Treatment of rapidly progressive rheumatoid pneumoconiosis

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Davies, D. (1973). British Journal of Industrial Medicine, 30, 396-401. Treatment of rapidly progressive rheumatoid pneumoconiosis. Rheumatoid pneumoconiosis is often a fairly benign condition but in some patients it progresses rapidly and causes severe disability. One patient with rheumatoid pneumoconiosis was treated with chloroquine followed by corticosteroids, and another patient was treated with corticosteroids only. Both showed considerable symptomatic and objective improvement with radiographic regression. Treatment has been continued for five and six years respectively but some progression of the disease has again occurred over this time and with a reduced dose of corticosteroids.

The development of progressive massive fibrosis (PMF) in coal workers correlates well with the total amount of dust in the lungs, as indicated by the background of simple pneumoconiosis. The prevalence of PMF rises from very low levels in men with category 0 or category 1 pneumoconiosis to about 30% in those with category 3 (Cochrane, 1962). When PMF appears with a background of category 0 or category 1 there is a likelihood that this will prove to be rheumatoid pneumoconiosis, the subjects having rheumatoid arthritis or rheumatoid factor in their serum (Lindars and Davies, 1967). The presence of rheumatoid disease reduces the amount of dust retention that is usually necessary for the development of PMF. For the purposes of this paper rheumatoid PMF is defined as rheumatoid pneumoconiosis where one or more lesions are 1 cm in diameter or larger.

Once PMF has appeared, its rate of progression is known to correlate only with age: the younger the man, the faster the progression (Cochrane et al., 1956). It might be expected that a faster rate of progression would be seen in rheumatoid PMF where there is an obvious disturbance of autoimmunity. But in a reasonably long-term study of coal miners with rheumatoid pneumoconiosis (Davies and Lindars, 1968) it was found that in some the disease was mild and static and not associated with disability. Some men, however, particularly those with obvious arthritis and a substantial background of simple pneumoconiosis, showed considerable progression and disability.

This paper reports the results of treatment in two coal miners with rapidly progressive rheumatoid PMF in which an attempt was made to slow down or reverse the PMF using drugs known to suppress rheumatoid arthritis.

Case reports

Case 1
H.T. worked underground from 1935 to 1961, with the exception of five years in the Army. The first chest radiograph taken in 1961, when he was 37 years old, showed category 2 simple pneumoconiosis, the opacities being predominantly of the punctiform type. His application to a Pneumoconiosis Panel for certification was rejected and he continued underground work. A further radiograph in 1963 showed category 3 simple pneumoconiosis and he was then certified. He remained at the coal face until 1965 when he changed to work on supplies.

In 1966 he became short of breath and the radiograph showed considerable deterioration (Fig. 1). There were multiple irregular opacities throughout both lungs, the largest being 1 cm in diameter. Six months later he developed rheumatoid arthritis and took an office job.

He was referred to me in December 1967 because of increasing dyspnoea and bilateral pleural pain of several
FIG. 1. Case 1. Chest radiograph in May 1966 showing multiple widespread irregular nodules measuring up to 1 cm in diameter.

FIG. 2. Case 1. By December 1967 there was marked progression. There is some cavitation in a nodule below the left clavicle.
months’ duration. He smoked a pipe but had very little
cough or sputum. On examination he had active poly-
arthritis but no subcutaneous nodules. He looked unwell
and there were pleural rubs over both lungs. Ventilatory
tests (vital capacity and forced expiratory volume in
1 second) showed marked restriction while the carbon
monoxide diffusing capacity and arterial carbon dioxide
were normal. Radiographs showed extensive large
irregular opacities in both lungs and cavitation below
the left clavicle (Fig. 2).

The pain and pleural rubs persisted and in January
1968 he was started on chloroquine sulphate, 600 mg
daily. Within three weeks the pain became much less
severe, the pleural rubs disappeared, he became less
dyspnoeic, and his arthritis improved. After six weeks he
was free of chest pain and went back to work. Chloro-
quine was reduced to 400 mg daily after two months
and was stopped after four months because of the appear-
ance of retinal changes, though vision was not affected.
He was then put on prednisolone, 15 mg daily. Moderate
radiographic improvement was seen throughout the
period of treatment and the appearances in June 1968
are shown (Fig. 3).

He remained fairly well thereafter, and the daily dose
of prednisolone was reduced in steps to 7.5 mg in August
1970. In 1971 he had left pleural pain for two weeks but
this cleared without a change in treatment.

At the end of 1972 he felt well but was dyspnoeic on
hills and stairs. He had a slight cough with sputum and
had given up smoking. The joints were occasionally
painful but showed no swelling nor deformity. His fundi
showed some macular pigmentation. The improvement in
vital capacity (VC) was maintained but there had been
a fall in the forced expiratory volume in one second
(FeV₁) from the best level reached in June 1968 (Table 1).
There had also been some radiographic deterioration
since that time but the appearances were similar to those
before treatment (Fig. 2). The dose of prednisolone was
increased to 10 mg daily. Tubercle bacilli were not found
in the sputum at any time.

