
Letter to the Editor

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The role of HPV2 infection in the occurrence of cervical neoplasia is clearly established worldwide (1). Numerous studies have stressed the association between cervical precancerous lesions (SILs) and HIV infection (2), which led to the introduction of ICC in the surveillance case definition of AIDS in 1993. Very few studies have, in fact, shown an association between the invasive stage of cervical carcinoma and HIV infection (3). The commonly accepted relationship between HIV and HPV infections (4) raises the question of the respective contribution of these two viruses in the development of cervical neoplasia. In other words, the independent role of HIV infection in the development of cervical neoplasia is questionable.

Recently, Mandelblatt et al. (5) reported a meta-analysis on the interrelation between cervical neoplasia and HIV and HPV infections, showing strong associations between HPV and neoplasia and between HIV and neoplasia and pointing out an interaction between HPV and HIV infections. They conclude that HIV seems to be a cofactor in the association between HPV and cervical neoplasia. We would like to summarize and share our experience in this field from a study conducted in African women with a high HIV prevalence, too recent to be included in the meta-analysis.

A total of 2170 women underwent a cervical screening in three outpatient gynecology clinics in Abidjan, Côte d’Ivoire (6). The prevalence of HIV infection was 22%. The prevalence of low-grade SILs, high-grade SILs, and ICCs was 7.6% (n = 165), 3.3% (n = 71), and 0.8% (n = 18), respectively. Low-grade and high-grade SILs were associated with HIV-1 infection. HIV-1-infected women with SILs were not deeply immunosuppressed, with a mean CD4 count of 460/μl in women with low-grade SILs and 530/μl in women with high-grade SILs, suggesting that SILs occurred at an early stage of HIV disease. ICCs were associated with HIV-2 infection, but not with HIV-1 infection, suggesting that cancers in HIV-1-infected women often may not reach the invasive stage. However, attention must be paid to a possible increase in the number of ICCs associated with HIV in the near future, especially in developed countries, because the lengthening of life span associated with the highly active antiretroviral therapies will increase the possibility that high-grade SILs will evolve to ICCs (7).

Genital HPV detection by PCR and HPV typing by RFLP were performed in Abidjan on a subset of women with a case-control design (151 women with low-grade SILs and 151 controls without lesions, 60 women with high-grade SILs and 240 controls; Ref. 8). We found a diversity of HPV types associated with SILs. HIV-1 seropositivity and HPV positivity were both independently associated with low-grade and high-grade SILs. However, adjusted odds ratio of HIV-1 infection for low-grade and high-grade SILs were markedly lower than unadjusted ones when HIV infection status was taken into account. Conversely, adjusted odds ratios were two times (with low-grade SILs) to six times (with high-grade SILs) greater for HPV infection than for HIV-1 infection, adjusted on this latter factor. The estimated population attributable risks of HPV infection were 51% for low-grade SILs and 73% for high-grade SILs in Abidjan, much higher than the attributable risks of HIV-1 infection, which were around 18% for both types of lesions. These findings suggest that the observed relation between cervical precancerous lesions and HIV-1 infection is mainly attributable to HPV infection. Of note, women infected with both HIV-1 and HPV were at much higher risk of SILs than women infected with each of these two viruses separately, in agreement with the study by Mandelblatt et al. (5).

Finally, 94 women with low-grade SILs were followed in Abidjan during an average of 5 months to assess the short-term evolution of the lesions (9). HIV-infected women had a higher percentage of persistent/progressive SILs (76%) than HIV-negative women (18%; relative risk = 4.3, 95% confidence interval 2.4–7.7). Furthermore, development of high-grade SILs was observed in 7 of the 38 HIV-positive women, but in none of the 56 HIV-negative women (P = 0.001). HIV-infected women with persistent lesions were not significantly more immunosuppressed than HIV-infected women with regression of SIL. Persistence of SILs was also more frequent among women infected with HIV at the time of enrollment or with persistent HPV infection, but these associations disappeared after adjusting for HIV serostatus. Women infected with both HIV and HPV (HPV infection at enrollment or persistent HPV infection) were at a higher risk for persistent SILs than women infected with each of these two viruses separately.

From a scientific point of view, it seems that HPV infection plays a central role in the occurrence of cervical SILs, although other factors are involved in the progression/regression of the lesions, particularly HIV infection. From a public health point of view, these results could have the following implications. First, the relation between SILs and HIV infection...
and the role of HIV on the progression of the cervical lesions suggest that HIV screening should routinely be offered to women with detected cervical SILs. Second, taking into account the likely regression of most of the low-grade SILs in HIV-negative women, the follow-up of these women may not be necessary in areas with scarce resources. Third, HIV-infected women with cervical SILs require specific gynecology follow-up.

References


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