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Chronic HCV treatment with peginterferon-ribavirin and severe tuberculosis re-activation

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Background

Tuberculosis (T) may be reactivated following a primary, silent, and unknown T infection, when immunodeficiency (often jatrogenic in origin), or other risk factors (e.g. cancer, cachexia), become apparent. Post-primary T episodes were described also decades after a primary *M. tuberculosis* infection, in patients (p) who show apparently limited radiographic signs at chest X-ray. Some grade of immunodeficiency may also depend on the administration of associated IFN-ribavirin for an underlying chronic HCV hepatitis, as expressed by the frequent emerging of leuko-neutropenia, and altered cytokine network.

Methods

In a p aged >50 years with negative history of T, an occasional chest X-ray showed fibrous-calcified infiltrates at upper right lobe. After 11 years, due to a progressive chronic HCV hepatitis, pegylated IFN-ribavirin were started for 7 months, until a sudden occurrence of cough-hemoptisis associated with a pulmonary lesion highly suggestive of T became apparent, in the same area where some reliquates of a primary T were demonstrated 11 years before.

Results

A HRCT examination pointed out 2 different excavated infiltrates. Both direct microscropy and culture of sputum-BAL proved positive for *M. tuberculosis* (susceptible to all tested compounds), while Mantoux-Quantiferon assays also tested positive. An absolute lymnphopenia (nadir 966 cells/μL), prompted a T-cell subset study, which showed an imbalance of the CD4/CD8 ratio (30/45%), and an absolute CD4 count of 290 cells/μL. Notwithstanding 7 consecutive weeks of isoniazide, ethambutol, rifampicin and pyrazinamide administration, sputum examination remained positive, thus confirming the role of immunodeficiency is prompting a difficult-to-treat T. The adjunct of levofloxacin-amikacin-linezolid attained clinical-bacteriological cure, after 12 weeks.

Discussion

Waiting for human experimental data, two animal models demontrated that an increased release of immunosuppressive cytokines (IL-10-TGF-β), may prompt T reactivation, while a maintained T-cell competence enhances T latency. Although a few cases of non-infectious lung involvement, interstitial pneumonia, and bronchiolitis obliterans were described during IFN therapy administered to transplant p, reactivated T was excepional. The expected increase of therapeutic use of IFN and potent agents for the management of chronic hepatitis or other diseases, might support the reactivation of latent T. A careful medical history, Mantoux reaction, IGRA testing, and a chest X-ray, are mandatory before starting IFN therapy. In fact, the jatrogenic immunosuppression related to IFN-ribavirin may go beyond the expected leuko-lymphopenia, and also act against the quantitative-functional role of CD4 lymphocytes. This last circumstance may play a key role in T reactivation, when T latency is of concern.

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