The lungs in rheumatoid arthritis — a clinical, radiographic and pulmonary function study

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Objective. To determine the prevalence and spectrum of pulmonary abnormalities in patients with rheumatoid arthritis (RA) in a developing country.

Design. This was a prospective hospital-based survey of a randomly selected group of patients with RA who were seen in a rheumatic diseases unit.

Setting. Groote Schuur Hospital and Princess Alice Orthopaedic Hospital, Cape Town.

Patients. A group of 104 patients with RA were randomly selected from a total of 330 patients with RA who were seen in the rheumatic diseases unit. All the patients were interviewed and a clinical assessment, chest radiographs and pulmonary function tests were performed.

Results. Fifty-six patients (53.8%) had evidence of one or more current or previous pulmonary diseases: rheumatoid nodules in 1 (1%), bronchiectasis in 2 (1.9%), fibrosing alveolitis in 5 (4.8%), pneumonia in 5 (4.8%), asthma in 9 (8.7%), pleural disease in 17 (16.3%) and tuberculosis in 25 (24%). Excluding patients who were smokers or ex-smokers or who had coexistent pulmonary disease, there were 20 patients (19.2%) who had pulmonary abnormalities that could be attributed to RA: rheumatoid nodules in 1 (1%), fibrosing alveolitis in 5 (4.8%) (1 of whom also had pleural disease), pleural disease alone in 8 (7.7%), diffusion defect in 5 (4.8%) and airways obstruction in 1 (1%).

Conclusion. This study provides clinical and lung function criteria that allow a clinically useful stratification of abnormalities in relation to a spectrum of common causes of pulmonary dysfunction that need to be distinguished from pulmonary abnormalities caused by RA. Pulmonary abnormalities are common and about 20% of RA patients may have an abnormality related to RA.


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Pulmonary complications of rheumatoid arthritis (RA) were first reported in 1948. These complications, also subsequently described by others, include interstitial lung disease,1 pleurisy,2 necrobiotic nodules,3 nodules associated with silico-anthracosis (Caplan's syndrome),4 obliterative bronchiolitis,5 pulmonary vascular disease6 and bronchocentric granulomatous lung disease.7 An increased incidence of pulmonary infection is also found in patients with RA.8

Some of these manifestations of rheumatoid disease are quite uncommon, and accurate assessment of their prevalence is made difficult by differing methods of case selection in different published series. Moreover, confounding variables such as smoking habit, occupation and patients excluded because of concurrent disease present further difficulties in estimating prevalence.

The aim of this study was to determine the prevalence and spectrum of pulmonary abnormalities in 104 patients with RA selected randomly from a rheumatic diseases unit.

Methods

Of 330 patients of mixed race with classic or definite RA9 who were seen at the rheumatic diseases unit of the University of Cape Town, 104 were selected by means of random number tables for inclusion in this study.

A detailed cardiorespiratory history included occupational and environmental exposure to dust and foreign proteins, smoking status, present and previous drug administration, and current symptoms, including breathlessness, cough, sputum production and chest pain. The American Rheumatism Association (ARA) functional classification was applied and recorded10 and a detailed physical examination performed.

A full blood count (Coulter-S-plus automated counter) and latex test for rheumatoid factor (slide agglutination technique, Orthodiagnostics) were performed on all patients. A titre of 80 or greater at the time of the study was considered an indication of seropositivity. Standard postero-anterior and lateral chest radiographs were taken, and the films were read independently by a respiratory physician and a radiologist, initially without reference to the clinical details. Inter-reader differences of opinion were few, and always minor. These were resolved by reference to a second respiratory physician, who had access to the patient's clinical details.

