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MULTIPLE AETIOLOGIC AGENTS CAUSING PENILE ULCERS IN AN HIV-ANTIBODY POSITIVE PATIENT

K D'Souza, UM Tendolkar, LP Deodhar

Multiple pathogens were found to cause penile ulcers in a patient infected with HIV, who failed to respond normally to treatment.

Key Words : Penile ulcers, Human immunodeficiency virus

Introduction

Sexualy acquired penile ulceration is a very common finding in our STD clinics. The agents responsible are *Haemophilus ducreyi, Treponema pallidum, Calymmatobacterium granulomatis, Herpes simplex virus, Chlamydia trachomatis (LGV strain).*

We report an HIV-positive patient with penile ulcers. The ulcers were morphologically suggestive of chancroid whereas bacteriologically they were shown to have been caused by multiple aetiologic agents and failed to respond to treatment.

Case Report

A 19-year-old married Nepalese man presented with painful penile ulcers and accompanying inguinal lymphadenopathy. The patient gave a history of contact with a commercial sex worker 1 month back following which he developed the ulcers. He denied multiple exposures. A right inguinal swelling developed 6-7 days after the appearance of the ulcers. The patient was not addicted to intravenous drugs nor had he received any blood transfusion. He complained of weight loss, persistent cough without expectoration, and occasional burning micturation. Prior treatment with 4 courses of injection Penidura was reported but with no improvement.

The patient did not reveal any abnormality except for the 2 ulcers, the larger one on the prepuce and the other on the urinary meatus. The ulcer was tender,non-indurated, painful, foul-smelling, having well- defined margin with a sloping edge. The surface was rough with yellowish slough and surrounded by an erythematous halo. Right inguinal lymph nodes were tender, non-fluctuant, and firm with erythema overlying the skin. Clinical presentation was suggestive of chancroid with inguinal bubo. The patient was put on cotrimoxazole (trimethoprim 80mg, sulphamethoxazole 400 mg) 2 tablets, twice daily for a week and injection streptomycin.

Direct gram-staining of ulcer material before starting the treatment revealed gram-negative coccobacilli (intra and extracellular), gram-negative diplococci (intra and extracellular), gram - positive cocci and clue cells. Dark-field examination was positive for *Treponema pallidum.* Donovan-bodies were seen in crushed granulation tissue stained by giemsa stain. Giemsa stained smears revealed

From the Department of Microbiology, L T M Medical College, Sion, Bombay - 400 022, India.

Adress correspondence to : Dr U M Tendolkar

multinucleated giant cells, chlamydial inclusion bodies, cytomegalovirus inclusion, and spirochaetes as well. 5ml patient's blood was drawn aseptically for culture (*H.ducreyi*) and serology (HIV by ELISA method and VDRL test). Swab-scrapings of ulcer material was inoculated on Mueller-Hinton agar supplemented with L-glutamine, Hemin, defibrinated rabbit's blood, vancomycin (MHBA) for H.ducreyi,[1] Thayer-Martin medium, ureaplasma broth, blood agar, and chocolate agar. Both clotted blood and MHBA were positive for *H.ducreyi*. *N.gonorrhoeae* and *Ureaplasma urealyticum* were also recovered.

The patient was positive for HIV antibody by ELISA and VDRL test was non-reactive. Six days later the patient presented again with a ruptured bubo. Normally, cotrimoxazole double strength is recommended and if good personal hygiene is maintained, healing takes place within a week in cases with chancroid. In our case no improvement was noticed with respect to the clinical features, culture, and staining even 10 days after the patient first reported. Culture from bubo was positive for *H.ducreyi* and *N.gonorrhoeae*.

Comments

Multiple aetiologic agents were identified in the ulcers which were otherwise clinically suggestive of chancroid. This justifies that clinical picture is not a reliable tool and should be aided with bacteriological findings for proper evaluation of genital ulcers.[2,3]

Similar to our findings, multiple or coexisting infections (i.e.,not more than 2 pathogens) in ulcers have been reported by other workers[2,4] Presence of numerous aetiologic agents in the ulcer could be explained by the concurrent HIV infection.

Failure to respond to therapy in this case could be due to the coexistence of multiple pathogenic agents and asymptomatic HIV-1 infection. Recently it has been observed that both men and women with chancroid and asymptomatic HIV-1 infection are much more likely to fail treatment. This observation has major implications for treatment protocols in countries in which HIV-1 is prevalent. In Kenya, 30% of men and women with chancroid are HIV-1 seropositive. If both pathogens are present chancroid and HIV-1 act synergistically with increased infectivity, susceptibility and, for *H.ducreyi*, failure to respond to treatment.[5]

References

1. VandenBerghe DA . Selenium and the growth of Haemophilus ducreyi. J Clin Pathol 1987; 40 : 1174.
2. Coovadia YM, Kharsany A, Hoosen A. The microbial aetiology of genital ulcers in black men in Durban, South Africa. Genitourin Med 1985; 61: 266.
3. Sturm AW, Stolting GJ, Cormane RH, Zanen HC. Clinical and microbiological evaluation of 46 episodes of genital ulceration. Genitourin Med 1987; 63: 98.
4. Kinghorn GR, Hafiz S, McEntegart MG. Pathogenic microbial flora of genital ulcers in Sheffield with particular reference to herpes simplex virus and and Haemophilus ducreyi. Br J Vener Dis 1982;58:377.
5. Ronald AR Albritton W.Chancroid and Haemophilus ducreyi. In Sexually Transmitted Diseases (Holmes KK, Mardh P,Sparling PF, Wiesner PJ, eds), 2nd edn.NewYork: Mcgraw-Hill Information Services Co,1990; 268.