typical HP</td>
<td>1,2,3</td>
</tr>
<tr>
<td>Probable HP</td>
<td>1+2</td>
</tr>
<tr>
<td>Indeterminate HP</td>
<td>1 or 2</td>
</tr>
</tbody>
</table>

H&E, 100x

Costabel et al. Nat Rev 2020
Pathology of Fibrotic HP

**Typical histopathological features of HP:**

- Bronchiolocentric fibrosis with dense fibrosis adjacent to bronchiole (br = bronchovascular bundle)
- Alveolar septal thickening

Vasakova et al. AJRCCM 2017
ATS Diagnostic Algorithm

Patient with newly detected interstitial lung abnormalities on chest imaging

Exposure assessment* and chest HRCT scan**

BAL with lymphocyte cellular analysis, with or without TBLB***

Exposure identified AND typical HP pattern on HRCT AND BAL lymphocytosis

Multidisciplinary discussion

High-confidence diagnosis of HP

All other combinations of exposure, HRCT, BAL, and TBLB findings

Unclear diagnosis

Multidisciplinary discussion

Consider TBLC## or SLB##

Multidisciplinary discussion

Diagnosis of HP per Figure 6

Reconsider exposures

ATS Guidelines, AJRCCM 2020
## ATS Diagnostic Algorithm

![ATS Diagnostic Algorithm Diagram](ATS DIAGNOSTIC ALGORITHM)

<table>
<thead>
<tr>
<th>History of exposure and/or serum IgG testing</th>
<th>Exposure +</th>
<th>Exposure -</th>
<th>Exposure +</th>
<th>Exposure -</th>
<th>Exposure +</th>
<th>Exposure -</th>
</tr>
</thead>
<tbody>
<tr>
<td>No BAL or BAL without lymphocytosis and either no histopathology or indeterminate histopathology</td>
<td>Moderate confidence</td>
<td>Low confidence</td>
<td>Low confidence</td>
<td>Not excluded</td>
<td>Not excluded</td>
<td>Not Excluded</td>
</tr>
<tr>
<td>BAL lymphocytosis without histopathology sampling</td>
<td>High confidence</td>
<td>Moderate confidence</td>
<td>Moderate confidence</td>
<td>Low confidence</td>
<td>Low confidence</td>
<td>Not excluded</td>
</tr>
<tr>
<td>BAL lymphocytosis with indeterminate histopathology</td>
<td>Definite</td>
<td>High confidence</td>
<td>Moderate confidence</td>
<td>Moderate confidence</td>
<td>Low confidence</td>
<td>Not excluded</td>
</tr>
<tr>
<td>Probable HP histopathology</td>
<td>Definite</td>
<td>High confidence</td>
<td>High confidence</td>
<td>Moderate confidence</td>
<td>Moderate confidence</td>
<td>Low confidence</td>
</tr>
<tr>
<td>Typical HP histopathology</td>
<td>Definite</td>
<td>Definite</td>
<td>Definite</td>
<td>Definite</td>
<td>Definite</td>
<td>High confidence*</td>
</tr>
</tbody>
</table>

ATS Guidelines, AJRCCM 2020
**Treatment for Nonfibrotic HP**

- **Elimination** of exposure most important intervention
  - Remove birds or feather bedding from house (antigens may persist despite cleaning)
  - Avoid hot tubs
  - Sterilize humidifiers and vaporizers
  - May require relocation to new job or home

- For acute HP with minimal or transient symptoms, complete antigen avoidance can be sufficient treatment

- Not always possible, in up to 60% cases inducer is never found

Fernandez Perez Chest 2013
Craig Ann Allergy 1992
Treatment for Nonfibrotic HP: Corticosteroids

• If avoidance alone is insufficient, consider steroid course
  • Persistent symptoms (dyspnea, cough, fatigue, weight loss)
  • Abnormal lung function tests
  • Hypoxemia
  • Radiologic evidence of extensive lung involvement

• Prednisone 0.5mg/kg per day
  • Initial dose for 1-2 weeks, then taper over 4-8 weeks
  • Dose and duration not formally studied

• Can accelerate initial recovery, but long-term outcome appears unchanged

Vasakova et al. AJRCCM 2017
Salisbury et al. AJRCCM 2017
Treatment for Nonfibrotic HP: Corticosteroids

- 36 patients with acute farmer’s lung randomly assigned to 8 weeks prednisolone vs. placebo.
- Improvement at one month in DLCO but no significant differences in FVC, FEV1 or DLCO found after that until study terminated at 5 years.