Spirometry was assessed by means of the flow volume loop, performed with a wedge spirometer and XY recorder (med-Science 570). The best of three satisfactorily performed manoeuvres was used and response to bronchodilator was assessed. All 104 patients were able to perform spirometry satisfactorily. Functional residual capacity (FRC) was measured by the helium dilution method (Expirograph, Godart). Total lung capacity (TLC) and residual volume (RV) were calculated from this test, which was performed once only; 5 patients were unable to co-operate fully. Transfer factor for carbon monoxide (TLCO) and transfer coefficient (KCO) were obtained by the single-breath method (Carbon Monoxide Transfer test, PK Morgan). The mean of two adequately performed measurements, 5
minutes apart and corrected for haemoglobin level, was used. Three patients were unable to sustain the required 10-second breath hold, but satisfactory results were obtained in 101 patients. Maximum inspiratory (MIP) and expiratory mouth pressures (MEP) were performed at RV and TLC respectively, according to the method of Black and Hyatt, in which the patient was required to sustain maximal effort against the pressure gauge for at least 1 second; the best of three such measurements was taken. All measurements were expressed as percentages of predicted values based on each subject's age, sex, height and weight.

Pulmonary function tests were interpreted by a respiratory physician without reference to the clinical or radiographic data. The presence and severity of airflow obstruction, restrictive abnormality, or both together was determined by examining the flow volume loop and full volume data. In the case of TLCO, KCO, MIP and MEP, the severity of abnormality was assessed individually for each test. The criteria used to interpret the pulmonary function tests are given in Table I.

Table I. Criteria for interpretation of pulmonary function tests

<table>
<thead>
<tr>
<th>Abnormality</th>
<th>General criteria</th>
<th>Grade</th>
<th>Specific grading criteria</th>
</tr>
</thead>
<tbody>
<tr>
<td>Airway obstruction</td>
<td>Appearance of flow-volume loop, plus specific criteria</td>
<td>Mild</td>
<td>Deviation of more than 2 standard deviations below or above predicted values for any of the following: FEV1/FVC ratio, PEFR, MEF50, FEV1, with normal FVC, RV, FRC, TLC; RV/TLC ratio, with normal TLC</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Moderate</td>
<td>FEV1/FVC ratio 50 - 65%, or MEF50 &lt; 40% predicted</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Severe</td>
<td>FEV1/FVC ratio &lt; 50%</td>
</tr>
<tr>
<td>Response to bronchodilator</td>
<td>–</td>
<td>Nil</td>
<td>No increase in FEV1, or FVC</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Slight</td>
<td>0 - 10% increase in FEV1, or FVC</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Marked</td>
<td>&gt; 10% increase in FEV1, or FVC</td>
</tr>
<tr>
<td>Restrictive pattern</td>
<td>Appearance of flow-volume loop, plus specific grading criteria</td>
<td>Mild</td>
<td>Deviation of more than 2 standard deviations below or above predicted values for any of the following: FVC, FRC, TLC</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Moderate</td>
<td>FVC or TLC 40 - 60% predicted</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Severe</td>
<td>FVC or TLC &lt; 40% predicted</td>
</tr>
<tr>
<td>TLCO, KCO, MIP, MEP</td>
<td>–</td>
<td>Minimal</td>
<td>60 - 80% of predicted value but within 2 standard deviations</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Mild</td>
<td>Deviation of more than 2 standard deviations below or above predicted value, but &gt; 50% predicted value</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Moderate</td>
<td>40 - 59% predicted value</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Severe</td>
<td>&lt; 40% predicted value</td>
</tr>
</tbody>
</table>

* No obstruction must be present if reduced FVC is the only criterion for diagnosis of restriction.

FEV1 = forced expired volume in 1 second; FVC = forced vital capacity; PEFR = peak expiratory flow rate; MEP = maximum expiratory flow rate at 50% FVC; RV = residual volume; FRC = functional residual capacity; TLC = total lung capacity; TLCO = transfer factor for carbon monoxide; KCO = transfer coefficient; MIP = maximum inspiratory pressure; MEP = maximum expiratory pressure.

Contingency analysis (chi-square) was used to test for possible relationships between variables; where expected cell sizes were less than 5, Fisher's exact test was used.

