ACUTE LUNG CHANGES IN RHEUMATOID ARTHRITIS

BY

E. R. BECK AND B. I. HOFFBRAND

From the Medical Research Council Department of Clinical Research, University College Hospital Medical School, London, and University College Hospital, London

Much has been written about the controversial subject of respiratory system involvement in rheumatoid arthritis since the paper of Ellman and Ball (1948). It is now generally accepted that certain respiratory conditions, although more or less uncommon, are true manifestations of rheumatoid disease. These are rheumatoid nodules occurring in pleura and lungs (Robertson and Brinkman, 1961) and nodular fibrosis associated with co-existing pneumoconiosis (Caplan 1953, 1959); also pleurisy and pleural effusions (Horler and Thomson, 1959; Ward, 1961) and diffuse interstitial pulmonary fibrosis (Lee and Brain, 1962; Brannan, Good, Divertie, and Baggenstoss, 1964) may occur in rheumatoid disease without any other aetiology.

Although mention is made in the last two papers of acute pneumonia-like illnesses at the onset or during exacerbations of the chronic pulmonary disease, little attention has been paid to acute lung changes in rheumatoid arthritis. In patients with rheumatoid arthritis there are obvious difficulties in excluding associated or purely coincidental causes of acute pulmonary lesions and in attributing such changes to the rheumatoid process itself. We report here three cases of rheumatoid arthritis with acute lung lesions believed to be manifestations of rheumatoid disease as they occurred in association with the recognized rheumatoid phenomena of pleurisy and pericarditis (Tarpley, 1961; Wilkinson, 1962).

The diagnostic criteria for rheumatoid arthritis are those of the American Rheumatism Association (Ropes, Bennett, Cobb, Jacox, and Jessar, 1959).

Case Reports

Case 1, a 46-year-old waitress, presented in 1959 with a history of precordial pain, anorexia, night sweats, and breathlessness on climbing stairs for one week, without cough or sputum. She gave a history of rheumatoid arthritis affecting the hands, elbows, wrists, knees, and shoulders since her late teens, for which she took aspirin.

Examination.—She was initially afebrile. A pericardial friction rub was heard. Marked changes of chronic rheumatoid arthritis were present in the hands and left knee and she had palmar erythema.

Investigations.—Hb 77 per cent. with a normochromic film; white blood count 8,000 with normal differential; erythrocyte sedimentation rate 63 mm. in 1 hr (200 mm. tube). Electrocardiogram showed T wave changes compatible with pericarditis. Serial chest radiographs showed a globular, enlarged cardiac silhouette with the appearance of linear opacities and, later, partial collapse in the right lower lobe; a left intra-pulmonary opacity and "pleural reaction" were present (Fig. 1).

Fig. 1.—Case 1, acute lung changes with pericardial effusion.

A frank left pleural effusion later developed. No L.E.-cells were found on several examinations of the
blood. The urine was sterile and contained no deposit on repeated examination. The following tests gave negative results: Mantoux test 1:100, Paul Bunnell, cold agglutinins, streptococcus M.G.

Progress.—She developed an irregular pyrexia up to 101° F. and signs of early pericardial tamponade with a slightly raised jugular venous pressure which was not paradoxical. Steroid therapy resulted in rapid clinical and radiological improvement.

When seen 5½ years later she had had no further symptoms related to the cardio-respiratory system. Chest x ray and spirometry were normal. Waaler-Rose test 1:512; R.A. latex test positive.

Comment.—Long-standing classical rheumatoid arthritis with a single episode of pleuro-pericarditis, in which pericarditis was the dominant clinical feature but in which acute lung changes occurred.

Case 2, a 68-year-old housewife, presented in 1962 with a 4-day history of right-sided pleuritic pain and dyspnoea, not associated with cough or sputum. Rheumatoid arthritis affecting most peripheral joints had been present and active since 1959, and had been treated with prednisone from its onset.

Examination.—She was acutely ill and febrile (102.6° F.). Pleural rubs were heard bilaterally. The jugular venous pressure was raised. Severe active rheumatoid arthritis was present.

