Letters

Adenosine deaminase activity in rheumatoid pleural effusion

SIR, We read with interest the article by Ocaña et al describing nine cases of rheumatoid pleural effusion, all with raised levels of adenosine deaminase activity in pleural fluid but none with specific changes in pleural biopsy. We have recently seen a rheumatoid patient who developed a pleural effusion with raised levels of adenosine deaminase and showed a typical rheumatoid nodule in the pleural biopsy specimen.

The patient, a 64 year old woman, had developed seropositive, nodular rheumatoid arthritis three years before admission. She had to stop gold salt injections because of a toxic skin reaction and she was receiving D-penicillamine at the time of admission to hospital, with good control of her arthritis.

Two months before admission she started a cough with mucoid sputum, mild left thoracic discomfort, and disturbed sensation. She also had mild constitutional symptoms. There was no flare up of her arthritis. A chest radiograph showed left pleural effusion. Physical examination showed an afebrile patient with signs of left pleural effusion, ulnar deviation, synovial proliferation of her wrists, and mild arthritis in her knees. No subcutaneous nodules were found. The rest of the general examination was normal. Laboratory findings were: erythrocyte sedimentation rate 86 mm/h, haemoglobin 118 g/l, white blood cell count normal, platelets 540×10⁹/l. Blood chemistry and urine analysis were normal. Rheumatoid factor was 1-090 IU. Antinuclear factor was positive at a dilution of 1/6400 with homogeneous pattern. Anti-dsDNA was negative.

Thoracentesis gave a yellowish fluid with 1-15×10⁶ cells/l, 75% mononucleated and 25% polymorphonucleated. Glucose was 56 mmol/l and proteins 56 g/l. Adenosine deaminase activity increased to 89 U/l. Pleural fluid cultures were negative and there were no malignant cells. Pleural biopsy with Abrams’ needle showed the presence of a typical rheumatoid nodule (Fig. 1). Ziehl’s stain and Löwenstein’s culture of pleural tissue were negative.

An infectious or neoplastic aetiology was ruled out, treatment started with 8 mg daily of methylprednisolone, and the patient’s condition improved.

In the cases of rheumatoid pleural effusion reported by Ocaña et al a study of the pleural fluid did not show any difference from the usual findings in tuberculosis patients, including increased adenosine deaminase activity. Pleural biopsy was also non-specific in all the cases reported. Therefore the diagnosis of rheumatoid pleural effusion was made by exclusion.

In our patient, findings in the pleural fluid were consistent with those reported by Ocaña et al, but diagnosis of rheumatoid aetiology was reinforced by the presence of a typical rheumatoid nodule in the pleural biopsy specimen.

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References


Pulse methylprednisolone therapy in rheumatoid arthritis

SIR, Over the last 12 months several publications about the use of pulse methylprednisolone therapy in the treatment of rheumatoid arthritis have appeared, reporting the