**Results**

**Clinical information** (Table II). The mean age of the patients was 51.1 years (range 21 - 80 years) and the mean duration of RA was 12.4 years (range 1 - 50 years). The female/male ratio was 2.3:1. Sixty-three patients (60.6%) were seropositive at the time of the study, although a total of 80 patients (76.9%) had at some time been seropositive and 31 patients (30.7%) had subcutaneous nodules. Nodules were significantly commoner in men (P = 0.013). Twenty-five patients were in ARA functional classes 3 or 4, and 16 of these were over 50 years old. Age, duration of RA, seropositivity rate and functional class did not differ significantly between men and women. The mean (SD) haemoglobin levels were 14.0 (SD 1.9) g/dl for men, and 12.4 (SD 1.7) g/dl for women.

Fifty-two patients were current smokers, 17 ex-smokers and 35 non-smokers. Mean cigarette consumption in smokers and ex-smokers was 20.1 pack-years, and a smoking history was significantly commoner in men (P = 0.003).

The occurrence of dyspnoea in 31 patients could be attributed to anaemia, lung or heart disease in 29 patients, some of whom were also smokers or ex-smokers; the remaining 2 were current smokers and did not have any other predisposing factor. Productive cough was present in 7 patients, all of whom had either bronchiectasis or a history of smoking. Twelve patients kept either pigeons or budgerigars and 1, a grindstone operator, was exposed to silica dust; in none of these 13 patients could any disease be attributed to their exposure.

Five patients had digital clubbing, of whom 2 had fibrosing alveolitis and 1 bronchiectasis. No cause of the clubbing was apparent in the remaining 2 patients; 1 of these had evidence of previous pleurisy on the chest radiograph, while the other had valvular heart disease. A total of 47 patients had received gold preparations (oral or intramuscular) and/or d-penicillamine. No relationship was found between any abnormality of the respiratory system and administration of these drugs.

**Prevalence of pulmonary disease.** The clinical, radiographic and physiological findings were used together to determine the prevalence of lung disease in the study population, and the results are given in Tables II and III.

One asymptomatic patient had evidence of a cavitating rheumatoid nodule on the chest radiograph, which regressed spontaneously on serial follow-up examinations. Bronchiectasis was present in 2 patients (1.9%), 1 with Kartagener's syndrome and the other with chronic suppurative lung disease following pulmonary tuberculosis.

Fibrosing alveolitis was diagnosed in 5 patients (4.8%), all of whom had a diffuse pulmonary infiltrate on the chest radiograph, with honeycombing in 3 patients. All 5 patients had clubbing and 1 had clinical evidence of pulmonary hypertension. There was no significant association with gender or with the presence of subcutaneous nodules or seropositivity. Only 2 of the patients had received gold preparations or d-penicillamine. Four of the 5 patients had a history of smoking, 3 of whom were smokers. The predominant pulmonary function abnormality was restriction of lung volumes (P = 0.008). Severe reduction of TLCO was present in 2 of the 4 patients in whom it could be measured.
Table II. Profile of patients with disease affecting the lungs

| Patient grouping | N | Diff inf | Prod cough | Birds/ dust | Sm | Dyspnoea | Clubbing | Mean age (yrs) | Male | Female | Mean durat RA (mo.) | Class | Nod | Sero-pos | Drugs | Obstn | Restn | TLCO | KCO | MIP | MEP | abn | abn | abn | abn |
|------------------|---|----------|------------|-------------|----|----------|----------|---------------|------|--------|-----------------|-------|-----|---------|-------|-------|-------|------|-----|-----|-----|-----|-----|-----|
| All patients     | 104 | 8        | 7          | 13          | 69 | 31       | 5        | 51.1          | 32   |         | 148             | 25    | 31  | 63      | 47    | 31    | 11    | 57   | 21  | 10  | 31  |     |     |     |     |
| Lung nodules     | 1   | 0        | 1          | 0           | 0   | 1        | 0        | 46.0          | 1    | 0      | 216             | 0     | 1   | 1       | 0     | 0     | 0     | 0    | 0   | 1   |     |     |     |     |
| Bronchiectasis   | 2   | 0        | 2          | 0           | 1   | 1        | 1        | 33.5          | 0    | 55     | 288             | 0     | 2   | 2       | 2     | 2     | 2     | 2    | 2   | 2   |     |     |     |     |
| Alveolitis       | 5   | 0        | 5          | 0           | 3   | 4        | 2        | 54.8          | 2    | 18     | 58              | 2     | 2   | 2       | 4     | 2     | 2     | 2    | 2   | 2   |     |     |     |     |
| Pneumonia        | 5   | 0        | 1          | 3           | 3   | 3        | 0        | 53.2          | 1    | 121    | 121             | 1     | 2   | 1       | 4     | 1     | 1     | 1    | 1   | 1   |     |     |     |     |
| Asthma           | 9   | 1        | 0          | 8           | 5   | 1        | 5        | 52.9          | 3    | 11     | 11              | 1     | 2   | 1       | 2     | 1     | 1     | 1    | 1   | 1   |     |     |     |     |
| Pleural disease  | 17  | 3        | 4          | 3           | 12  | 6        | 2        | 54.8          | 7    | 175    | 7                | 5     | 7   | 7       | 12    | 7     | 8     | 2    | 5   | 6   | 1   | 5   |     |     |
| Tuberculosis     | 25  | 4        | 2          | 4           | 18  | 7        | 0        | 53.6          | 10   | 177    | 6              | 7     | 16  | 9       | 6     | 14    | 4     | 3    | 8   |     |     |     |     |     |

