

Letters

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Infiltrating Lobular Breast Carcinoma in a Woman with HIV Infection

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THE SURVIVAL of HIV patients has increased over recent years and so, although not strictly associated with HIV, there is likely to be an increased incidence of breast cancer in the HIV setting [1]. Cuvier and associates have recently suggested that breast cancer in HIV-infected patients may have atypical characteristics, such as an earlier onset, marked aggressiveness and an unusual behaviour [2]. We here report the case of an infiltrating lobular breast tumour in a female HIV-seropositive patient (heterosexual transmission). We describe its biopathological features and indicate its related clinical implications.

The patient was diagnosed as being HIV-seropositive in 1988 when she was 24 years of age, but has so far remained asymptomatic. In October 1995, she presented with a bifocal right breast neoplasm. She has no family history of breast cancer. A radical mastectomy was performed and histological examination showed the presence of a double T1c and T1b infiltrating lobular carcinoma with all 25 examined nodes being negative. Both tumours were positive for oestrogen and progesterone receptors, and Ki67 and S-phase fraction indicated a low proliferative rate. Their DNA content was diploid and p53 expression negative. The results of chest radiography, bone scan and liver ultrasound were negative. The CD4 counts before and after surgical treatment were $582 \times 10^3/L$ and $573 \times 10^3/L$, respectively. No adjuvant oncological therapy was administered. In January 1997, she developed a bifocal left breast tumour with extensive neoplastic infiltration in the residual parenchyma and massive axillary and supraclavicular metastases. Histological examination revealed an infiltrating lobular carcinoma with all positive nodes out of 28 classified as T3N1bivM1. The biological characterisation was similar to that of the previous tumours. The CD4 count before surgical treatment was $550 \times 10^3/L$.

Chemotherapy and hormonotherapy was refused by the patient. In June 1997, pleural and skin metastases occurred. At this time, no antiviral therapy was given because the CD4 count had increased to $620 \times 10^3/L$. The patient is now receiving supportive care.

To our knowledge, lobular infiltrating breast carcinoma in HIV-seropositive patients has not been previously reported. In this case, the relapse observed after the first diagnosis occurred despite good clinical and biological prognostic factors, and when the patient's CD4 count was more than $500/mm^3$. Neither biological characterisation nor the CD4 count predicted the progression of the disease. Unlike the unfavourable prognostic factor described in the case of invasive mixed colloid breast carcinoma, which the authors considered to be related probably to immunodeficiency [3], our data may support a previous observation of the lack of any relationship between the natural history of a case of infiltrating ductal adenocarcinoma and HIV infection [4]. Nevertheless, as in a previously reported case in which favourable prognostic markers were not sufficient to avoid a poor clinical outcome [5], our results may also support the hypothesis that HIV infection may have a permissive effect on breast cancer development regardless of tumour biology and CD4 lymphocyte count.

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