IRREVERSIBLE LUNG DAMAGE IN EXTRANISIC ALLERGIC ALVEOLITIS FROM PIGEON DUST INHALATION*

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Irreversible lung damage can occur in hypersensitive subjects after prolonged or repeated exposure to organic dusts. This is known to occur in farmer's lung,\(^1\) maple bark disease,\(^2\) bagassosis,\(^3\) byssinosis,\(^4\) and silo-filler's disease,\(^5\) but to date there is only one report of its occurrence in bird fanciers.\(^6\) The following case report records the finding of irreversible lung damage and cor pulmonale in a hypersensitive subject exposed to pigeon dust for many years.

CASE REPORT

A 63-year-old White female was referred to the Cardiac Clinic, Groote Schuur Hospital, for investigation of her cough and shortness of breath. The patient dated the onset of her symptoms to an attack of 'bronchitis' in 1963, 5 years previously; following that episode, her cough persisted with the production of yellow sputum in the mornings only with no haemoptysis. She had observed dyspnoea since 1963, and at the time of observation it had become so severe that she could only walk about 30 yards slowly before stopping for a rest. She noticed wheezing occasionally and had mild postnasal catarrh; at no time had she experienced nocturnal dyspnoea, palpitation or significant ankle oedema.

She had lost 60 lb in weight over this period. Her course had been interrupted by two other episodes of fever with increased cough and sputum, and partially responded to antibiotic therapy. Chest radiographs had been taken at intervals at the West End Hospital, Kimberley (Figs. 1 - 3); sputum cultures were negative for fungi and \textit{M. tuberculosis} in July 1966 and February 1967.

The patient had never smoked cigarettes. Her husband had been an enthusiastic pigeon-fancier for over 18 years and she had often helped him by feeding the birds, sitting in the loft for long hours during the process. Some 3 years before this visit, her friends had discouraged her from doing this, as they had noticed that her symptoms were worse after visiting the loft. She had kept away from the birds thereafter. She had had no other major illnesses or operations. There was no history of lung disease or allergies in her family. Her husband was fit and well.

On physical examination, the patient was an alert, moderately cyanosed, emaciated woman (weight 100 lb (46 kg), height 5 ft 5 in (165 cm) who became distressed on the least physical effort. She had finger clubbing 1+. There was no dependent oedema, no lymph nodes were palpable, and skin and joints were normal. Her blood

\*Date received: 26 November 1969.

\[\begin{align*}
\text{Fig. 1. Chest radiograph taken on 27 July 1964. Slight increase in bronchovascular markings in both lung fields.} \\
\text{Fig. 2. A further increase in lung markings could be seen on 19 July 1966.}
\end{align*}\]
pressure was 110/90 mmHg, pulse 70/min and regular. The jugular venous pressure was normal. A left parasternal heave was present, the apex beat being in the 5th intercostal space in the mid-clavicular line. The second heart sound in the pulmonary area was loud, and a grade 2 systolic murmur was heard over the precordium, maximal in the 4th left intercostal space, probably tricuspid in origin. Respiratory frequency was 25/min, the accessory muscles were in use, and the trachea was central. Chest expansion was poor but the shape normal. Hyperresonance to percussion was present in the upper zones but resonance was impaired at the bases. Loud bronchovesicular breath sounds could be heard all over, with extensive fine crepitations at both lung bases. There were no wheezes. The liver was felt 2 cm below the costal margin. The rest of the examination revealed nothing abnormal.

Investigations

The electrocardiogram showed a rate of 80/min, sinus rhythm, PR 0·15 seconds, right atrial hypertrophy, right bundle-branch block. The chest radiograph showed extensive reticulation with honeycomb translucencies in both lung fields, the left side being more translucent than the right. The cardiac shadow was increased in size, chiefly to the left (Figs. 4 - 6). Serum proteins, total 8·39 g/100 ml. Electrophoretic pattern normal, albumin 4·39 g/100 ml, alpha,-globulin 0·31 g/100 ml, alpha,-globulin 0·68 g/100 ml, beta-globulin 0·98 g/100 ml, gammaglobulin 2·03 g/100 ml. Lung-function studies showed a severe combined restrictive and obstructive defect, with marked ventilation/perfusion inequalities resulting in hypoxia and mild uncompensated respiratory acidosis (Tables I and II).
TABLE I. LUNG-FUNCTION DATA 13/2/68 after steroid treatment

<table>
<thead>
<tr>
<th>Studies</th>
<th>6/2/68</th>
<th>Predicted normal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Inspiratory capacity (ml)</td>
<td>625</td>
<td>650</td>
</tr>
<tr>
<td>Expiratory residual volume (ml)</td>
<td>430</td>
<td>500</td>
</tr>
<tr>
<td>Vital capacity (sum)</td>
<td>1055</td>
<td>1150</td>
</tr>
<tr>
<td>Continuous breath vital capacity</td>
<td>950</td>
<td>990</td>
</tr>
<tr>
<td>Functional residual capacity (ml)</td>
<td>1590</td>
<td>2220</td>
</tr>
<tr>
<td>Residual volume (ml)</td>
<td>1160</td>
<td>1820</td>
</tr>
<tr>
<td>Total lung capacity (ml)</td>
<td>2215</td>
<td>2870</td>
</tr>
<tr>
<td>RV/TLC %</td>
<td>52%</td>
<td>63%</td>
</tr>
<tr>
<td>Forced expiratory volume (ml) (1st sec)</td>
<td>860</td>
<td>950</td>
</tr>
<tr>
<td>Maximal voluntary ventilation (litre/min)</td>
<td>39</td>
<td>45</td>
</tr>
<tr>
<td>Respiratory frequency</td>
<td>57</td>
<td>35</td>
</tr>
<tr>
<td>Tidal volume (ml)</td>
<td>470</td>
<td>480</td>
</tr>
<tr>
<td>Minute ventilation (litre/min)</td>
<td>174</td>
<td>167</td>
</tr>
</tbody>
</table>

