reports a 1-year and 3-year survival among patients with CWP that is similar to those with other indications for lung transplantation; six of the eight miners remained alive after a mean follow-up of 1,013 days after transplantation.  

Enfield et al  at the University of Virginia also recently reviewed outcomes in 30 patients at multiple centers who underwent lung transplantation for severe CWP since 1997, using the database of the United Network for Organ Sharing. In the University of Virginia analysis, after accounting for age, lung allocation score, and type of transplant, 1-year survival after transplantation appeared to be significantly lower in patients with CWP (61%) compared with non miner patients with COPD (82%) or interstitial lung disease (78%). However, independent of the exact prognosis of CWP after transplant, the reality of advanced CWP is that medical treatment may ameliorate symptoms but does not reverse the lung damage or halt the progressive fibrotic process.

Because of this, we agree that lung transplantation must be considered an option, particularly for the younger miners who are now developing this disorder. Dr Diaz-Guzman and colleagues encourage increasing awareness of advanced CWP and early referral to a transplant center, in recognition that massive fibrosis in coal miners often progresses even after removal from dust exposure. We concur that the medical community needs to be more effective in enhancing awareness of the continuing human toll from these dust diseases and in assuring optimal medical care and fair compensation for affected miners. Progressive massive fibrosis is entirely preventable, since it is virtually only caused by excessive dust inhalation and does not occur from tobacco use or other causes. Effective dust controls should have eliminated this type of lung disease in a modern mining industry, and the failure of the US industry to tackle this ongoing problem has been highlighted internationally.

In addition to drawing attention to the role of lung transplantation to improve survival and functional status in these patients, we hope our report’s findings can motivate timely implementation of the necessary effective measures to reduce dust exposures and provide a healthful working environment for our country’s coal miners.

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REFERENCES


Serum Procalcitonin and Infective Exacerbations of Asthma

To the Editor:

Bafadhel and colleagues report in a recent issue of CHEST (June 2011) that serum procalcitonin levels are high in patients admitted to hospital with pneumonia but not in those admitted with exacerbations of asthma or COPD. We examined the usefulness of serum procalcitonin in patients with moderate to severe exacerbations of asthma due to infections.

We recruited 25 patients (11 men) with confirmed diagnosis of asthma during what was considered an infective exacerbation (increased symptoms as measured by a seven-point Likert scale, increased sputum volume and purulence) that was not severe enough to require hospitalization. None of the patients had radiologic evidence of pneumonia. Spirometry was performed and nasopharyngeal swabs and sputum were obtained for virology; bacterial culture, and quantitative cell counts. Measurements were repeated at 1, 4, and 6 weeks until symptoms had completely resolved. Procalcitonin was measured in serum at the time of exacerbation and at 6 weeks. Procalcitonin was measured in duplicate from 50 μL of serum using a time-resolved amplified cryptate emission technology assay (Kryptor TRACE PCT; Brahms; Berlin, Germany). The lower limit of detection is 0.02 ng/mL, and the assay functional sensitivity was 0.06 ng/mL.

<table>
<thead>
<tr>
<th>Measurement</th>
<th>At Exacerbation</th>
<th>At Resolution (6 wk)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Symptoms*</td>
<td>3.5 (1.2)</td>
<td>6.0 (7.0)</td>
<td>&lt;.05</td>
</tr>
<tr>
<td>FEV₁, %</td>
<td>60.6 (25.6)</td>
<td>67.0 (23.0)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Sputum TCC, 106/g</td>
<td>40 (9.7)</td>
<td>6.0 (1.5)</td>
<td>&lt;.05</td>
</tr>
<tr>
<td>Sputum N, %</td>
<td>72.7 (9.3-98.7)</td>
<td>53.7 (8-93)</td>
<td>&lt;.05</td>
</tr>
<tr>
<td>Sputum E, %</td>
<td>3.7 (0.7-10)</td>
<td>0.8 (5-10)</td>
<td>&lt;.5</td>
</tr>
<tr>
<td>Serum procalcitonin, ng/mL</td>
<td>0.08 (0.03)</td>
<td>0.08 (0.02)</td>
<td>.9</td>
</tr>
</tbody>
</table>

Values are given as mean (SD) unless otherwise noted. E = eosinophil; N = neutrophil; TCC = total cell count.

*Symptoms of cough, chest tightness, wheeze, and shortness of breath were measured on a seven-point Likert score (7 best, 1 worst).

*Median (interquartile range).

*Median (minimum-maximum).

Table 1—Measurements During an Exacerbation and 6 Weeks Later
All patients gave written informed consent, and the study was approved by the Research Ethics Board of St. Joseph’s Healthcare Hamilton.

Test results in 15 subjects (60%) had positive identification of a pathogen; five (20%) were viral (one influenza B, one respiratory syncytial virus, one human metapneumovirus, and one parainfluenza 4 and rhinovirus, human coronavirus NL63), two (8%) were bacterial (Streptococcus pneumoniae and Haemophilus parainfluenzae), and four (16%) were both bacterial and viral (Moraxella and rhinovirus, Streptococcus pneumoniae and human coronavirus NL63, Streptococcus pneumoniae and parainfluenza B, and Staphylococcus aureus and parainfluenza 3).

Symptoms and FEV1 improved significantly, and the sputum cell counts returned to normal at 6 weeks (Table 1). However, there was no significant difference in procalcitonin levels between the initial measurement and at 6 weeks (Table 1). There were also no differences between patients who had infective vs noninfective exacerbations or those with viral vs bacterial bronchitis.

Our data confirm the observations of Bafadhel and colleagues1 that serum procalcitonin is unlikely to be useful to identify infective exacerbations of asthma. Erupted sputum total cell count with predominant neutrophilia is a more reliable indicator of an infective bronchitis.

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Response
To the Editor:
We thank Dr D’silva and colleagues for their interest in our recent article in CHEST.1 Most asthma exacerbations are considered to be associated with viruses,2 and current guidelines do not advocate the use of antibiotics for exacerbations of asthma.3 Interestingly, Dr D’silva and colleagues only identified viruses in 36% of cases and bacteria in 24%. In COPD, where bacterial infection is implicated as a major cause of exacerbations, current evidence does not support the use of antibiotics in the management of mild to moderate exacerbations.4 Whether patients with asthma and bacterial-associated exacerbations benefit from antibiotic therapy is uncertain; however, further detailed analysis of the microbiology of asthma exacerbations using both standard and molecular techniques is warranted. We and others have found that procalcitonin is not strongly associated with an exacerbation of COPD or asthma,5 but is elevated in patients with pneumonia. It is, therefore, a good biomarker of a systemic inflammatory response to pneumonia and may have potential clinical utility in directing antibiotic therapy.6 Importantly, the value of procalcitonin might not be greater than the more widely available C-reactive protein. Controlled trials of antibiotics directed by biomarkers such as C-reactive protein or sputum cell counts at exacerbations of asthma and COPD are required. Biomarker-guided therapy is commonplace in other medical specialties, such as cardiology. This approach needs urgent investigation and to be embraced by respiratory medicine if we are to make a change in the management of exacerbations of airways disease.

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