Letter to the Editor

Anal condylomas: predictors of recurrence and progression to high-grade dysplasia/carcinoma in situ

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Submitted Jul 24, 2017. Accepted for publication Sep 20, 2017.
doi: 10.21037/jgo.2017.09.09
View this article at: http://dx.doi.org/10.21037/jgo.2017.09.09

Condyloma acuminatum is caused by human papilloma virus (HPV) infection. Individuals with condylomata acuminata are at an increased risk for anogenital cancers, mainly anal condylomas with dysplasia are precursors of anal squamous cell carcinoma. There are over 70 distinct HPV subtypes; approximately 35 types are specific for the anogenital epithelium and have varying potentials to cause malignant change, such as cervical or anal cancer (1). Despite ninety percent of anogenital condylomas harbor HPV types 6 or 11 (low-risk subtypes), HPV serotypes 16 and 18 are most commonly associated with squamous cell carcinoma, being considered high-risk subtypes. HPV types 16, 18, 31, 33, and 35 can be associated with a focus of high-grade squamous intraepithelial neoplasia, especially in immunocompromised individuals, such as those with human immunodeficiency virus (HIV) infection (2). Intermediate risk subtypes can cause high-grade dysplasia (HGD), which persists but rarely progresses to the invasive stage (3). However, there are few studies concerning predictors of recurrence and progression to high grade lesions [HGD and carcinoma in situ (CIS)].

To better understand this issue, we decided to evaluate clinical characteristics and predictors of recurrence and progression to HGD/CIS in patients with anal condylomas. To do so we conducted a retrospective analysis of all biopsies and subsequent excision of anal condylomas performed in proctology consultation between 2011 and 2015, in a tertiary referral center. During this period of time, 152 biopsies to anal condylomas were performed in 82 patients, most of them male in 80%. The mean age was 39±11 years and the mean follow-up time was 9±12 months. As expected, HIV infection was present in the majority of patients (76%), 95% in asymptomatic phase, and 82% under anti-retroviral therapy. Previous history of anal condylomas was reported in 61%, 39% of whom with previous dysplasia, including HGD in 65%. The most frequent HPV genotypes were 16 (49%), and HPV-18 (27%). Sixty-six percent of condylomata samples obtained by biopsy or excisions showed some degree of dysplasia (33% with HGD, and 2% with CIS). Local treatments applied to the identified condylomas included argon plasma coagulation in 83%, and excision with scalpel in 12%. No immediate or late complications were reported independently of the therapeutic approach. The recurrence rate after apparent successful treatment was 64%, with presence of HGD/CIS on histology bring the sole predictor factor. Looking to predictors of high-grade lesions, we identified associations with shorter time to recurrence (P=0.003); HPV-16 positive condylomas (69% *vs.* 31%, P<0.001) and HGD/CIS in previous lesions (64% *vs.* 36%, P=0.035).

In our large cohort of patients followed by anal condylomas, we found high incidence of anal condylomas with HGD, particularly in patients without HIV infection. The large numbers of these lesions, and in particular the detection of two cases of CIS, reflect the need for regular monitoring of patients with HPV infection. The predictors identified factors can aid in the stratification of that surveillance.

Acknowledgements

None.

Footnote

Conflicts of Interest: The authors have no conflicts of interest to declare.

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Cite this article as: Peixoto A, Silva M, Castro R, Coelho R, Sarmento A, Macedo G. Anal condylomas: predictors of recurrence and progression to high-grade dysplasia/carcinoma in situ. J Gastrointest Oncol 2017;8(6):1114-1115. doi: 10.21037/jgo.2017.09.09