Investigations.—Hb 60 per cent. with normal red cell indices; white blood count 18,400 (84 per cent. polymorphs) rising to 57,000 (91 per cent. polymorphs); erythrocyte sedimentation rate 37 mm. in 1 hr. Waaler-Rose titre 1:2,048; R.A. latex test strongly positive; No L.E.-cells seen in the blood on numerous occasions during and after admission. Electrocardiogram showed typical changes of acute pericarditis. Chest radiograph showed pericardial and left pleural effusions with a “collapse-consolidation” appearance of the left lower lobe (Fig. 2). The pleural fluid contained many red cells and polymorphs and was sterile on culture for routine organisms and acid-fast bacilli on two occasions.

Progress.—She was treated with antibiotics and an increased dosage of prednisone. No sputum was produced at any stage. Culture of urine and nose swab was negative. A 48-hour episode of hypotension and coma responded to pressor agents and further prednisone and intravenous hydrocortisone. Anticoagulation was undertaken because of the possibility of pulmonary embolism, though there was no haemoptysis, deep-vein thrombosis, or associated Electrocardiogram change in support of this diagnosis. She made a slow recovery over the next 4 weeks.

When seen in 1965 she had no chest symptoms and a chest radiograph showed complete clearing.

Comment.—Severe active classical rheumatoid arthritis of late onset complicated by acute pleuropericarditis with dominant pleural changes and lung involvement. A marked polymorpholeucocytosis occurred without evidence of bacterial infection.

Case 3, a 27-year-old housewife, was admitted to hospital in 1963 with a 2-day history of left-sided pleurisy, dyspnoea, non-productive cough, and delirium. After the birth of her third child 2 years previously she had developed rheumatoid arthritis affecting the hands, wrists, shoulders, knees, and ankles and had been treated with aspirin and chloroquine. She had a long history of Raynaud’s phenomenon in the hands.

Examination.—She was acutely ill and febrile (101° F.). A pleural friction rub and signs of consolidation were present at the base of the left lung and there was tachypnoea. Swelling of the metacarpophalangeal joints was present.

Investigations.—Hb 92 per cent.; white blood count up to 15,700 (88.5 per cent. polymorphs); erythrocyte sedimentation rate 56 mm. in 1 hr. Serial chest radiographs showed consolidation at the base of the left lung with opacities in the right mid-zone and right base and, later, pericardial and bilateral pleural effusions (Fig. 3, opposite).

Waaler-Rose titre 1:512; R.A. latex test positive; no L.E. cells seen in the blood on numerous occasions. Electrocardiogram showed changes compatible with
LUNG CHANGES IN RHEUMATOID ARTHRITIS

pericarditis. The following bacteriological and virological investigations were negative: culture of sputum for pathogens and acid-fast bacilli; culture of blood and urine; monkey kidney and HeLa cell culture for cytopathogenic agents; culture for influenza A, B, and C; complement-fixation tests on paired sera for influenza A, B, and C, parainfluenza 1, 2, and 3, adenovirus and psittacosis-lympho-granuloma venereum groups, Q fever; streptococcus M.G. agglutinin.

Progress.—She remained very ill with fever (104° F.) and pleural and pericardial friction rubs despite treatment with various antibiotics, but the introduction of steroids produced rapid clinical improvement and resolution of the lung changes, though 6 months later she had a recurrence of pleurisy. She was well with a normal chest radiograph 2 years later, but she still has some breathlessness despite continuing on steroids (Fig. 4).

Lung Function Tests.—Spirometry showed restricted ventilation (FEV₁: FVC 1,950 : 2,250 ml.) associated with reduced lung compliance (0·034 1/cm.H₂O) when studied 3 months after the onset of the illness. Carbon monoxide diffusion capacity at rest and on exercise, mixed venous CO₂ tension, and peak expiratory flow rate were within normal limits. Serial spirometry shows improvement though it is still impaired 18 months later [FEV₁: FVC = 2,300 : 2,600 ml. (predicted value FVC = 3,350)].

Comment.—Mild definite rheumatoid arthritis with acute lung changes associated with pleuro-pericarditis resulting in a serious pneumonic illness. A polymorpholeucocytosis occurred without evidence of bacterial infection, and extensive virological studies proved negative. Lung function studies showed a restrictive type of defect which still persists in this patient.

