

Case Report

Thyroid cancer in a long-term nonprogressor HIV-1 infection

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Abstract

Long-term non-progressor HIV infection (LTNP-HIV) is seen in <1 percent of HIV-afflicted population. There are definite criteria for the diagnosis of LTNP-HIV. Malignancies either solid tumors or haematological cancers have not been reported in such population. We report here a rare case of follicular thyroid carcinoma in LTNP-HIV infection. She never had any opportunistic infections. She did not receive anti-retroviral therapy in the entire course of illness and continued to have good quality of life. Treatment of follicular thyroid cancer was similar to other patients without HIV infection. This could be the first case study from India.

Key words: Follicular thyroid cancer, HIV-1, long-term nonprogressor HIV infection

INTRODUCTION

Long-term nonprogressor HIV infection (LTNP-HIV) is seen in <1% HIV positive population. Natural history of this subset of patients is entirely different.^[1] So far, there are no studies on cancers in LTNP-HIV patients in the literature. We report here a very rare case of follicular thyroid carcinoma in LTNP-HIV infection. This could be the first case report from India.

CASE REPORT

A 30-year-old female, doctor by profession, presented with midline painless swelling in the neck for 3 months. It was slowly progressive but did not cause any pressure effects on nearby structures. She was having HIV-1 infection for last 10 years. She never had fever, weight loss, or any opportunistic infections due to HIV-1 infection. Details of investigations done 10 years ago such as HIV-1 viral load and CD4 and CD8 counts are not available at

present. She was never treated with prophylactic drug treatment for opportunistic infections or with antiretroviral therapy during this period. She never suffered from thyroid illness before. None of her family members had history of thyroid dysfunction.

Clinical examination revealed a solitary nodule of 4 cm in the left lobe of thyroid. Cervical lymphadenopathy was not found. Ultrasonic study of thyroid gland showed an isoechoic solid nodule. Fine-needle aspiration cytology reported as a cellular follicular lesion. Thyroid function tests were normal. She underwent left hemithyroidectomy. Histopathological gross evaluation of left hemithyroidectomy specimen measuring 6.3 × 6.3 × 3.0 cm showed a well-circumscribed homogenous, nodular brownish mass measuring 4.5 × 3.5 × 2.8 cm. Adjacent nonneoplastic thyroid was nodular grey-white. Microscopy revealed a widely invasive, follicular carcinoma demonstrating

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10.4103/0253-7184.167175

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How to cite this article: Phatak UA, Chitale PV, Jagdale RV. Thyroid cancer in a long-term nonprogressor HIV-1 infection. Indian J Sex Transm Dis 2015;36:195-7.

prominent capsular and vascular invasion with tumor plug completely transgressing the fibrous capsule and present within a blood vessel covered by endothelium. No extra-thyroid extension was seen. Adjacent thyroid parenchyma shows lymphocytic thyroiditis [Figure 1]. There was no spread to other organs. Stage I follicular carcinoma was diagnosed.

Completion thyroidectomy was performed. Histopathological evaluation of completion thyroidectomy specimen showed Hashimoto's thyroiditis. Eleven adjacent lymph nodes were free of tumor. After 4 weeks, she underwent radioactive-iodine whole body scan that demonstrated residual disease in the neck. Radioiodine ablation of the disease was done. She was treated with levothyroxine 100 µg daily for hypothyroidism after radioiodine treatment and calcium carbonate 1 Gm 3 times a day along with weekly cholecalciferol 60,000 IU for immediately for postoperative hypoparathyroidism. Her CD4 and CD8 counts were 756 cells/mm³ and 819 cells/mm³, respectively and the viral load was 136 copies/ml. Diagnosis of LTNP-HIV infection was considered as per the current criteria of LTNP-HIV.

DISCUSSION

HIV-1 infection is common viral infection in India. There are subsets of HIV-1 infection in which the viral load is not very high, T cell subpopulations (helper/suppressor) cells are slightly reduced and patients can survive more than 8 years in the absence of antiretroviral therapy for HIV-1 infection. This subset is seen in only <1% of HIV-positive population. We considered LTNP-HIV infection rather than elite controller as the viral load was more than 100 HIV-RNA copies/ml and helper

T cell count was stable over the past 10 years, in the absence of antiretroviral therapy in this case study. The diagnostic criteria of LTNP-HIV include: (i) Helper cell population (CD4 cells) more than 500 cells/mm³ (ii) viral load <1000 copies/ml (iii) stable disease over a period of 8 years without antiretroviral therapy for HIV infection. Prevalence of LTNP is <1% of HIV-positive patients in clinical practice.^[1] Most of the patients are asymptomatic.

Incidence of cancers either AIDS-defining cancers (ADCs) or non-AIDS-defining cancers (NADCs) in LTNP-HIV infection has not been reported earlier. Prevalence of cervical lesions in LTNP-HIV patients was studied in Africa.^[2] Thyroid involvement in HIV-positive patients may have variety of causes. It may be involved due to infections or there could be drug-related thyroid dysfunction in HIV infection,^[3] but primary malignancy of thyroid in LTNP-HIV-1 patients is not known. Etiology of the NADCs and ADCs is not well understood. Most of the patients with AIDS-associated cancers have viral etiology. Human papillomavirus is responsible for oral and cervical cancers, Epstein-Barr virus is related to non-Hodgkin's lymphoma (NHL) and human herpes virus 8 for Kaposi's sarcoma (KS). No such viral etiology is attributed in the pathogenesis of thyroid malignancy.^[4]

HIV/AIDS-related cancers, either AIDS-defining malignancies (ADMs) or non-ADMs (NADMs) are often seen in HIV infection with advanced stage. With highly active antiretroviral therapy, the prevalence of KS and NHL has declined significantly. Thyroid cancers in HIV/AIDS are an uncommon and unusual type of NADM.^[5] Mbulaiteye *et al.* reported rising incidence of cancers of thyroid, kidney, and uterus and of conjunctiva in HIV/AIDS in Africa.^[6] Whether genetic factor(s) play any role in the pathogenesis of thyroid cancers in HIV-positive patients is not clear.^[4] Papillary thyroid carcinoma^[7] and medullary thyroid carcinoma^[8] were reported in advanced HIV positive patients. They were receiving antiretroviral therapy for HIV infection unlike our patient.

Pathogenesis of LTNP-HIV infection is a mystery. Viral, genetic and host-related factors have been postulated in the development of LTNP-HIV infection. Patients with HIV-1 infection progress if they have abnormalities of nef gene or have high level of beta-2-microglobulin. While some genes protect against the progression,^[1] CCR5 is a co-receptor for transmission of HIV-1 infection. Mutation of CCR5 gene is the most common



Figure 1: Widely invasive follicular carcinoma of thyroid. Arrow shows capsular invasion. Inset shows adjacent Hashimoto's thyroiditis

abnormality in LTNP-HIV. Such mutation can be seen in Indian families as well.^[9] Usually, individuals with homozygous delta 32 allele are resistant to HIV infection in spite of multiple exposures to HIV-infected persons while those with heterozygous delta 32 mutation have lesser viral replication and slower progression of HIV infection.^[10] We have not evaluated our patient for molecular markers. Until date, patient has got good quality life following total thyroidectomy. How long will she remain LTNP-HIV or will she progress in future is not known.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

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