Desquamative interstitial pneumonitis is an interstitial lung disease most commonly associated with smoking. It causes respiratory symptoms including indolent cough and dyspnea. Characteristic findings on computed tomography include bilateral ground-glass opacities, septal thickening and preserved structure. Diagnosis is made by tissue sampling, which classically demonstrates alveolar macrophages, and thickened alveolar septa with an eosinophilic infiltrate lined with hyperplastic type II pneumocytes. Treatment is immune suppression with steroids or other agents, and avoiding the causal agent. The case reported describes a 27-year-old woman with no smoking history who worked in a potato chip factory, presenting with cough, dyspnea and dizziness. The patient had characteristic findings on imaging and was diagnosed via biopsy with desquamative interstitial pneumonitis. She improved clinically with reduced exposure and steroid therapy. While food production workers are at risk for respiratory illness, there are no reported cases of desquamative interstitial pneumonitis in this setting.

Key Words: Desquamative interstitial pneumonia; Interstitial pneumonitis; Occupational exposure; Potato chip factory

Learning objectives
• To recognize that clinical findings and imaging alone are insufficient for the diagnosis of desquamative interstitial pneumonitis (DIP); tissue sampling is required.
• To recognize that occupational history may be very important in someone with persistent respiratory symptoms.

CanMEDS Competency: Health Advocate
Pretest
• What histological findings are typical of DIP?
• What is the initial management for DIP?

CASE PRESENTATION
A 27-year-old woman presented to a walk-in clinic with an 18-month history of dry cough and was started on moxifloxacin and salbutamol. Within one month of initial presentation, she presented to the emergency department with worsening cough, exertional dyspnea and dizziness. Her medical history was notable only for wisdom teeth extraction, and there was no personal or family history of respiratory disease. She had no allergies or history of illicit drug use. She was a lifelong non-smoker with no exposure to second-hand smoke, as well as no history of exposure to asbestos or tuberculosis. Recent travel included visits to Iceland and Scotland. The patient had been employed in a managerial role at a potato chip factory for 18 months but spent time on the production floor without a mask. Her temperature was 37.1°C, with a heart rate of 98 beats/min, a blood pressure of 125/77 mmHg, a respiratory rate of 18 breaths/min and oxygen saturation of 98% on room air. Physical examination was unremarkable. Laboratory investigations included hemoglobin 119 g/L (normal 115 g/L to 155 g/L), leukocytes 5.3×10^9/L (normal 4.0×10^9/L to 11×10^9/L), platelets 197×10^9/L (normal 145×10^9/L to 400×10^9/L). Electrolyte and creatinine levels were within the normal range. Her lactate dehydrogenase level was 251 U/L (normal 110 U/L to 215 U/L) and erythrocyte sedimentation rate was 25 mm/h (normal 0 mm/h to 20 mm/h). Cultures were negative for fungi, tuberculous or any pathogenic bacteria. Bloodstream infection, antinuclear antibodies, extractable nuclear antigen and angiotensin-converting enzyme and aspergillus testing were all negative. Hepatitis C testing was not performed due to the absence of risk factors.

Initial chest radiographs showed an ill-defined density in the lingula (Figures 1 and 2). Computed tomography (CT) scan of the chest without contrast revealed ground-glass opacities in all lobes, with no predominance of lower lobe involvement. Linear bibasilar reticular fibrosis was also noted. There was no lymphadenopathy or suspicious nodules, and all other structures appeared to be normal (Figures 3 and 4).

The patient was referred to a respirologist and pulmonary function tests (PFTs) were performed. The results were consistent with a restrictive defect (Table 1).