Numbers, except where indicated, refer to numbers of patients with each characteristic. The 6 columns on the right of the table indicate pulmonary function abnormalities. Diff inf = diffuse infiltrates on CXR; Sm = smokers or ex-smokers; Durat RA (mo.) = duration of rheumatoid arthritis in months; Class 3/4 = patients in functional classes 3 or 4; Nod = patients with subcutaneous nodules; Drugs = therapy with gold or d-penicillamine; Obstn = airways obstruction; Restn = restrictive abnormality; abn = abnormality.

Five patients (4.8%) had radiographic evidence of past or present pneumonia. In 1, this took the form of a chronic bilateral necrotising pneumonia, in the absence of a history or other evidence of tuberculosis, while a second had collapse and consolidation of the right middle lobe. The remaining 3 had areas of atelectasis on the radiograph. Only the patient with necrotising pneumonia had restriction of pulmonary function, and 2 others, both smokers, had airways obstruction.

Nine patients were diagnosed as having bronchial asthma, all of whom had reversible airways obstruction. TLCO was normal in 7; the 2 in whom an abnormal TLCO was obtained had additional lung disease to account for it — previous tuberculosis, in 1, and fibrosing alveolitis in the other.

Seventeen patients (16.3%) had evidence of past or present pleural disease. In 9 patients, this took the form of diffuse infiltrate on the chest radiograph. A small pleural effusion was present in 2, apical pleural capping in 12, and nonspecific pleural shadows in 3. There was no significant association between pleural abnormality and gender, seropositivity or subcutaneous nodules. Eight of these patients had coexistent cciardiopulmonary disease, viz. tuberculosis in 5, rheumatic fever in 2 and cardiac disease in 1. Pleural disease in 9 patients may therefore be attributable to RA.

The predominant pulmonary function abnormality in the patients with pleural disease was restriction of lung volumes, which was present in 5 of the 17 patients. This association was significant ($P = 0.016$) and remained so even when the patients with previous tuberculosis were excluded. Airflow obstruction was present in 6 patients, but 5 of these were smokers and the sixth had cardiac disease. Eleven patients gave a history of having received treatment for pulmonary tuberculosis, while a further 14 had radiographic evidence of previous tuberculosis. Therefore 25 patients (24.0%) were considered to have had tuberculosis in the past. There was a significant association between a restrictive pulmonary function abnormality and tuberculosis ($P = 0.022$).

Pulmonary function abnormality. As shown in Table II, more patients exhibited abnormalities of pulmonary function than had clinically and radiologically diagnosed lung disease. Airways obstruction was present in a total of 31 patients (29.8%). The association between obstruction and smoking was significant ($P = 0.044$); 25 of the 89 smokers and ex-smokers had obstruction compared with only 6 of the 35 non-smokers. The obstruction in the 6 non-smokers was not severe. The clinical details of these 6 patients were as follows: 2 patients had had previous pulmonary tuberculosis, 1 of whom was also asthmatic; 2 patients had cardiac disease (1 had a coronary artery bypass graft with cardiomegaly and 1 had hypertension and mitral incompetence with a dilated left ventricle); 1 patient, who had a 12.5% increase in FEV, post bronchodilator, had a...
unilateral radiolucent lung. The remaining patient, whose
airways obstruction was not responsive to bronchodilator,
and whose TLCO was 82% of the predicted value, had no
obvious cause of airways obstruction and may have had
bronchiolitis obliterans. Eleven patients, all of whom had
pulmonary disease (Table II), had evidence of a restrictive
pulmonary function defect.