The laboratory report on serum precipitins was as follows: 'Serum precipitins were titrated against several suspensions of pigeon droppings collected from a number of different pigeons including material from the patient's own home. A maximum of 4 lines of precipitins was obtained and the end-points against the different antigens were at dilutions of serum up to 1/32. There were precipitins against pigeon egg-white and pigeon serum with end-points at 1/8 in both cases (Fig. 7). No precipitins were detected in the patient's serum when tested against the droppings, egg-white and serum of the domestic chicken. No precipitins against any of the pigeon or chicken antigens were detected in the serum of the patient's husband.'

DISCUSSION

Most subjects identified thus far as having hypersensitivity responses to pigeon dusts have presented with the features of an active interstitial pneumonitis. The onset has been either acute with fever and chills, or insidious, with cough, dyspnoea and weight loss. Physical signs have been confined to tachypnoea and a few crepitations. Radiological changes have been minimal, namely fine, diffuse nodular densities in the lung fields. The lung-function studies have indicated a restrictive defect, with gas transfer difficulty, and the histological changes have been those of an interstitial invasion with lymphocytes, plasma cells and eosinophils, with knots of large foamy mononuclear cells. In all patients, strong serum precipitin reactions to pigeon products could be shown. Such an acute process has been shown to be reversible if the subject is removed from contact with the birds or is treated with corticosteroids. In our experience, it is possible for the subject to resume keeping birds without an immediate return of symptoms, but a rising titre of serum precipitins after a period of re-exposure suggests that subclinical hypersensitivity responses are occurring.

Fig. 7(a). Pigeon droppings 1/32 dilution in central well. 7(b). Pigeon egg white 1/2 dilution in central well. 7(c). Pigeon serum undiluted in central well.
In a recent paper on pulmonary function in pigeon breeders' disease, Schluter et al. recorded the lung-function findings in 13 subjects. They defined acute, subacute and chronic disease patterns, recognizing that there were considerable variations in individual responses, which could be explained by differences in the intensity and chronicity of exposure to pigeon products, and individual susceptibility.

The two subjects classified as having the chronic form had the physiological features of chronic airways obstruction with hyperinflation of the lungs, a raised residual volume and reduced diffusing capacity. The findings were similar to those found in emphysema of the lungs. The histological changes in the one subject were different from the lung parenchymal changes seen in the acute form, in that there were no foam-cell aggregates in the alveoli, but a few in the lumina of the small bronchioles. There was a mild interstitial pneumonitis with sarcoid-like granulomatous chronic organizing bronchiolitis and focal fibrosis of a few alveolar septa were noted. Because of the deflated nature of the specimen, emphysema could not be demonstrated. Steroid therapy having been given, it is likely that some of the histological changes were modified by this.

In our subject, the presenting clinical and radiological features were more like those found in extensive diffuse fibrosis alveolitis which produces considerable dyspnoea and extensive honeycomb changes in the lungs. Her lungs were reduced in total size; she had a disproportionately large residual volume and reduced maximal voluntary ventilation in addition to severe ventilation/perfusion inequalities. A hypersensitivity reaction was not suspected in 1963, although, interestingly enough, the subject herself was eventually persuaded to keep away from the birds. Despite this, her disability increased. Steroid therapy, given late in her course, produced a very slight symptomatic response, but there were no significant changes in the physical signs or lung-function studies. Her cardiorespiratory failure increased over the subsequent months and she died on 3 October 1968. An autopsy was not performed. Her course is strikingly different from the dramatic response to steroids by subjects who present in the acute phase, where symptomatic relief is rapid, but full restoration of lung function may take many months, and also from the previously reported chronic disease form in which emphysema occurred. There is mounting evidence that hypersensitivity reactions to pigeon products induce significant physiological and pathological changes in the lungs, and that, in some subjects, irreversible damage can occur, which may be related to the intensity and chronicity of exposure to pigeons or the reactive responses of the subject.

**SUMMARY**

The case report records the findings of diffuse pulmonary fibrosis and cor pulmonale in a hypersensitive subject exposed to pigeon dust, in whom serum precipitins could be found in significant titres. It seems likely that this represents the end-stage of hypersensitivity to the inhalation of pigeon dust, with a natural history similar to the other pulmonary fibroses that occur in some hypersensitive subjects exposed to organic dusts.

I wish to thank Prof. V. Schrire and Dr. H. Samy-Padiachi who referred the patient for study; the Department of Bacteriology, for serological studies; and Dr. C. A. Sleggs who supplied the early chest radiographs. I should also like to thank Dr. J. G. Burger, Medical Superintendent of Groote Schuur Hospital, for permission to publish, and the South African Council for Scientific and Industrial Research Cardiovascular-Pulmonary Research Group and the City Council of Cape Town for their financial assistance.

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