Discussion

These cases illustrate lung involvement of a wide range of severity in rheumatoid arthritis, associated with pleuro-pericarditis. Case 1 closely resembles some of the cases of Tarpley (1961) and Wilkinson (1962) which, although reported as "rheumatoid pericarditis", showed similar acute pleural and pulmonary lesions. In Case 2 the major clinical feature was pleurisy with effusions. In Case 3 the effects of the lung changes themselves dominated the clinical picture.

Our criteria of diagnosis depended on serous membrane involvement, but it seems probable that some patients with rheumatoid arthritis may develop just such lung changes without detectable pericarditis or pleurisy, when and in such cases a correct
diagnosis may prove difficult if this possibility is not borne in mind.

The clinical picture produced by the lung changes described here is complicated by the associated pleurisy and pericarditis. However, there was dyspnoea and toxaemia with little or no cough and sputum and, radiologically, atelectasis was prominent. These features, together with the lung function studies in Case 3, who remained dyspnoeic for some weeks after the acute episode and still has some respiratory disability, suggest a relationship to the lung changes in systemic lupus erythematosus (SLE) (Hoffbrand and Beck, 1965). Although diffuse interstitial pulmonary fibrosis occurs in rheumatoid arthritis, with or without the finding of L.E.-cells, we have been unable to find a record of it in SLE, despite the frequency of pulmonary involvement in this condition (Purnell, Baggenstoss, and Olsen, 1955; Brannan and others, 1964). Our patients differed from the typical rheumatoid subject with chronic lung changes described by Brannan and others (1964) in not having subcutaneous nodules, not developing pulmonary fibrosis at a later stage, and in being female. It is probable that the absence of fibrosis after such acute pneumonic episodes in rheumatoid arthritis leads to the apparent discrepancies in studies such as that of Talbott and Calkins (1964). These workers found no “specific pathology” at autopsy in 37 unselected cases of rheumatoid arthritis compared with a control group, yet they made no attempt to explain the considerably higher incidence in these cases of “non-specific increased densities” and “pneumonitis” recorded radiologically during life.

We believe that acute lung changes do occur in rheumatoid arthritis and are often associated with acute pleuro-pericarditis. They are presumably of an inflammatory nature although atelectasis is prominent and cough and sputum minimal. These lung changes can dominate the clinical picture, and steroid therapy in such cases (as in our Case 3) can be life-saving.

**Summary**

Attention is drawn to the occurrence in otherwise typical rheumatoid arthritis of acute lung changes, often with pleurisy and pericarditis. These changes are believed to be distinct from those giving rise to chronic pulmonary disease such as nodule formation and diffuse fibrosis.

We wish to thank the Physicians of University College Hospital for permission to study their patients.

**REFERENCES**


**Lesions pulmonaires aigües dans l’arthrite rhumastrmale**

**Résumé**

On attirer l’attention sur le fait que dans l’arthrite rhumastrmale bien typique on peut voir l’occurrence des lésions pulmonaires aigües, souvent accompagnées de pleurésie et de péricardite. On pense que ces lésions sont distinctes de celles qui provoquent la maladie pulmonaire chronique, telles que formation de nodules et fibrose diffuse.

**Lesiones pulmonares agudas en la artritis reumatoide**

**SUMARIO**

Se llama atención al hecho de que en la artritis reumatoide por lo demás típica pueden ocurrir lesiones pulmonares agudas, a menudo con pleurésia y pericarditis. Se cree que estas lesiones son distintas de las que producen la enfermedad pulmonar crónica, tales como formación nodular o fibrosis difusa.
Acute Lung Changes in Rheumatoid Arthritis

E. R. Beck and B. I. Hoffbrand

doi: 10.1136/ard.25.5.459

Updated information and services can be found at:
http://ard.bmj.com/content/25/5/459.citation

**Email alerting service**

Receive free email alerts when new articles cite this article. Sign up in the box at the top right corner of the online article.

**Notes**

To request permissions go to:
http://group.bmj.com/group/rights-licensing/permissions

To order reprints go to:
http://journals.bmj.com/cgi/reprintform

To subscribe to BMJ go to:
http://group.bmj.com/subscribe/