In 57 patients the TLCO was reduced — severely in 5,
moderately in 9, mildly in 10 and minimally in 33. One
patient with mild reduction and 9 with minimal reduction
were non-smokers who had not had TB. Of these 10
patients 3 had cardiac disease with cardiomegaly. Of these,
1 had aortic incompetence, 1 mitral incompetence and 1
had a previous coronary artery bypass graft. One further
patient had previously had pneumonia, and had residual
pleural and pulmonary shadows on the chest radiograph,
and 1 had a unilateral radiolucent lung with mild airflow
obstruction. The remaining 5 patients had no obvious
explanation for the reduced TLCO. The KCO was abnormal
in 21 patients, all of whom were smokers.

Abnormalities of MIP and MEP occurred in 10 and 31
patients respectively, but these were mild or minimal in all
cases, and no clinical association could be found.

Altogether, 56 of 104 patients (53.8%) had one or more
abnormalities of the respiratory system based on clinical,
radiographic or pulmonary function data. Fourteen of these
patients were diagnosed as having pulmonary disease
related to RA, i.e. rheumatoid nodule in 1 patient, fibrosing
alveolitis in 5 patients (1 of whom also had pleural disease)
and pleural disease alone in 8 patients. A further 6 patients
had abnormality of pulmonary function which could not be
explained by clinically or radiographically apparent disease
— fixed airways obstruction in 1 patient and reduced TLCO
in 5 patients. It is therefore possible that a total of 20
patients had pulmonary pathology attributable to rheumatoid
disease.

Discussion

Pulmonary complications are frequently reported in RA, but
their prevalence varies depending on the selection of
patients, the investigations performed, and the criteria used
in the interpretation of investigations, particularly pulmonary
function tests. Estimates of prevalence of interstitial lung
disease, for example, varied from 1.1% when radiological
criteria only were employed to 41% when pulmonary
function criteria were used. The prevalence of interstitial
lung disease based on the presence of diffuse
reticulonodular radiographic shadows was 4.8% in the
present study, which is in close agreement with the 4.5% of
309 patients reported by Jurik et al. and the 5% of 155
patients reported by Hyland et al. Since a further 4.8% of
our patients had an abnormal gas transfer value not
attributable to smoking or other clinically apparent disease,
it is likely that the prevalence of interstitial lung disease in
RA may be underestimated if pulmonary function tests are
not included in the diagnostic work-up of patients. Indeed
the inclusion of lung biopsy, even in patients without
infiltrates on radiographs, may increase the prevalence to
even higher levels. This view is supported by Yousem et al.
who reported on the lack of correlation between
radiographic and histological findings. The use of high-
resolution computed tomography of the chest has also been
shown to help detect early lung disease in RA and it has a
high sensitivity for the diagnosis of interstitial lung disease.

A recent survey, which included broncho-alveolar lavage,
reported a prevalence of interstitial lung disease of 15% in
unselected RA patients. Treatment with gold salts,
penicillamine and other disease-modifying anti-rheumatic
drugs has been reported to cause interstitial lung disease,
but no such association was noted in this study or by
Whorwell et al.

