



JANEWAY LESIONS, A BLESSING IN DISGUISE, UNRAVELED THE HIDDEN RETICENT: SYSTEMIC LUPUS ERYTHEMATOSUS (SLE)

Ritwik Ghosh

Post Graduate Trainee, Department of General Medicine, Burdwan Medical College and Hospital, Burdwan.

Arpan Mandal*

Associate Professor, Department of General Medicine, Burdwan Medical College and Hospital, Burdwan. *Corresponding Author

Souvik Dubey

Senior Resident, Department of Neurology (Neuromedicine), Bangur Institute of Neurology (BIN), IPGMER and SSKM Hospital, Kolkata.

ABSTRACT Janeway lesions are erythematous or hemorrhagic macular non-tender lesions on palms and soles and are the consequence of septic embolic events, a fairly suggestive sign of infective endocarditis (IE). Systemic lupus erythematosus (SLE) is a chronic inflammatory disease that has protean manifestations and follows a relapsing and remitting course. Cutaneous lesions are important as a diagnostic aid in SLE as these account for 4 out of 11 revised American Rheumatism Association criteria for disease classification. We are citing a case which presented merely with few constitutional symptoms and Janeway lesions on her soles and eventually diagnosed to be a case of SLE in absence of infective or Libman-Sacks endocarditis.

KEYWORDS : Janeway lesions, Systemic lupus erythematosus

INTRODUCTION:

Janeway lesions are nontender, erythematous or violaceous macules on the palms and/or soles that are usually found in a case of infective endocarditis can also be found in several noninfective disease processes, such as myxoma and eosinophilic endomyocarditis and even in systemic lupus erythematosus [1]. Dermatologic manifestations of SLE include 3 American College of Rheumatology (ACR) criteria: malar rash, photosensitivity, and discoid lupus, which make skin manifestations a cornerstone for diagnosis of SLE. Janeway lesions albeit rare can be found in SLE patients with or without presence of endocarditis. Herein, we present a case with Janeway lesions without an endocarditis that arose in a background of SLE and faded away as the underlying disease improved with treatment.

CASE REPORT:

A lady in her 30s came to our out-patient department complaining of extreme fatigue, arthralgia, weight loss and asymptomatic skin lesions over the toes and soles [images 1,2,3,4] for approximately 1 month. She was afebrile, anemic, conscious and with normal vital signs. Multiple nontender, small, and erythematous macules on the toes and soles suggestive of Janeway lesions were observed. No linear reddish-brown streaks were noted on the distal portions of the nail plates of all fingers. There was no history of direct trauma to the toes and soles. She does not have any history of pregnancy loss, nonhealing oral or vaginal ulcers, hair loss, pedal swelling, breathlessness, any other skin eruptions, seizures and altered behavioral patterns.

Complete metabolic panel were unremarkable except the presence of normocytic normochromic anemia. Thyroid and diabetes panels were normal and tuberculosis was excluded as per protocol. X-ray of chest and ultra sonogram of thorax and abdomen failed to show any serositis and renal echotexture abnormality. Transthoracic echocardiography (TTE) and later transesophageal echocardiography (TEE) proved futile to detect any cardiac lesion that would suggest Libman-Sacks or infective endocarditis. Even in absence of fever repeated blood culture sets were sent for organisms specific for IE including HACEK organisms and *Coxiella burnetii*. Antiphase I IgG titer for *Coxiella* was negative too. Thus possibility of IE was excluded according to modified Duke's criteria. ANA (Anti Nuclear Antibody) tested by Hep2 method was strongly positive. ANA profile displayed discretely high titers of anti Smith, U1RNP, anti ds-DNA and anti-Ro and anti-La antibodies. Complements C3 and C4 levels decreased noticeably without significant proteinuria. Thus diagnosis of SLE was established according to SLICC criteria [2]. Antiphospholipid antibodies (APLA) profile was negative. Beta 2 microglobulin level was pretty high. A full thickness skin biopsy was performed to rule out vasculitis revealed microabscesses in the dermis with thrombosis of small vessels [image5] without vasculitis substantiating the fact that these Janeway lesions were due to the disease process itself; and not due to any

vasculitis. With systemic steroids and hydroxychloroquine therapy, the patient's general condition, anemia and synovitis were mostly improved at the end of the first month of therapy. As she recovered from the underlying disease, the Janeway lesions eventually completely disappeared.



Images 1,2,3 and 4 showing Multiple nontender, small, and erythematous macules on the toes and soles suggestive of Janeway lesions [marked by black arrows].

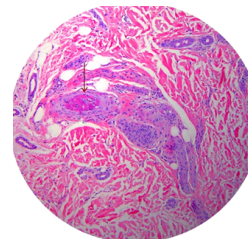


Image 5 showing Skin Biopsy Specimen from the abovementioned areas [images 1,2,3,4] of sole revealing histopathological findings thrombi or thromboemboli within the blood vessels in the dermis [marked by brown arrow] (H/E Stain, OM × 100).

DISCUSSION:

The present case has reminded us that SLE should be included in the differential diagnoses of Janeway lesions apart with infective and noninfective. In addition, these cutaneous signs may indicate how well the treatment for the underlying disease works. Notwithstanding the fact that Janeway lesions are quite non specific finding in SLE, it has prognostic significance [3]. And whenever these characteristic lesions are found in a case of SLE, IE alongside Libman-Sacks endocarditis

should be ruled out as a medical emergency with utmost vigilance.

REFERENCES:

1. Kittisupamongkol W. Janeway lesions, Osler nodes, or neither? *Arch Neurol.* 2010;67(3):373.
2. L, Murphy G, Isenberg D. Systemic lupus erythematosus. *Lancet* 2014;384(9957):878-88.
3. Provost TT, Flynn JA. Lupus Erythematosus. In: Provost TT, Flynn JA, editors. *Cutaneous Medicine: Cutaneous Manifestations of Systemic Disease.* Ontario: PMPH-USA; 2001;41-81.