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SHORT COMMUNICATION

Low value of adenosine deaminase in tuberculous pleural effusions

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It has been claimed that a value of adenosine deaminase (ADA) activity lower than 43 $U \cdot l^{-1}$ excludes tuberculous aetiology of pleural effusion [1, 2].

We report nine cases of tuberculous pleurisy with at least one determination of ADA (by Giusti and Galanti) activity less than 43 $U \cdot l^{-1}$, and two other cases with an ADA value below 44 $U \cdot l^{-1}$, out of 114 ADA determinations (representing 8% or 10%). The tuberculosis diagnosis was confirmed by bacteriological cultures of pleural fluid or biopsy material and the pathological finding of a granulomatous lesion in the pleural tissue.

In the literature we have found eighteen cases of tuberculous pleurisy with an ADA activity value <43 $U \cdot l^{-1}$ [3–5]; however, among the largest series of ADA and tuberculosis no cases with a value of ADA activity <43 $U \cdot l^{-1}$ were described [1, 2, 6]. In five of the nine cases reported with low ADA activity values, a second determination a few days later evidenced a rise in the ADA activity over 43 $U \cdot l^{-1}$ to values well within the range of tuberculous effusion (fig. 1). In another two cases a second ADA determination six and eight days later, respectively, still showed a value below 43 $U \cdot l^{-1}$. In the last two cases only one determination was obtained.

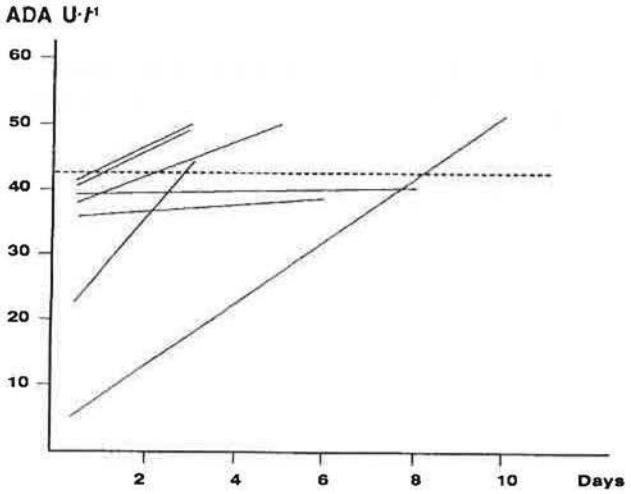

Fig. 1. – Individual values of adenosine deaminase (ADA) activity. On the left first ADA determination, the days between, and on the right the value of the second determination.

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In 1988 [7] a single case of a low first determination of ADA activity (27 $U \cdot l^{-1}$) was reported, which increased to a value of 91 $U \cdot l^{-1}$ on a second sample.

In agreement with previous studies [8] we did not find a correlation between the cellular and differential count in the exudate and the level of ADA activity.

Determination of ADA activity in countries with high prevalence of tuberculosis is considered to be of great value in the diagnosis of tuberculous pleural effusion; so that frequently the diagnosis is based on clinical grounds plus a high ADA determination in pleural exudate, 43 $U \cdot l^{-1}$ being the reference limit widely accepted [1, 2, 6]. Nevertheless in a few patients the ADA activity remains low, making it difficult to accept a sensibility of 100% as it has been reported in the largest series.

Furthermore, new aetiologies with ADA activity in the range of tuberculosis, that is >43 $U \cdot l^{-1}$, are described, including: lymphoproliferative disorders, bronchogenic carcinoma, pulmonary embolism, systemic lupus erythaematosus (SLE), rheumatoid arthritis (RA), empyema, liver disease, mesothelioma, parapneumonic and idiopathic effusions. This fact decreases the specificity of ADA determination.

When a clinical suspicion of tuberculous aetiology for a pleural effusion exists, we recommend a second determination of ADA activity in pleural fluid, if the first was negative for tuberculosis (<43 $U \cdot l^{-1}$), a few days later to obviate the possibility of the fact here reported. In our experience an ADA activity of less than 43 $U \cdot l^{-1}$ cannot exclude the tuberculous aetiology of an effusion.

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Faible valeur de la déaminase adénosine dans les épanchements pleuraux tuberculeux. J.M. Querol, F. Barbe, F. Manresa, L. Esteban, C. Cañette.

RÉSUMÉ: L'on a affirmé qu'une faible activité de la déaminase adénosine (ADA) (<43 $U \cdot l^{-1}$) dans les exsudats pleuraux, permettait d'exclure l'origine tuberculeuse de l'épanchement. Nous présentons neuf cas de tuberculose dont les valeurs sont inférieures à 43 $U \cdot l^{-1}$ (et deux autres inférieures à 44 $U \cdot l^{-1}$). En outre, nous avons trouvé 18 cas de ce type dans la littérature. Tant parmi les cas publiés que parmi les nôtres, une deuxième détermination faisant suite à la première après quelques jours a montré des valeurs plus élevées, largement au-dessus de 43 $U \cdot l^{-1}$.

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