The prevalence of pleural involvement varies from 5% for
pleural effusion to 21% for pleuritic chest pain to 75% for
pleural abnormalities in postmortem studies of patients with
longstanding RA. An analysis of the chest radiograph in
309 RA patients in Denmark showed evidence of previous
pleurisy in 18.8% and pleural effusion in 0.6%. A large
British study of 516 RA patients recorded a history of
pleurisy in 21%, and 3.3% had pleural effusion which was
attributed to RA. We found pleural abnormalities in 17
patients (16.3%), but these may be attributed to RA in only 9
patients (8.7%) who did not have any coexistent
cardiopulmonary diseases. However, it is possible that some
of these patients may have had previous asymptomatic
pleural involvement caused by tuberculosis without evidence
of parenchymal involvement, but this cannot be determined
with certainty. Hyland et al. did not find any evidence of an
increased prevalence of pleurisy in RA. However, the
characteristic changes in the pleural fluid in RA provide
convincing evidence that it is a definite entity.

A significant reduction in the pulmonary gas transfer has
been reported in RA, although Hyland et al. failed to
detect any difference between 155 RA patients and 95
controls. The prevalence of abnormal TLCO was 23.8% and
41% in the studies of Davidson et al. and Frank et al.
respectively. We found a reduction in TLCO in 54.8% of our
patients but the majority were smokers or ex-smokers. A
relationship between smoking and a reduction in TLCO has
been reported in other studies. We found that only 6
(5.8%) of our patients did not have any other contributory
factors for a reduction in the diffusion capacity. These
patients may have had occult interstitial or pulmonary
vascular disease. A similar low prevalence of an abnormal
TLCO was reported by Banks et al., who found a reduced
TLCO in 6 (2.2%) of 264 RA patients in the absence of
clinical or radiographic evidence of lung disease.

Airways obstruction was reported in 32% and 61% of
patients by Geddes et al. and Collins et al. respectively,
but a large Canadian study failed to detect any increase
compared with controls. Acute obliterative bronchiolitis
with a poor prognosis was reported in a series of 6 patients
by Geddes et al. Progressive obtrusive disease of the
peripheral airways in 5 lifetime non-smokers with RA, with
documentation of the histological abnormalities, was
reported by Begin et al. We detected airflow obstruction in
31 (29.8%) of our patients but the majority were smokers.
Only 1 patient had airflow obstruction which could not be
related to asthma, smoking or any other disease; this patient
may have had bronchiolitis obliterans.

The association of infection with RA has been noted in
several reports. A fourfold increase in mortality over an
age- and sex-matched general population has been
reported as a result of respiratory infections in RA.\textsuperscript{14} Tuberculosis is common in the coloured population of South Africa and was noted in 25 (24\%) of our RA patients. A Danish radiographic study reported showed evidence of healed tuberculosis in 9.1\%, with a significant increase in 17.2\% of the RA men compared with 6.1\% in male controls.\textsuperscript{20} We found that 6 patients (5.9\%) had evidence of previous pneumonia and 2 (1.9\%) had bronchiectasis. Bronchiectasis was detected in 16 (3.1\%) of 516 patients reported by Walker and Wright.\textsuperscript{3} A recent high-resolution computed tomography study of 20 patients with radiological evidence of interstitial lung disease and 20 controls without interstitial lung disease detected bronchiectasis in 6 patients with interstitial lung disease and 4 controls without interstitial lung disease, suggesting that bronchiectasis is more common than previously recognised.\textsuperscript{41}

Pulmonary manifestations such as pleural involvement and interstitial lung disease have been reported to occur more commonly in association with nodules and a positive rheumatoid factor.\textsuperscript{42} However, in our survey there was no relationship between these manifestations and the prevalence of nodules and seropositivity.

In conclusion, pulmonary involvement is common in RA and was detected in 20 patients (19.2\%). It is possible that the true prevalence is higher; some of the abnormalities noted in smokers and patients with other diseases (such as tuberculosis) may be due to a combination of factors, including RA, but this cannot be determined with certainty. The pulmonary abnormalities attributable directly to RA were pleural involvement (7.7\%), interstitial lung disease (4.8\%), gas transfer defect in the absence of clinicoradiographic evidence of disease (4.8\%), airflow obstruction in the absence of clinicoradiographic evidence of disease (1\%), and pulmonary nodule (1\%).

Our study provides a perspective on the spectrum of lung disease that may be found in patients with RA in a developing country. It also demonstrates how the use of simple clinical, radiographic and lung function data can be used to stratify abnormalities in a clinically useful manner.

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REFERENCES