Computed tomography of the thorax in workers exposed to hard metals

David S Mendelson, Ellen S Gendal, Cynthia L Janus, Alf Fischbein

Hard metal disease is an occupational lung disorder occurring primarily among workers employed in the cemented tungsten carbide industry.1-6 Interstitial pulmonary fibrosis, acute obstructive pulmonary disease (asthma), and dermatitis are the main clinical manifestations of such exposure. The workers in this industry are at risk from exposure to powders of a variety of hard metals during the manufacture of tools. These metals include tungsten, titanium, tantalum, vanadium, niobium, and cobalt.1-3 They provide the hardness, strength, and heat resistance required in tools. Many are inert and harmless but cobalt is believed to be a causative agent of pulmonary disease.

We performed thoracic computed tomography (CT) scans on 11 hard metal workers and describe the findings here.

Materials and methods

Eleven male patients from a single hard metal manufacturing facility were referred for evaluation because of exposure to hard metal dust. Chest CTs were performed on a General Electric CT 8800, without contrast, at 10 mm thick consecutive intervals. The CTs were graded by two independent reviewers regarding parenchymal, pleural, and nodal changes.

Results

The table displays the clinical, chest, and CT data. The chest x-ray films were graded by International Labour Office (ILO) criteria. Duration of exposure varied from three to 17 years. Three patients were identified by chest x-ray film as having appreciable interstitial changes (1/1-1/2). The same three patients were also judged to have pronounced (2 to 3+) interstitial changes on chest CT (fig 1). All other patients had negative or minor parenchymal changes on both x-ray film and CT.

Eight patients had increased attenuation lymph nodes, often at multiple sites (fig 2). One further patient had enlarged soft tissue attenuation subcarinal nodes. Calcified or metallic nodes were not found on plain film, even in retrospect. In our patient

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age (y)</th>
<th>Employment duration (y)</th>
<th>Smoking history (y)</th>
<th>ILO x-ray film classification</th>
<th>PPDM</th>
<th>Nodes</th>
<th>CT parenchyma (interstitial changes)</th>
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<tbody>
<tr>
<td>1</td>
<td>39</td>
<td>17</td>
<td>N</td>
<td>1/1</td>
<td>(-)</td>
<td>Ca** hila, paratracheal</td>
<td>(+ + +)</td>
</tr>
<tr>
<td>2</td>
<td>30</td>
<td>9</td>
<td>12</td>
<td>0/1</td>
<td>(-)</td>
<td>(-) Subcarinal but no cobalt</td>
<td>(+)</td>
</tr>
<tr>
<td>3</td>
<td>30</td>
<td>5</td>
<td>15</td>
<td>0/1</td>
<td>(-)</td>
<td>(+) Ca** paratracheal, hila, subcarinal</td>
<td>(+)</td>
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<tr>
<td>4</td>
<td>37</td>
<td>15</td>
<td>24</td>
<td>0/0</td>
<td>(-)</td>
<td>(+) Ca** paratracheal, hila, subcarinal</td>
<td>(+)</td>
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<tr>
<td>5</td>
<td>51</td>
<td>17</td>
<td>Ex</td>
<td>0/1</td>
<td>(-)</td>
<td>(+) Ca** hila, retrocrural</td>
<td>(-)</td>
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<tr>
<td>6</td>
<td>41</td>
<td>3</td>
<td>N</td>
<td>0/0</td>
<td>(-)</td>
<td>(-) Subcarinal but no cobalt</td>
<td>(-)</td>
</tr>
<tr>
<td>7</td>
<td>23</td>
<td>3</td>
<td>N</td>
<td>0/0</td>
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<td>(+) Ca** right paratracheal</td>
<td>(+)</td>
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<tr>
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<td>32</td>
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<tr>
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<td>38</td>
<td>14</td>
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<td>(+ +)</td>
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<tr>
<td>11</td>
<td>44</td>
<td>5</td>
<td>N</td>
<td>1/2</td>
<td>(+)</td>
<td>(+) Ca** adenopathy</td>
<td>(+ +)</td>
</tr>
</tbody>
</table>

Smoking history; N = non-smoker, Ex = stopped smoking before this study.
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Figure 1  A and B; representative axial sections photographed at lung windows showing interstitial changes. These were regarded as severe and correlated with chest x ray film changes. High attenuation nodes at the hila can also be seen.

Figure 2  A and B; these patients have high attenuation lymph nodes at the hila and subcranial levels. C and D; a third patient, also shown in fig 1, with high attenuation hila nodes. This diffuse distribution was fairly typical.
population, duration of exposure did not clearly correlate with either parenchymal or nodal abnormality. Two patients with minimal emphysematous changes also had positive smoking histories.

Discussion

Hard metal disease is a well described entity, with pulmonary disease a major manifestation. Cobalt is believed to be the common noxious agent in patients exposed to a variety of hard metals.

We examined a group of hard metal workers with chest CT to establish how CT correlated with other examinations and to ascertain whether any new information could be obtained. Our results suggest that CT and plain film show parenchymal changes equally. The examinations were performed before high resolution lung techniques were introduced at our institution. It is conceivable that we might have had a greater yield of detected minimal interstitial changes had these techniques been employed.

Results of CT showed mild emphysematous changes in two patients who had normal chest x ray films. Imaging by CT has been shown to identify such changes, perhaps more reliably than chest x ray films. The relation of these changes to the workers' dust exposure is uncertain. This exposure can, however, result in bronchitis and asthmatic syndromes and we may be seeing the morphological effect of these manifestations of hard metal disease.

Increased density of mediastinal nodes at numerous sites was shown by CT, particularly in the hila and subcarinal regions. This was unexpected as these densities were unappreciated on chest x ray film. Increased concentrations of cobalt and other metals have been found, however, in the lymph nodes of hard metal workers and also in one of our patients. Lundgren and Öman reported enlarged hila lymph nodes in hard metal disease. Spectrographic analysis showed high concentrations of tungsten and titanium, but not cobalt. Whether we are detecting depositions of the metals in lymph nodes, or calcium deposited dystrophically cannot be determined by CT. It should be noted that only one patient with dense lymph nodes had a positive purified protein derivative test (PPD). We consider this diffuse pattern of calcification unusual in tuberculosis. These patients were not skin tested for exposure to fungal agents.

We believe that the increased density lymph nodes are pertinent as they may represent a means of identifying patients exposed to heavy metal dusts. Although some of the patients with dense lymph nodes showed other manifestations of hard metal disease, correlation with severity of disease cannot be determined in such a small group. The presence of dense lymph nodes, however, may offer a means of identifying which patients have had significant exposure to dust. Also, CT can be used simultaneously to evaluate the lung parenchyma. Early interstitial disease as well as emphysematous changes may be detected.

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