

Title:

Successful evolution of morphea after hepatitis C virus eradication with direct-acting antiviral agent treatment

Authors:

Adriana R. Guerra Romero , Manuel Pérez Figueras , Sonia Alonso López

DOI: 10.17235/reed.2019.6461/2019

Link: [PubMed \(Epub ahead of print\)](#)

Please cite this article as:

Guerra Romero Adriana R. , Pérez Figueras Manuel , Alonso López Sonia . Successful evolution of morphea after hepatitis C virus eradication with direct-acting antiviral agent treatment. Rev Esp Enferm Dig 2019. doi: 10.17235/reed.2019.6461/2019.



This is a PDF file of an unedited manuscript that has been accepted for publication. As a service to our customers we are providing this early version of the manuscript. The manuscript will undergo copyediting, typesetting, and review of the resulting proof before it is published in its final form. Please note that during the production process errors may be discovered which could affect the content, and all legal disclaimers that apply to the journal pertain.

CC 6461

Successful evolution of morphea after hepatitis C virus eradication with direct-acting antiviral agent treatment

Adriana R. Guerra Romero¹, Manuel Pérez Figuera² and Sonia Alonso López¹

Departments of ¹Gastroenterology, and ²Internal Medicine. Hospital Universitario Fundación Alcorcón. Madrid, Spain

Correspondence: Adriana R. Guerra Romero

e-mail: adriaguerra17@gmail.com

Key words: HVC. Hepatitis C virus infection. Morphea. Sofosbuvir. Tledipasvir.

Dear Editor,

We report the case of a 36-year-old, Equatorial Guinean female, who was referred due to the detection of anti-HCV antibodies. She was evaluated by a dermatologist 10 years previously due to the appearance of two plaques in the right inguinal region and thigh, which were identified as morphea via a biopsy. She received treatment with topic corticoids which was unsuccessful. She had also been referred to the rheumatologist due to joint pain. She was positive for anti-centromere antibodies, cryoglobulins and antinuclear antibodies (1/640). Systemic sclerosis was excluded due to a normal chest X-ray, echocardiography and capillaroscopy.

HVC genotype 1a with a low viral load was diagnosed. The patient was treated in 2001 with peg interferon and ribavirin without a response. Although there was no liver fibrosis by Fibroscan® (5.6 Kpa/F0-F1), the patient started a 12 week course of sofosbuvir/ledipasvir in October 2016, achieving a sustained virological response. The morphea plaques progressively lightened after three months of follow-up until they disappeared. The patient has been in remission for over one year.

Discussion

HCV infection has been associated with dermatologic conditions such as lichen planus and cryoglobulinemia. Recently, an association between HCV and systemic sclerosis has been reported. Morphea is a localized scleroderma characterized by a chronic and inflammatory involvement of the connective tissue that affects the skin and the underlying tissue. Although the etiology of morphea is unknown, a relationship with infections has been described. The role of the hepatitis C virus (HCV) in the pathogenesis of morphea is poorly understood. It has been suggested that virus replication in T and B cells stimulate the synthesis of collagen (1,2).

There are some reported cases of morphea healing in VHC patients (3). However, there are no data of the resolution after direct-acting antiviral (DAA) treatment. This is the first reported case of morphea and HVC that showed a clinical improvement after DAA treatment. These findings strengthen the association between both conditions.

References

1. Puzenat E, Aubin F. Sclérodermies. Encyclopédie Médico-Chirurgicale. Dermatologie. Paris: Elsevier. p. 15.
2. Rocca B. Extrahepatic manifestations of hepatitis C virus infection. En: Enfermedades infecciosas y microbiología clínica. Enferm Infecc Microbiol Clin 2004;22(8). DOI: 10.1016/S0213-005X(04)73142-0
3. De Oliveira FL, De Barros Silveira LKC, Rambaldi MLC, et al. Localized scleroderma associated with chronic hepatitis C. Case Rep Dermatol Med 2012 ;2012:743896. DOI:10.1155/2012/743896



Fig. 1. Cutaneous lesions before treatment.