International Journal of Infectious Diseases 38 (2015) 153–155

Contents lists available at ScienceDirect

International Journal of Infectious Diseases

journal homepage: www.elsevier.com/locate/ijid

Medical Imagery

Giant Molluscum Contagiosum in an HIV positive patient

ARTICLE INFO

Article history:
Received 4 May 2015
Received in revised form 26 July 2015
Accepted 30 July 2015

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Keywords:
HIV
Molluscum Contagiosum
Immunosuppression
Dermatology

SUMMARY

Molluscum Contagiosum (MC) is a skin infection caused by a double-stranded DNA virus of the family Poxviridae that replicates in the human epidermis, affecting mainly children and young sexually active adults and causing flesh colored papular lesions with central umbilication with an average size of 3-5 mm, although atypical lesions that reach great size (Giant Molluscum Contagiosum), 10-15 mm, can be seen in almost any immunodeficiency condition. We report the case of a 35 year old male patient with C3 HIV disease with an abdominal pathology associated to skin lesions predominantly in the forehead and scalp that reached sizes over 5 mm, diagnosed as Giant Molluscum Contagiosum by skin biopsies.

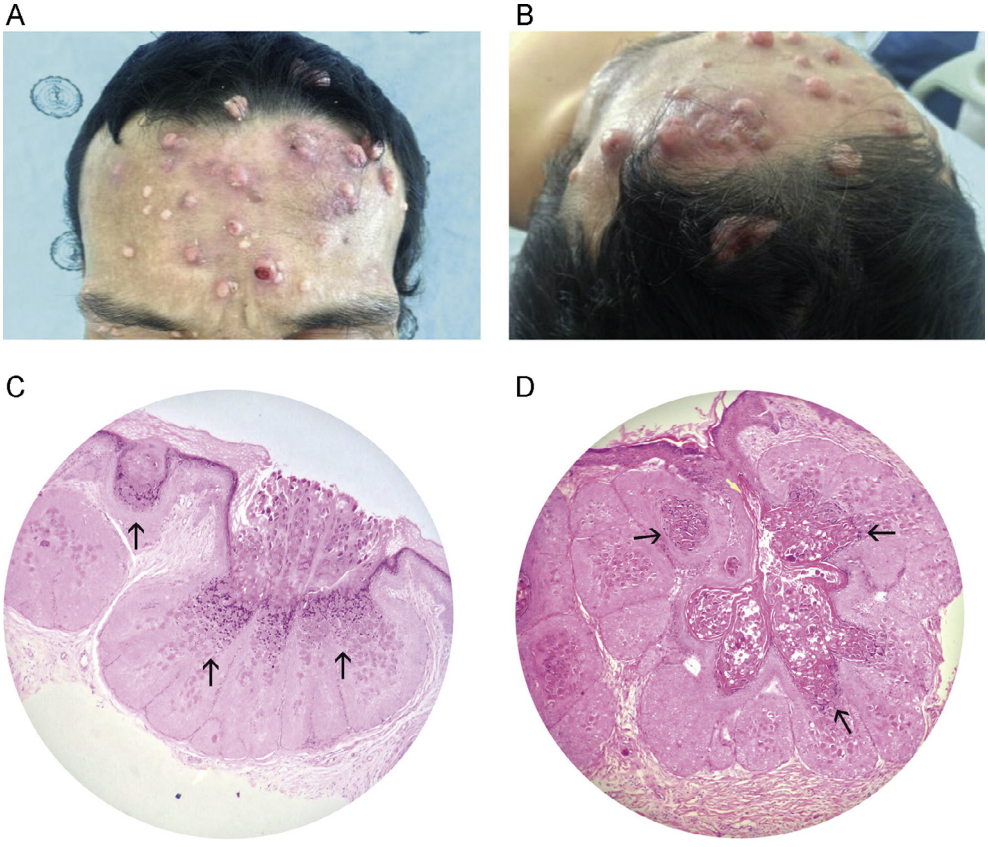

Figure 1. (A) Patient's forehead with several exophytic lesions with irregular edges, some of them with an area of hemorrhage. (B) Scalp of the patient with a predominant lesion in left side. (C and D) Haematoxylin and eosin stain that shows Acanthosis with inverted pyriform lobules of hyperplastic squamous epithelium and a central crater filled with keratin fragments and Henderson-Paterson bodies (Arrows) in infected cells.

http://dx.doi.org/10.1016/j.ijid.2015.07.021

1. Introduction

Molluscum Contagiosum (MC) is a skin infection caused by a double-stranded DNA virus of the family Poxviridae[1] that replicates in the human epidermis enhancing cell mitosis and disrupting epidermal cell differentiation by the upregulation of the expression of epidermal growth factor receptor.[2] There are two different strains of the virus MCV-1 and MCV-2, both universally distributed.[3] Common affected areas are the trunk, armpit and genital area,[4] rarely affecting the palms and soles or mucous membranes;[2] it has two peaks of incidence, one during the first years of life (1 to 5 years of age) and the other one in sexually active young adults[3] with no difference between sexes; vertical transmission has also been reported as congenital disease.[2]

Flesh colored papular lesions with central umbilication appear between two to three months after the initial contact with the virus, with an average size of 3-5 mm, although atypical lesions that reach great size (Giant Molluscum Contagiosum), 10-15 mm,[5] can be seen in almost any immunodeficiency condition, such as corticosteroid and immunosuppressive therapy, leukemia, atopic dermatitis, Wiskott-Aldrich syndrome, sarcoidosis, and AIDS.[1,3] The aforementioned suggests that host cell-mediated immunity plays a crucial role in controlling and eliminating MC virus,[1,3] as MC virus in an uninflamed lesion is invisible to the immune system since it does not cross the basement membrane, causing no systemic response.