• HP less frequent in smokers or ex-smokers

• Mechanisms unclear, may be related to nicotine dampening macrophage activation, decreasing lymphocyte proliferation and impairing T cell function

• Short term reduces inflammation, long term increases inflammation and fibrosis
Fibrotic HP Associated with Worse Prognosis

Presence and extent of fibrosis associated with worse survival

Treatment for Fibrotic HP: Immunosuppression

- No prospective studies of corticosteroids, but Prednisone 0.5mg daily x 4-8 weeks, tapering to 10mg by 3 months recommended

- MMP and AZA associated with improved lung function and reduction in prednisone dose in multicenter retrospective study

Morisset et al. Chest 2017
• Pts who required immunosuppression had increased mortality

• MMF or AZA associated with less treatment-emergent adverse events

• No difference in lung function decline or survival compared to prednisone alone
Treatment for Fibrotic HP: Rituxan

- Case report of cHP responsive to RTX
- IHC showed scattered CD20 follicular aggregates on background of CD3+ T cells
- No causative agent identified
- Failed steroids and Cytoxan
- Improved lung function after RTX

Lota et al. Thorax 2013
• Rituxan appears safe though response is variable
• 7/23 (30%) had stable/improved FVC 6 months af
Nintedanib in Progressive Fibrosing Interstitial Lung Diseases


• Eligible with progression of ILD within prior 24 months despite treatment (FVC down 10%)

• Excluded pts on AZA, cyclosporine, MMF, tacrolimus, RTX, cyclophosphamide, or steroids >20mg

• At 6 months these meds could be added if clinical deterioration of ILD or CTD

• HRCT eligibility = fibrosing lung disease >10% lung involvement

The following co-existing features were accepted:
• ground glass opacity
• upper lung or peribronchovascular predominance
• mosaic attenuation/air trapping
• centrilobular nodules
Nintedanib for Progressive Fibrosing ILD

**Figure 2. Decline from Baseline in Forced Vital Capacity (FVC).**

Shown is the observed mean change from baseline in FVC over the 52-week trial period in the overall population and in patients with an imaging pattern of usual interstitial pneumonia (UIP) on high-resolution computed tomography in the nintedanib group and the placebo group. The I bars indicate the standard error.
### Section H: Clinical ILD diagnoses

#### Table S2: Clinical ILD diagnoses (grouped) in the overall population

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Nintedanib (n=332)</th>
<th>Placebo (n=331)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypersensitivity pneumonitis</td>
<td>84 (25.3)</td>
<td>89 (26.9)</td>
</tr>
<tr>
<td>Autoimmune ILDs</td>
<td>82 (24.7)</td>
<td>88 (26.6)</td>
</tr>
<tr>
<td>Rheumatoid arthritis-associated ILD</td>
<td>42 (12.7)</td>
<td>47 (14.2)</td>
</tr>
<tr>
<td>Systemic sclerosis-associated ILD</td>
<td>23 (6.9)</td>
<td>16 (4.8)</td>
</tr>
<tr>
<td>Mixed connective tissue disease-associated ILD</td>
<td>7 (2.1)</td>
<td>12 (3.6)</td>
</tr>
<tr>
<td>Other autoimmune ILDs</td>
<td>10 (3.0)</td>
<td>13 (3.9)</td>
</tr>
<tr>
<td>Idiopathic non-specific interstitial pneumonia</td>
<td>64 (19.3)</td>
<td>61 (18.4)</td>
</tr>
<tr>
<td>Unclassifiable idiopathic interstitial pneumonia</td>
<td>64 (19.3)</td>
<td>50 (15.1)</td>
</tr>
<tr>
<td>Other ILDs*</td>
<td>38 (11.4)</td>
<td>43 (13.0)</td>
</tr>
</tbody>
</table>

Data are no (%) of patients.

*Included sarcoidosis, exposure-related ILDs and selected other terms in “Other fibrosing ILDs”.

Flaherty et al. INBUILD, NEJM 2019
Take Home Points About HP

• Immune mediated lung disease triggered by variety of inhaled antigens in susceptible individuals

• Search for the offending inducer and eliminate it

• Immunosuppression may help non-fibrotic HP

• Some people develop progressive fibrosis, and anti-fibrotics may help in fibrotic HP

• Include fibrotic HP in the differential for a new ILD
Take Home Points About HP

*Goal to recognize HP in timely manner to potentially change disease course