Case 2
W.P. began working underground in 1926 at the age of
14. He did not go on to the coal face until 1937 and he
became a shot firer in 1958. He was referred to me in
1960 complaining of some cough, sputum, and mild
dyspnoea for two years. He had given up smoking in
1955. There was no significant abnormality on examina-
tion and his chest radiograph showed category 2 simple
pneumoconiosis. The nodules were fairly scanty and
variable in size, a few measuring up to 8 mm in diameter.
He was certified by the Pneumoconiosis Medical Panel
as a case of pneumoconiosis.

He continued with underground work but was off
periodically with episodes of cough and sputum. His
ventilatory tests (vital capacity and forced expiratory
volume in 1 second) showed evidence of moderate airways obstruction. Rheumatoid arthritis developed in 1963 and he left underground work. By 1964 there was marked radiographic deterioration. A large opacity on the right measured $8 \times 3.5$ cm and there were multiple nodules up to $1.2$ cm in diameter in the remainder of the lungs. A year later he had melanoptysis and haemoptysis and several cavities appeared in the area of PMF in the right lung. The cavities filled up again and the other opacities on both sides became larger by early 1967 (Fig. 4). At that time he was more short of breath, still coughing up some blood occasionally, and he had some right-sided pleuritic pain.

In January 1967 he was given prednisolone, 15 mg daily. Over a few weeks the pleuritic pain disappeared, his breathlessness became less, and the ventilatory and diffusing capacity tests improved (Table 2). The arthritis remained troublesome. There was considerable radio-

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**TABLE 1**

**CASE 1. RESULTS OF VENTILATORY TESTS, ROSE-WAALER TEST, AND ERYTHROCYTE SEDIMENTATION RATE: STARTED ON CHLOROQUINE JANUARY 1968**

<table>
<thead>
<tr>
<th></th>
<th>$VC$ (l.)</th>
<th>$FEV_1$ (l.)</th>
<th>Rose-Waaler titre</th>
<th>ESR (Westergren) (mm in 1 hr)</th>
<th>Prednisolone (mg daily)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Expected value 1968</td>
<td>3.50</td>
<td>2.90</td>
<td>1:320</td>
<td>96</td>
<td></td>
</tr>
<tr>
<td>January 1968</td>
<td>1.90</td>
<td>1.70</td>
<td>1:160</td>
<td>84</td>
<td></td>
</tr>
<tr>
<td>February 1968</td>
<td>2.60</td>
<td>2.50</td>
<td>1:160</td>
<td>59</td>
<td>15</td>
</tr>
<tr>
<td>May 1968</td>
<td>2.60</td>
<td>2.55</td>
<td>1:160</td>
<td>59</td>
<td>12.5</td>
</tr>
<tr>
<td>June 1968</td>
<td>3.00</td>
<td>2.85</td>
<td>1:80</td>
<td>45</td>
<td>10</td>
</tr>
<tr>
<td>May 1969</td>
<td>2.80</td>
<td>2.20</td>
<td>Negative</td>
<td>44</td>
<td>7.5</td>
</tr>
<tr>
<td>September 1971</td>
<td>2.75</td>
<td>2.15</td>
<td>1:64</td>
<td>34</td>
<td></td>
</tr>
<tr>
<td>November 1972</td>
<td>2.70</td>
<td>2.00</td>
<td></td>
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<td></td>
</tr>
</tbody>
</table>

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**FIG. 4. Case 2. Radiograph in January 1967 showing multiple irregular opacities in both lungs, more extensive on the right.**
**TABLE 2**

**CASE 2. RESULTS OF VENTILATORY TESTS, CARBON MONOXIDE DIFFUSING CAPACITY (DCO, STEADY STATE), ROSE-WAALER TEST AND ERYTHROCYTE SEDIMENTATION RATE**

<table>
<thead>
<tr>
<th>Date</th>
<th>VC (l)</th>
<th>FEV1 (l)</th>
<th>DCO (ml/mm Hg/min)</th>
<th>Rose-Waaler titre</th>
<th>ESR (Westergren) (mm in 1 hour)</th>
<th>Prednisolone (mg daily)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Expected value 1962</td>
<td>3.80</td>
<td>3.30</td>
<td>17.9</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>May 1962</td>
<td>4.30</td>
<td>2.90</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>July 1964</td>
<td>3.30</td>
<td>2.90</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>December 1966</td>
<td>3.10</td>
<td>2.25</td>
<td>9.9</td>
<td>Positive</td>
<td>1:160</td>
<td>22</td>
</tr>
<tr>
<td>January 1967</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>March 1967</td>
<td>3.20</td>
<td>2.70</td>
<td>17.3</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>September 1967</td>
<td>3.50</td>
<td>2.70</td>
<td>16.1</td>
<td>1:320</td>
<td>3</td>
<td>10</td>
</tr>
<tr>
<td>July 1968</td>
<td>3.60</td>
<td>2.85</td>
<td>16.8</td>
<td>1:160</td>
<td>22</td>
<td>7.5</td>
</tr>
<tr>
<td>June 1970</td>
<td>3.60</td>
<td>2.60</td>
<td></td>
<td>1:320</td>
<td>90</td>
<td>10</td>
</tr>
<tr>
<td>December 1971</td>
<td>3.10</td>
<td>2.40</td>
<td>9.4</td>
<td>1:64</td>
<td>16</td>
<td>6.25</td>
</tr>
<tr>
<td>October 1972</td>
<td>2.90</td>
<td>2.40</td>
<td>11.4</td>
<td>1:32</td>
<td>10</td>
<td>6.25</td>
</tr>
</tbody>
</table>

