Bronchoalveolar lavage in ankylosing spondylitis

D Wendling, J C Dalphin, B Toson, A Depierre, M Guidet

Abstract
Bronchoalveolar lavage and bronchial biopsies were performed in 15 patients with ankylosing spondylitis (AS) and 17 control subjects. There was no difference in total cell count, number of lymphocytes, CD4+/CD8+ ratio, or β2 microglobulin concentrations in bronchoalveolar lavage fluid between these two groups. Bronchoalveolar lavage IgA concentrations were not increased, but bronchial IgA deposits were more common in AS. This study failed to show any subclinical alveolitis in AS.

Pulmonary manifestations are well recognised extrarheumatological features of ankylosing spondylitis (AS). Fibroblaluss apical pulmonary disease is classic but uncommon, estimated to occur in 1–2% of patients with AS. Restrictive pulmonary function impairment is more common, probably related to mechanical factors and limited chest expansion and sometimes associated with reduced diffusion capacity for carbon monoxide (TLCO).

Bronchoalveolar lavage allows further investigation and comprehension of pulmonary involvement in chronic inflammatory diseases such as Sjögren’s syndrome, rheumatoid arthritis, and collagen-vascular diseases, but, to the best of our knowledge, has not yet been performed in AS.

The purpose of this study was to evaluate bronchoalveolar lavage data and study bronchial immune deposits in AS.

Patients and methods
Fifteen consecutive patients with AS (11 male, four female), meeting the New York criteria, were included in the study. Mean age (SD) was 38 ± 1 (13–1) years and 13 were B27 positive. Mean duration of the disease was 6.9 years (range 1–20). Ten were cigarette smokers (four light, four medium, two heavy; mean 19 pack-years (number of packs a day × number of years of smoking)).

Five patients had history of exposure to metallic dust. None had history of chest infection.

This group was compared with 17 controls with no inflammatory disease or chest infection (mean age (SD) 54 ± 2 (11–8) years). Ten were cigarette smokers (in a range matching that of the group with AS; mean 25 pack-years). Two patients had history of exposure to industrial dust and four to agricultural dust.

Informed consent was obtained from all patients.

Each patient underwent physical pulmonary examination by chest x ray, pulmonary function tests, bronchic fibrosopy, and bronchoalveolar lavage according to the technique described elsewhere. Cytological, immunological (CD4 and CD8 staining), and immunoglobulin evaluations (expressed as an IgG/K+ ratio) were performed on bronchoalveolar lavage fluid. Bronchial biopsy specimens were also studied by light microscopy and immunofluorescence examination.

Statistical analysis of the data used was by Student’s t test and the Mann-Whitney test.

Results
In the group with AS physical pulmonary examination was normal in all patients. Chest x rays showed basal interstitial syndrome in two cases.

Four patients had restrictive pulmonary function impairment and four reduced TLCO in pulmonary function tests. Light microscopy of the bronchial biopsy specimens showed no specific modification.

Bronchoalveolar lavage and immunofluorescence data for the patients with ankylosing spondylitis and for controls

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*Values given as mean (SD).
†AS = ankylosing spondylitis.
‡Fibrinogen was not measured in all cases.

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Table 3
The table summarises the bronchoalveolar lavage and immunofluorescence data. Total cell count and macrophage percentage were greater in smokers than in non-smokers (as expected) both in controls and in patients with AS.

There was no change in the bronchoalveolar lavage fluid concentrations of \( \beta_2 \) microglobulin and angiotensin converting enzyme in patients with AS compared with controls and no correlation between bronchoalveolar lavage fluid IgA concentrations and total cell count or lymphocyte percentage in the group with AS.

Discussion
The difference in total cell count, lymphocyte percentage, and CD4⁺/CD8⁺ ratio in bronchoalveolar lavage fluid between patients with AS and controls was not statistically significant. This study failed to show any subclinical alveolar inflammation in AS in comparison with rheumatoid arthritis or other chronic inflammatory diseases.

Bronchoalveolar IgA concentrations are lower, though not significantly, in patients with AS. The lung does not seem to contribute to the serum IgA increase commonly observed in AS.

As previously shown in the skin, IgA deposits in bronchial mucosa are more common in patients with AS than in controls.

Conclusion
This absence of subclinical alveolar inflammation is an argument in favour of mechanical rather than immunological pathogeny of pulmonary fibrosis in AS.
