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Necrotizing Granulomas in a Construction Worker

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Introduction
Sarcoidosis is a granulomatous lung disease that predisposes individuals to pulmonary pathogens. Patients with sarcoidosis Stages II-IV, and those with fibrocystic changes, are at an increased risk for aspergillus infection. Chronic pulmonary aspergillosis (CPA) can complicate late-stage sarcoidosis, leading to necrotizing granulomas, hemoptysis, and potentially disseminated infection.

Case Report
A 37-year-old male with multiple exposures presented with dyspnea, fever, and cough productive of blood-streaked sputum for 5 months. Symptoms initially resolved with levofloxacin, but later recurred and then progressed. Imaging revealed bilateral middle and lower lobe cavitary lung lesions with surrounding fibrosis, atelectasis, and bronchiectasis.

Pulmonary function tests revealed a moderately-severe obstructive deficit.

<table>
<thead>
<tr>
<th>Spirometry</th>
<th>Pred</th>
<th>ULN</th>
<th>LLN</th>
<th>Actual</th>
<th>% Pred</th>
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<tbody>
<tr>
<td>FVC (L)</td>
<td>3.75</td>
<td>5.6</td>
<td>2.99</td>
<td>*1.16</td>
<td>*44</td>
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<tr>
<td>FEV1 (L)</td>
<td>82</td>
<td>92</td>
<td>72</td>
<td>*66</td>
<td>*81</td>
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<tr>
<td>FEV1/FVC (%)</td>
<td>4.57</td>
<td>5.38</td>
<td>3.76</td>
<td>*2.43</td>
<td>*53</td>
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<tr>
<td>FEV1/FVC (%)</td>
<td>82</td>
<td>90</td>
<td>74</td>
<td>*68</td>
<td>*83</td>
</tr>
<tr>
<td>FEF 25-75% (L/sec)</td>
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<td>5.74</td>
<td>2.24</td>
<td>*0.87</td>
<td>*22</td>
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<td>FEF Max (L/sec)</td>
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<td>11.39</td>
<td>7.27</td>
<td>*5.00</td>
<td>*53</td>
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<tr>
<td>Expiratory Time (sec)</td>
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<td></td>
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</tbody>
</table>

Figure 1. Bilateral pulmonary infiltrates and cavitary lesions on plain film.

Two sputum fungal cultures grew Aspergillus niger. Extensive serum testing including ANA, ANCA, B-D-glucan, galactomannan, ABF/quantiferon gold, cryptococcal antigen, and a fungal antibody panel were negative. EBUS transbronchial fine needle aspiration of node station 11L and transbronchial biopsy with bronchoalveolar lavage (BAL) of the left lower lobe was performed. Node station 11L revealed necrotizing granulomatous disease. Bronchial fungal cultures, aspergillus antigen and PJP were negative. Flow cytometry samples were unremarkable. The patient was treated with 4 months of voriconazole and a prolonged steroid taper. Repeat bronchoscopy revealed multiple necrotizing and non-necrotizing granulomas in the right upper lobe. All cultures were again negative. Surgical right lower lobe wedge resection demonstrated multiple non-necrotizing granulomas that coalesced primarily along the bronchovascular bundles with prominent fibrosis, suspicious for sarcoidosis. The patient was then started on steroids and trimethoprim-sulfamethoxazole, and then maintained on hydroxychloroquine.

Discussion
CPA is a serious complication of sarcoidosis that is associated with poor outcomes. Up to 10% of patients with sarcoidosis develop aspergillus infections. CPA should be suspected in all patients with known lung disease who have cavitary lung lesions for at least three months, with direct evidence of aspergillus infection. Over 50% of patients have no visible aspergilloma on imaging. If left unchecked, CPA can progress into chronic fibrosing pulmonary aspergillosis. The mechanism of injury is possibly due to alveolar macrophage-induced free radical damage. Laboratory testing often reveals elevated serum aspergillus IgG levels; however, the most definitive evidence is positive direct microscopy or fungal cultures from BAL. Patients often require lifelong antifungal therapy, and extensive steroids.

Conclusion
This case highlights the importance of early detection of aspergillus infections in patients with known pulmonary disease, particularly sarcoidosis. Early recognition and intervention are crucial to avoid progressive fibrosis.

REFERENCES
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