

Tri-chloroacetic Acid: A Cause of Vulvar Vestibulitis

Sir,

Vulvar vestibulitis is an unexplained inflammatory condition of the vestibule, characterised by burning, stinging and dyspareunia (1). Although a clinical diagnosis can easily be made, the aetiology of the condition remains elusive and management of patients is difficult. We here report 2 cases of severe vulvar vestibulitis following treatment with tri-chloroacetic acid (TCA), a standard treatment for genital warts.

CASE REPORTS

Case 1

A 32-year-old woman presented with a recurrent attack of genital warts. She was otherwise asymptomatic and a full sexually transmitted disease screen was negative. Six months prior to this presentation, she had had her first attack of genital warts, which had been successfully treated with cryotherapy. Her cervical smear was normal. On examination, she had multiple vulval and vestibular warts which were treated with TCA. The surface of each wart was coated with TCA and the area was then covered with talcum powder to prevent TCA from spreading to the normal skin. Standard simple vulval hygiene measures were given to the patient whilst on treatment, including the use of only water to the area and the avoidance of soaps and bubble baths. Following TCA application, she complained of burning which was initially short-lived. Following the second treatment a week later, the burning became prolonged and distressing. On clinical examination a week later on review, there was marked erythema and tenderness of the vulvar vestibule, mostly at the posterior fourchette. A single residual wart was left untreated and there were no areas of ulceration or of tissue desquamation. A high vaginal swab and wet mount film of vaginal discharge was normal.

She had an unsuccessful trial of a variety of topical treatments, including aqueous cream and 1% hydrocortisone. These all failed to resolve her symptoms and the burning persisted for 15 weeks. She was eventually treated successfully with systemic interferon-alpha 3MU subcutaneously for 15 days and currently remains symptom-free 4 months after the onset of symptoms.

Case 2

An 18-year-old woman presented with a first attack of genital warts. She was otherwise asymptomatic and a full sexually transmitted disease screen was normal, including cervical smear. On examination, multiple warts were present on the lower vulvar and vestibule area. She was initially treated with topical TCA using the same technique as in case 1. This caused burning and stinging on application, which soon settled. On her second review a week later, TCA was reapplied to the residual warts; however, this left her with continual soreness confined to the vestibule area. On examination a week later, there was tenderness and erythema of the vestibule area, with small areas of skin fissuring at the posterior fourchette. A high vaginal swab and wet mount examination of vaginal discharge was normal. She was initially started on topical zinc cream for 2 weeks, which did not alleviate symptoms. Her symptoms eventually settled, however, with a regime of topical 1% hydrocortisone cream and strict vulval

hygiene including the complete avoidance of soaps, perfumes and shampoos. She is now currently asymptomatic 7 weeks following the onset of her symptoms.

Many factors have been implicated in the development of vulvar vestibulitis. The human papilloma virus has been implicated as the cause of inflammation; however, recent evidence suggests that this link is coincidental (2). Some authors believe that there is an acute insult to the skin, commonly candidal vulvovaginitis which, once treated with anti-fungals, persists as vulvar vestibulitis (3). Many of these aetiologies, however, are speculative and there is still no explanation for the diffuse, chronic inflammatory changes that are found solely in the vestibule area (4). Anatomical variations exist in the response of skin to topical agents, and this can in part be explained by the physiology of the skin involved. When compared to forearm skin, vulval skin has significantly higher transepidermal water loss and percutaneous absorption, suggesting that the stratum corneum of the vulval skin functions as a less efficient barrier (5). Vestibule skin is even thinner, as it is devoid of the keratinized surface present on labial skin, and this may play a role in its response to irritants and allergens.

TCA is an effective, widely used treatment for genital warts. Even when applied conservatively, it is highly traumatic and destructive to skin. When used in excess, pain, ulceration and secondary infection may occur. Many patients complain of subjective burning when TCA is applied; however, this is often short-lived, associated with acute irritation and inflammation. In these 2 patients, diffuse vestibular inflammation was clinically apparent several weeks after treatment despite the initial correct use of TCA, limited to the warts only. Physicians should be aware of this potential complication of treatment, and we conclude that TCA should not be used in patients who have a current or past history of vulvar vestibulitis. Other methods of treatment should be considered in such patients.

REFERENCES

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