A 35-year-old male soldier with human immunodeficiency virus (HIV) since 2001, oropharyngeal candidiasis and Cytomegalovirus ulcers in the colon in 2014, with poor adherence to antiretroviral treatment (Tenofovir + Emtricitabine + Efavirenz) due to adverse effects, presented to the emergency department referred from Manizales, Colombia due to nausea, abdominal pain, distension, and absence of bowel movements for four days. The laboratory investigation showed a normal complete blood count, an absolute CD4 cell count of 89 cells/μL, and an HIV virus load of 1750 copies/mL.

On physical examination, His temperature was 36.5 °C, and blood pressure was 132/85 mm Hg. Pulse was regular with a rate of 103 beats per minute, and respiratory rate was 19 per minute. Abdominal examination revealed abdominal tenderness and rigidity in the central and mid-abdomen. There was no apparent organomegaly. Auscultation revealed a silent abdomen or minimal peristalsis. Patient was hospitalized and taken to surgery for an exploratory laparotomy; intraoperative exploration revealed a peritoneal mass attached to the abdominal wall and a peritoneal biopsy was performed, the further analysis showed a plasmablastic lymphoma.

Additionally, the patient presented skin lesions described as exophytic lesions with irregular edges, some with areas of hemorrhage (Figure 1A and B), predominantly in the forehead, scalp, armpits, groins, and four extremities with no mucosal compromise, with appearance 3 years ago. Therefore, the dermatology department performed a biopsy of the exophytic lesions of the forehead and scalp (Figure 1C and D).

The patient presented Gram negative bacilli and abundant neutrophil cells in peritoneal fluid, and developed abdominal sepsis, for this reason the oncology department contraindicated lymphoma management, but despite antimicrobial treatment the patient required intensive care unit management.

Biopsies of the exophytic lesions showed Henderson-Paterson bodies (the so-called molluscum bodies), that are large, eosinophilic, intracytoplasmic inclusion bodies (Figure 1C and D), confirming the diagnosis. The patient did not receive any treatment for the Giant Molluscum Contagiosum as he unfortunately died of abdominal sepsis; the skin histopathological diagnosis was made afterwards.

2. Discussion

In patients with HIV infection, lesions spread widely and do not show signs of healing,[3] it may signify advanced immunosuppression as it tends to occur during advanced phases of the disease,[4] although it can appear at any stage.[3]

The diagnosis of MC depends on whether the lesions are typical or atypical; in typical lesions just the observation of the umbilicated papules is enough; in atypical lesions histopathology, molecular diagnosis by in-situ DNA hybridization, fluorescent antibody test and PCR can be realized to make the diagnosis.[2,5] Differential diagnosis may be basal cell carcinoma, keratoacanthoma, cutaneous horn, warts, varicella, intradermal nevi, lichen planus, and opportunistic infections as cryptococcosis or histoplasmosis with cutaneous compromise.[3,4]

The treatment in immunocompetent patients is not always recommended as autoresolutive course of lesion in about 2 months, but it must be considered if the untreated lesions carry more potential complications than the side effects of the treatment; it includes curettage, cryotherapy, trichloroacetic acid 100% solution, phenol 10% solution, podophyllotoxin 0.3–0·5% cream, salicylic acid gel 12%, retinoic acid 0.5% cream, potassium hydroxide aqueous solution 5–10% and Imiquimod 5% cream, among others.[2]

In immunocompromised patients MC lesions will not auto resolve, thus different treatment options are described as: cryosurgery,[2] trichloroacetic acid 100% solution,[2] cidofovir cream or I.V., and intralesional interferon alpha injection.[5] Cidofovir is a nucleoside analogue of deoxycytidine that inhibits the DNA polymerase activity of the virus; indicated in giant and recalcitrant lesions in immunocompromised patients, but renal toxicity is the most relevant adverse reaction in systemic use.[2]

Therefore, MC should be considered in any AIDS patient with cutaneous lesions, especially when typical lesions are not presented, and chronicity is patent. Thus requiring directed treatment by dermatological specialist or infectious disease specialist, with special awareness to IRIS, as major concern.

Acknowledgments

The authors thank the Pathology Department of the Hospital Militar Central, whose collaboration with images and reports for the report of this case were fundamental.

Conflict of interest: All authors: No conflict of interest

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