CASE REPORT

Occupational asthma caused by heated triglycidyl isocyanurate

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Abstract

Six workers exposed as bystanders to heated triglycidyl isocyanurate (TGIC) developed occupational asthma confirmed by serial peak expiratory flow measurement and Oasys analysis. Specific inhalation challenge testing resulted in late or dual asthmatic reactions to heated TGIC in four of four tested and was negative in three control asthmatics. One worker tested only with unheated TGIC had a negative specific challenge test. Heated TGIC can cause occupational asthma from bystander exposure.

Key words

Oasys; occupational asthma; peak expiratory flow; specific inhalation challenge; triglycidyl isocyanurate.

Introduction

Triglycidyl isocyanurate (TGIC) is a hardening agent used in powder paints. TGIC has been reported as causing allergic eczema [1,2] and occupational asthma in powder paint sprayers [3,4]. There were 11 reports to SWORD (Surveillance of Work-related and Occupational Respiratory Disease) between 1989 and 2010 attributed to TGIC (10 cases of occupational asthma and 1 inhalation accident, including the six workers reported here) and a further two reports of TGIC asthma from occupational physicians reported via Occupational Physicians Reporting Activity [5]. We report five cases of occupational asthma due to indirect exposure to heated TGIC from the same plant and a sixth case who worked elsewhere.

The factory employing workers 1–5 made domestic gas fires that were assembled and tested in an open plan area. Gas fire appliances had a powder coat containing 10% TGIC applied to them electrostatically to provide a protective and decorative finish. The process involved pretreatment of metal components with a phosphate wash. Items then passed through an extracted powder coat booth where they were sprayed by hand and then went into ovens for curing at 200°C for 18 min. There was no extraction after the items left the ovens and were distributed on conveyors to the assembly lines where the subjects worked. Isocyanates were not used within the factory and no other known sensitisers could be identified. The factory employed ~350 workers and the cases were referred to our clinic by their occupational physician. Subject 6 worked in a factory where architectural metal products were spray painted with powder coatings containing TGIC. The curing ovens ran the length of the building and were not extracted. He was indirectly exposed as the ovens were situated close to where he worked in the metal treatment area.

Case reports

Demographic information, serial peak expiratory flow (PEF) outcomes and specific inhalation challenge results are shown in Table 1. Challenges were carried out in a single-blind fashion so that the smallest dose and duration was given on the second day after a suitable control exposure on day 1. If no immediate or late reaction occurred, exposures were increased on subsequent days until an exposure likely to represent a day’s work exposure was achieved. Control challenges to 3 g of either calcium carbonate or lactose heated to 200°C in a boiling tube for 15 min or tipped powder coat (without TGIC) were negative in all cases. Following TGIC exposure, three subjects had dual asthmatic reactions and one had an isolated late reaction (Figure 1). Most of the subjects exposed to heated TGIC also complained of upper airway soreness and headache similar to that reported at work. Worker 6 showed no reaction to unheated powder coat containing 5% TGIC nor to the chrome pre-treatment chemicals he was also exposed to. Three workers with positive challenges to colophony, glutaraldehyde or photocopier toner had exposures to heated TGIC 0.1–15 g heated for 5–20 min as their control exposure. All had increased non-specific bronchial reactivity and no reaction to the TGIC.
Table 1. Patient demographics, serial PEF results and specific inhalation challenge outcomes

<table>
<thead>
<tr>
<th></th>
<th>Case 1</th>
<th>Case 2</th>
<th>Case 3</th>
<th>Case 4</th>
<th>Case 5</th>
<th>Case 6</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, sex</td>
<td>36, F</td>
<td>55, F</td>
<td>43, F</td>
<td>58, F</td>
<td>48, M</td>
<td>41, M</td>
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<tr>
<td>Latent interval (years)</td>
<td>12</td>
<td>12</td>
<td>13</td>
<td>17</td>
<td>11</td>
<td>6</td>
</tr>
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<td>History of smoking</td>
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<td>Yes</td>
<td>Yes</td>
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<td>Atopy</td>
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<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
<td>No</td>
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<tr>
<td>FEV1 % predicted</td>
<td>121</td>
<td>76</td>
<td>136</td>
<td>69</td>
<td>41</td>
<td>90</td>
</tr>
<tr>
<td>FEV1/FVC (%)</td>
<td>85</td>
<td>74</td>
<td>76</td>
<td>68</td>
<td>55</td>
<td>84</td>
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<tr>
<td>Histamine PD20 (µmol)</td>
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<td>&gt;8</td>
<td>0.8</td>
<td>1.1</td>
<td>75 mcg</td>
<td>&gt;8</td>
</tr>
<tr>
<td>Oasys score</td>
<td>2.89</td>
<td>2.56</td>
<td>3.5</td>
<td>3.0</td>
<td>3.14</td>
<td>3.95</td>
</tr>
</tbody>
</table>

Specific inhalation challenge test results

| Amount of TGIC heated (g) | 0.3 | 3   | 3   | –   | 2   | –   |
| Total duration of challenge (min) | 7   | 11  | 16  | –   | 60  | 15  |
| Max early reaction to TGIC (%) | 25  | 16  | 24  | –   | 0   | None|
| Max late reaction (%)       | 30  | 16  | 24  | –   | 23  | None|

*Methacholine PD20 (normal > 2000 mcg). Latent interval is between first exposure and first symptoms. Oasys score ≥2.51 is positive for occupational asthma. F, female; M, male; FEV1, forced expiratory volume in 1 s; FVC, forced vital capacity.

Figure 1. (a–e) Specific bronchial challenge test for (a) case 1 (b) case 2 (c) case 3 (d) case 5 (shown as % fall from predicted due to differences in baseline between control and active days) and (e) case 6. The grey area is the exposure to either heated TGIC, tipped TGIC or the control.
Workers were requested to complete 2 h PEF measurements from waking to sleeping for a total of 4 weeks. The PEF records of all six workers were suggestive of occupational asthma (Table 1) when plotted in the Oasys programme [6]. The records showed a consistently lower PEF on days at work but with no discernible significant early or late reactions. Specific immunoglobulin E (IgE) to TGIC was measured in cases 3 and 4 and was negative. Case 5 had a sputum eosinophilia (13.6%) and a raised NO of 18 ppb (at a flow rate of 250 ml/s; upper limit for normal individuals in our laboratory is 9.6 ppb).

All the workers have been removed from exposure (relocated, retired on ill-health or made redundant). Subjects 1 and 5 improved slightly but still had significant symptoms, waking with nocturnal asthma on most nights. Symptoms were much improved in the others.

Gaseous products from a sample of heated TGIC were analysed by mass spectrometry and TGIC was the only compound present when heated to 200°C. When the mol file (a file format that holds information about the atoms, bonds, connectivity and coordinates of a molecule) for TGIC was entered into the asthma hazard prediction programme, it had an asthma hazard index of 0.99 (undoubtedly hazardous). [7]. This programme was set up to identify asthmagens of a low molecular weight nature, giving a predictive score from 0 (unlikely to be hazardous) to 1 (undoubtedly hazardous). We conclude that TGIC released during the curing of powder coat finished products can cause occupational asthma.

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**Conflicts of interest**

None declared.

**References**


