Abstract

Background: Diffuse infiltrative lymphocytosis syndrome (DILS) is characterised by a persistent CD8+ lymphocytosis and lymphocytic infiltration of various organs. The exact prevalence isn’t known but some studies have reported between 0.85 – 3%, and appears to be more common in African population. Patients with DILS tend to have higher CD4 cell counts and survive longer than those patients without DILS. Most patients present with bilateral parotid gland enlargement and features of the Sicca syndrome. Common sites of extra glandular involvement are the lungs being the most common site, followed by peripheral neuropathy and liver. With the high incidence of HIV in our population it is likely that DILS is under diagnosed probably due to our ignorance of this disease. Awareness of its various presentations may bring to light undiscovered patients with DILS. Objectives: To review pathogenesis, diagnostic approach and current trends in the management of Diffuse interstitial lymphocytic syndrome.

Data source: Literature review of relevant published literature from both Africa and the rest of the world.

Data synthesis: Pathologically, under light microscopy, DILS resembles the focal sialadenitis seen with Sjogren’s syndrome, although it tends to be less destructive of the glandular architecture than in Sjogren’s syndrome. Most of the inflammatory infiltrate is composed of CD8+ lymphocytes unlike Sjogren’s which are CD4+. Lymphoepithelial cysts are frequently observed in the parotid glands of patients with DILS. The variation in CD8 count in the course of HIV disease is less understood. The variation in CD8 lymphocytes is implicated in the pathogenesis of a number of clinical manifestations in HIV diseases including diffuse infiltrative lymphocytic syndrome (DILS) and HIV associated CD8+ lymphocytosis syndrome. Parotid gland enlargement in a patient with HIV infection should prompt clinicians to suspect DILS. In addition, clinicians should be aware that the pulmonary process associated with DILS may mimic clinically and radiographically the pneumonic process caused by Pneumocystis carinii. Other manifestations of DILS to consider include a severe form of peripheral neuropathy; lymphocytic infiltration of the liver, evident as hepatitis; myositis; and lymphocytic interstitial nephritis. Management of DILS is determined by the severity of glandular and extra glandular features. Data on therapeutic trials are lacking although there are isolated reports of good response to antiretroviral and steroid therapy. Conclusion: DILS, a subset of HIV disease manifestation, may present as parotid gland swellings. In general, an HIV patient presenting with DILS has a better prognosis than a patient with HIV alone. With the high incidence of HIV in our population it is likely that DILS is under diagnosed probably due to our ignorance of this disease. Awareness of its various presentations may bring to light undiscovered patients with DILS. Clinicians should watch for the possible transformation into B-cell lymphoma. There is still paucity of data about this disease from pathophysiology to treatment to studies correlating the plasma viral load with CD8 lymphocyte count in patients with HIV disease.