To make a definitive diagnosis, wedge resections of both the left upper and lower lobes were obtained. The biopsies showed prominent accumulations of alveolar macrophages with a small number of eosinophils. The alveolar septa were mildly thickened by a chronic inflammatory cell infiltrate consisting of both lymphocytes and plasma cells, with the former predominating, and were lined by hyperplastic type II pneumocytes. There was no evidence of granulomatous inflammation, malignancy, fibroblastic foci or honeycombing. The pathological diagnosis was a DIP (Figures 5 and 6). Based on the clinical picture, the CT findings and the biopsy results, the patient was diagnosed with DIP. The patient was removed from the production floor environment, but did not clinically improve until she was started on prednisone 30 mg/day. At the time of submission, the patient was...
DIP in a healthy nonsmoker: A rare diagnosis

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being tapered off the prednisone; repeat PFTs have not yet been conducted.

DISCUSSION

DIP is a rare, serious, potentially fatal, inflammatory pulmonary disease first described in 1965 by Liebow et al (1). While DIP is most commonly found in tobacco smokers in their fourth and fifth decades of life, it is also associated with other entities including rheumatoid arthritis, use of sirolimus, nitrofurantoin, sulfasalazine and tocanide, cytomegalovirus, aspergillus exposure, hepatitis C, surfactant dysfunction in children, myeloid neoplasms, diesel fumes and marijuana smoking; it can also be idiopathic (1,2). DIP has been reported in individuals with occupations that expose them to inorganic dusts and fumes. These occupations are described by Benoît et al (3) and include tool grinding, textile manufacturing, arc polishing, tire manufacturing, arc welding and plastic maching.

Patients with DIP tend to present with insidious onset of nonspecific respiratory symptoms, including chronic cough that may be productive, chronic dyspnea and chest pain over the course of weeks to months (2,4). Constitutional symptoms, such as weight loss and fatigue, have also been described (4). Signs on physical examination may or may not be present, and can include bibasilar end-inspiratory crackles and digital clubbing (2,4).

Bloodwork findings vary in DIP, in some cases revealing eosinophilia, as well as elevated immunoglobulin (Ig) G and IgE levels, and increased erythrocyte sedimentation rate (5). PFTs in patients with DIP usually demonstrate either normal volumes or a restrictive pattern, with decreased forced vital capacity (FVC), decreased forced expiratory volume in 1 s (FEV1), a normal FEV1/FVC ratio and decreased diffusing capacity of the lung for carbon monoxide (4).

Findings on imaging in patients with DIP are variable and often inconclusive. Chest radiographs are insensitive in detecting DIP; they
The patient discussed was a known nonsmoker who was employed at a potato chip factory with no history of respiratory issues before her employment at this site. While not linked to potato chip manufacturing, there are cases of food production workers developing respiratory conditions, such as bronchiolitis obliterans, following exposure to diacetyl, a compound used in popcorn flavouring (7,8). A PubMed search using the keywords “desquamative interstitial pneumonia” or “desquamative interstitial pneumonitis” and “potato” revealed no reported cases of occupational DIP in this setting. The patient’s respiratory function did not fully improve after reducing her exposure to the work environment, but did improve following steroid therapy. The identity of a causal agent remains unknown and an industrial hygienist has not assessed the workplace at the time of submission. Investigations into the association between DIP and other workers in similar working environments may suggest a possible occupational etiology.

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**REFERENCES**


**TABLE 1 Selected pulmonary function tests**

<table>
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<tr>
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<th>Prebronchodilator</th>
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<tr>
<td></td>
<td>Actual</td>
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<tr>
<td>FVC, L</td>
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<td>FEV₁/FVC</td>
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<td>Total lung capacity, L</td>
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<tr>
<td>DLCO</td>
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</tr>
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</table>

DLCO: Diffusing capacity of the lung for carbon monoxide; FEV₁: Forced expiratory volume in 1 s; FVC: Forced vital capacity

**Post-test**

- What histological findings are typical of DIP?
  Typical findings in DIP include pigmented alveolar macrophages, thickened alveolar septa, a chronic inflammatory infiltrate, as well as hyperplastic type II pneumocytes lining the alveoli. Lymphoid follicles and giant cells are sometimes seen.
- What is the initial management for DIP?
  Systemic corticosteroid therapy and cessation of the irritant are the main components of initial treatment of DIP.
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