Case description: A 39-year-old Ugandan man was diagnosed HIV-1 positive in 1996 (baseline CD4 count 130/uL, viral load (VL) 235,678 copies/ml), and was commenced on Zidovudine, Lamivudine and Nevirapine therapy. Ten years later the patient presented with sharp, right-sided chest pain. He did not describe any symptoms suggesting infection, nor relating to eyes, skin or joints. Auscultation of the chest was unremarkable. He had tenderness on palpation over the right anterior chest wall. Chest radiograph revealed bilateral hilar lymphadenopathy (BHL). Blood tests showed CD4 count 385 with an undetectable viral load. Full blood count, renal, liver and bone profile were unremarkable. Serum angiotensin-converting enzyme was raised at 119 U/ml (normal range <67 U/ml).

Computed tomography of the chest confirmed extensive mediastinal lymphadenopathy, with bilateral hilar, pretracheal, right para-tracheal, pre-aortic and subcarinal nodes involved. The differential diagnoses included tuberculosis and lymphoma. The patient underwent left anterior mediastinoscopy and mediastinal lymph node biopsy. Microbiology cultures of the biopsy were negative and no acid-fast bacilli were seen. Histopathology showed the normal lymph node architecture was effaced by confluent non-necrotising granulomas, with lymphocytes interspersed between the granulomas. There were no micro-organisms on special stains and no evidence of malignancy. The findings were consistent with sarcoidosis. As this patient had BHL only, he did not require steroid treatment, and has remained well.

**Apoptosis of CD8+ T-cells in HIV-1-infected typical progressors, but not in long-term non-progressors**

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CD4+CD25+ Regulatory T-cells (Tregs) have been demonstrated to down-regulate immune activation in HIV-1 infection. However, persistent HIV infection results in a decrease of Treg absolute counts. Whether the decreased Treg also play an important role in the regulation of excessive activation and apoptosis of CD8+ T-cells in HIV-1-infected patients remains undefined. To address this issue in the cross-sectional study, we characterized Treg among 83 HIV-1 infected individuals, including 19 long-term non-progressors (LTNPs), 51 typical progressors (TPs) who were treatment naive, and 13 HAART treated AIDS patients, 9 of whom produced complete responses (CRs) to antiviral therapy and 4 of whom were non-responders (NRs) to the treatment. TP but not LTNPs had significantly decreased absolute counts of circulating Tregs, which inversely correlated with up-regulated activation of CD8+ T-cells. Isolated Treg could significantly inhibit the spontaneous and anti-CD3-induced apoptosis of total and peptide-stimulated HIV-specific CD8+ T-cells in vitro. More importantly, CR patients to antiviral treatment exhibited an increase in circulating total CD4+ T-cells and Treg counts that were associated with reduced activation and apoptosis of CD8+ T-cells compared with NR patients. Thus, our findings indicate that decreases in Treg correlate with disease progression, and decreases in Treg in CR patients efficiently blocked excessive activation and apoptosis of CD8+ T-cells.

**Specific T-cell responses to CFP-10 antigens of Mycobacterium tuberculosis in Chinese HIV positive individuals**

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Tuberculosis (TB) is still a major public health problem around world, especially in China, which is the second largest TB patient population in the world. AIDS epidemic have make TB infection more complex than that before, it is especially very difficult to make a diagnosis when an AIDS patient combined with extra-pulmonary TB diseases. Traditional methods for diagnosis of TB infection such as TSTs are not sufficient for confirmation of TB infection in AIDS patients. Here, we use use the recombinant CFP-10 protein as stimulus to detect TB specific T-cell responses in Chinese HIV (+) patients.

**Methods:** CFP-10 was cloned into prokaryotic expression vector pET-32a (+) and transfected into E. coli BL21(DE3) to produce the recombinant CFP-10 protein, and use CFP-10 protein as stimulus to detect specific T-cell responses in HIV(+); persons with or without clinical manifestation of TB diseases.