Pregnancy with HPV associated Viral Warts

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Introduction

Human papilloma viruses (HPV) are members of the Papillomaviridae family of DNA viruses. Out of more than 100 types, over 30 infect the genital area. These genital types are generally characterized as “high-risk” types which are associated with high grade squamous intraepithelial lesions and invasive cancer while “low-risk” types cause genital warts, low grade squamous intraepithelial lesions and recurrent respiratory papillomatosis [1]. Most infections are subclinical, while few cause genital warts. Treatments are directed to abnormal cells associated with HPV rather than the virus itself, since there is no curative treatment for HPV infection.

The risk factors consistently associated with HPV infection in women are early age of first sexual intercourse and male partner’s promiscuity [2]. Less consistently identified risk factors include smoking, oral contraceptive use, nutritional factors and lack of circumcision of male partner. In addition, immune suppression is associated with HPV detection [3].

We present an interesting case of a young primigravida presenting with a vaginal warty growth.

Case Report

A 23-year-old primigravida, at 14 weeks of gestation reported with intensely pruritic, cauliflower like growth over external genitalia, labia majora, extending upto labia minora and vaginal orifice. There was no bleeding, ulceration, depigmentation or discharge from the growth. There was no history of trauma, drug intake, diabetes mellitus, oral ulcers, urinary complaints or bleeding or discharge per vaginum. There was no history of similar lesion in spouse. She did not give history of sexual encounter outside matrimony, husband’s history could not be elicited. First sexual contact was at 22 years of age. Her last menstrual period was on 04 April 2007, with expected date of confinement on 11/01/2008.

General and systemic examination were unremarkable.

Obstetric examination revealed a gravid uterus of 14 weeks size. External genitalia showed soft, friable papillomatous growth with ill-defined margins over both labia majora extending upto labia minora and vaginal orifice. Oral and nasal mucosa, eyes, nails, palms, soles, external genitalia and perianal region were normal. Per speculum examination showed normal healthy cervix and vaginal walls.

Routine biochemical and haematological investigations were normal. HPV IgM and IgG titers were found to be positive. She was non-reactor for VDRL. TPHA and HIV tests were negative. Cervical PAP smear was within normal limits. On histopathology, tissue showed hyperplastic epithelium in papillary configuration with koilocytic changes and minimal atypia suggestive of viral warts due to HPV infection (Fig.1).

Discussion

The most common approach to reducing infectiousness of sexually transmitted disease (STD) is treatment. In contrast to bacterial STD, for which transmission can be prevented through curative treatment, there is only limited evidence that treatment of HPV-associated lesions is useful in preventing HPV transmission. Treatment of genital warts and cervical cancer precursors might reduce infectiousness [4]. However, clinically normal skin and mucosa near HPV-

Fig. 1 : Histopathology showing hyperplastic epithelium in papillary configuration with koilocytic changes

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associated lesions often contain HPV. This reservoir is thought to explain the typical recurrence rates of 10–20% after treatment of cervical lesions and 20–50% after treatment of genital warts [5].

Genital warts are usually flat, papular or pedunculated growths on the genital mucosa and are usually asymptomatic, but depending on the size and anatomic location, genital warts can be painful, friable or pruritic. Diagnosis of genital warts is made by visual inspection and may be confirmed by biopsy. No data support the use of HPV nucleic acid tests in the routine diagnosis or management of visible genital warts. The application of 3–5% acetic acid usually turns HPV-infected genital mucosal tissue to a whitish colour.

HPV types 16, 18, 31, 33, and 35 are found occasionally in visible genital warts and have been associated with external genital (vulvar, penile, and anal) squamous intraepithelial neoplasia (squamous cell carcinoma in situ, bowenoid papulosis, Erythroplasia of Queyrat, or Bowen's disease). Patients who have visible genital warts are frequently infected simultaneously with multiple HPV types [6].

These warts respond to various treatment modalities. Factors that might influence selection of treatment include wart size, number, anatomic site, morphology, patient preference, cost, convenience, adverse effects, and provider experience. Factors that might affect response to therapy include the presence of immunosuppression and compliance with therapy [7]. The majority of patients require a course of therapy rather than a single treatment. Because of uncertainty regarding the effect of treatment on future transmission of HPV and the possibility of spontaneous resolution, an acceptable alternative for some persons is to forego treatment and wait for spontaneous resolution. HPV induced lesions tend to get worse during pregnancy due to natural immune suppressive state. Warts may

increase dramatically in size so as to obstruct urinary and reproductive passage. Also, a possibility of infecting the fetus exists at the time of labor. Hence caesarian section in such cases is the method of choice.

This case has been presented to increase awareness of the spectrum of HPV infections, which can range from asymptomatic infection to cancer. Due to erratic therapeutic response, the importance of prophylaxis needs to be stressed upon. All sexually active women should undergo PAP smear for early detection of carcinoma cervix which is also caused by HPV.

Conflicts of Interest

None identified

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