Graphic improvement by June 1968 (Fig. 5) and prednisolone was reduced to 7.5 mg daily. A year later the dose was increased to 10 mg because of radiographic deterioration and a rise in erythrocyte sedimentation rate. He gave up work because of arthritis, some shortness of breath, and domestic difficulties.

He is now on 6.25 mg of prednisolone a day. He is still unable to keep up with people of his age on the flat, and the lung function test results have fallen to the 1966 levels. Gradual radiographic deterioration has occurred but the position is still better than when he started prednisolone (Fig. 4). Sputum tests for tubercle bacilli...

**FIG. 5.** Case 2. In June 1968, after 18 months on prednisolone, the opacities are much smaller.
have been repeatedly negative. The arterial carbon
dioxide tension was normal before the start of treatment
and remains so. The oxygen tension is also normal but
was not measured at the start.

Discussion
Kandus (1971) treated 22 patients with rheumatoid
pneumoconiosis with chloroquine sulphate, 250 mg
daily, for a minimum of six months. No effect on the
evolution of the disease was observed. The duration
of treatment seems to have been too short and it is
doubtful if the dose was adequate. Personal experi-
ence has shown that sarcoid lesions suppressed by
chloroquine in a larger dose may progress when the
chloroquine is reduced to this level. Some beneficial
effect from corticosteroids has been claimed by

The best way of judging whether treatment pro-
duces regression or slows the progression of
rheumatoid pneumoconiosis would be to allocate a
number of patients at random to treatment and
control groups. Because some patients have non-
progressive or slowly progressive disease it seems
likely that a fairly large number would be required
and treatment should be continued for some years
before conclusions could be reached. Many patients
have obviously been treated with corticosteroids and
other drugs solely because of their arthritis. An analysis
of the rate of progress of the pneumoconiosis in such patients, compared with others not so treated,
would be feasible though very difficult. As studies of
this kind have not been reported it seems reasonable
to draw some conclusions from observations on two
rapidly deteriorating cases.

The rheumatoid pneumoconiotic nodules in case 1
were evidently subpleural, as is often the case with
rheumatoid nodules in the lungs of people not
exposed to dust (Davies, 1966). Chloroquine was
chosen as initial treatment simply to see if it worked.
The response before changing to corticosteroids was
gratifying and further improvement was seen on
prednisolone. Though there has been some deteri-
oration since, the radiographic picture now is no
worse than five years ago and he has no pleural
complications. The striking early improvement in
ventilatory tests was more the result of pain relief
than of changes within the lung.

In case 2, the early radiographic improvement was
more striking. Again, with the passage of time the
disease is showing slow progression but the position
is still better than six years ago.

The evidence is strong that treatment produced a
marked reversal of previously rapid deterioration.
The dose of prednisolone was kept low and a larger
one might have prevented the gradual deterioration
that is now occurring. In judging the results of treat-
ment in rheumatoid pneumoconiosis it has to be
remembered that the nodules often cavitate and
shrink. They usually fill up again but this is not
invariable and some are replaced by linear scars.
Though cavitation was evident at various stages in
these patients, it does not account for their improve-
ment.

The majority of patients with rheumatoid pneu-
moconiosis do not merit treatment, but in a few with
rapidly progressive disease the prognosis is bad and
there is now some evidence that treatment can help.
These two men were selected for treatment from
about 100 patients with rheumatoid pneumoconiosis
known to me.

References
Cochrane, A. L. (1962). The attack rate of progressive massive
fibrosis. British Journal of Industrial Medicine, 19, 52-64.
——, Carpenter, R. G., Clarke, W. G., Jonathan, G., and
Moore, F. (1956). Factors influencing the radiological
progression rate of progressive massive fibrosis. British
Journal of Industrial Medicine, 13, 177-183.
——, and Lindars, D. C. (1968). Rheumatoid pneu-
moconiosis, a clinical study. American Review of Respiratory
Diseases, 97, 617-629.
Kandus, J. (1971). Results of long-term chloroquine therapy
in rheumatoid pneumoconiosis. Studia Pneumologica et
Phisiolegica, Czechoslovakia, 31, 229-232. (In Czech,
English abstract).
Lindars, D. C., and Davies, D. (1967). Rheumatoid pneu-
moconiosis; a study in colliery populations in the East
Midlands coal field. Thorax, 22, 525-532.
Caplan's syndrome. A clinicopathologic study. American
Journal of Medicine, 37, 643-652.

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