

A Case of Rhupus with Rowell Syndrome

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Abstract: We report a case of 30-year-old female who presented initially with hair loss, photosensitive malar rash, morning stiffness and synovitis. She was diagnosed with Rhupus syndrome based on clinical and laboratory findings. Few months after starting hydroxychloroquine, esomeprazole and azathioprine, and failing methotrexate (because of erosive pill-induced esophagitis), she presented with generalized maculopapular dusky reddish rash in her body, back and extremities. Her anti-double stranded-DNA, anti-nuclear antibody, anti-Ro/SSA and anti-La/SSB were positive. Anti-cyclic citrullinated peptide antibody was moderately positive. She had low complements: C3 and C4. Herpes simplex IgM and mycoplasma tested negative. Skin biopsy from right arm showed evidence of erythema multiform. She met the criteria for the diagnosis of Rowell syndrome. We managed her with hydroxychloroquine, prednisolone, mycophenolate mofetil and topical agents and discontinued esomeprazole. We also review the management of Rowell syndrome in the literature.

Keywords: Rowell syndrome, systemic lupus erythematosus, erythema multiform, cutaneous lupus erythematosus, rheumatoid arthritis

Introduction

Rowell syndrome is a diagnosis of erythema multiform (EM) that is rarely associated with systemic lupus erythematosus (SLE). It was first described in 1963 by Rowell et al.¹ It occurs mostly in middle-aged women. To reach a diagnosis, all major criteria plus one minor criterion must be met.² Major criteria include the presence of systemic or cutaneous lupus erythematosus (CLE), erythema multiform-like lesions and antinuclear antibodies (ANA). Minor criteria include the presence of chilblains, anti-Ro or anti-La antibodies or rheumatoid factor (RF). While Rhupus was defined by meeting the clinical and laboratory criteria of both SLE, as per 2019 European League Against Rheumatism (EULAR)/American College of Rheumatology (ACR) classification criteria for SLE,³ and that of rheumatoid arthritis (RA), as per the 2010 ACR/EULAR classification criteria of RA.⁴

Case Report

A 30-year-old lady who lives in Taif in Saudi Arabia presented to our hospital in Jeddah with maculopapular reddish dusky rash around tips of fingers and toes, spreading to involve both upper and lower limbs and back.

She first presented to our clinic in late August 2019 with history of hair loss, oral ulcers, photosensitivity and bilateral symmetrical elbow, wrist, metacarpophalangeal (MCP) and proximal interphalangeal (PIP) joint pain with swelling in the wrists. She also suffered from morning stiffness (lasted for 20 mins), fatigue and weight loss. No

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history of abortion, miscarriage, venous thromboembolism or other features of antiphospholipid syndrome was reported previously. She has some features suggestive of Raynaud's phenomenon in form of whitish to bluish and reddish discoloration of her fingers with cold weather, especially in the winter. She is married with three children. No joint deformity was observed. She had a positive ANA, anti-double-stranded DNA (Anti-dsDNA), anti-ribonucleoprotein (anti-RNP), anti-smith and anti-Ro/SSA and anti-La/SSB antibodies. She also had a moderately positive anti-cyclic citrullinated peptide antibody (anti-CCP), normal creatinine and negative RF, lupus anticoagulant, anticardiolipin antibodies and beta-2-glycoprotein-1 antibodies. Additionally, she had minimal proteinuria (Table 1). The possibility of having Rhus syndrome was entertained and we started her on prednisolone 15mg once daily and hydroxychloroquine (HCQ) 200mg twice a day. She was seen in clinic in two months' time and showed improvement. Few weeks later, her joint pain got worse and she sought help in another hospital in Taif. She was thought to have RA mainly and was started on methotrexate (MTX) 12.5mg once weekly orally, with discontinuation of HCQ. Two weeks later, she suffered from nausea, severe heart burn and reflux symptoms. She was admitted and gastroscopy was done and showed erosive esophagitis. The erosive esophagitis was thought to be caused by MTX, so it was discontinued. Azathioprine, prednisolone, and esomeprazole were initiated. We planned to treat her with rituximab therapy, due to unavailability of belimumab.

Few weeks after this admission, in early February 2020, she presented to the ER with new rash that started around the tips of her fingers and toes. Azathioprine was escalated to 150mg and she was discharged on 10mg prednisolone. Her rash progressed further to involve both hands, forearms, her back, lower leg and soles of her feet with ulcerations on the toes' tips. There were bullae formation in both soles of feet; more on the left foot. The rash was maculopapular, blanchable, dusky, red in color and non-itchy (Figure 1). Her face was also involved with the rash spreading on both of her cheeks. The patient also had oral ulcerations in her mouth, with evidence of mucositis, that were painful and located beneath the lips and on the hard palate. Her laboratory investigations showed improving anti-dsDNA, low C3 and C4, and negative mycoplasma IgG, IgM and IgA, negative herpes simplex IgM; however, the IgG antibody was positive. The patient was admitted and started on prednisolone 40mg along with azathioprine 150 mg and HCQ. Topical agents including betamethasone and fusidic acid were used. Dermatology

Table 1 Laboratory Results of the Patient Initial and Follow-Up Serology

Investigation	Initial Result	Follow-Up	Reference Range	Unit
Anti-CCP IgG	58.20	–	<20 is negative 20–39 is weak positive 40–59 is mod. positive ≥ 60 is strong positive	Unit
Anti-RNP	652.7	–	<20 is negative >80 is strong positive	Unit
Anti-Ro/SSA	118.68	–	<20 is negative >80 is strong positive	Unit
Anti-La/SSB	124.29	–	<20 is negative >80 is positive	Unit
AMA	<1:20	–	<1:20 is negative	Titer
Anti-Smith	150	–	<20 is negative >80 is strong positive	Unit
Anti-dsDNA	1722.53	305	0–200 negative 201–300 equivocal 301–800 mod. Positive >800 strong positive	IU/ mL
RF	<10.7	–	<10.7	IU/ mL
ANA IFA	Positive	–	Negative	-
C3	0.38	0.96	0.9–1.8	g/L
C4	<0.06	0.11	0.1–0.4	g/L
Anti-Jo	2.54	–	<20 is negative	Unit
HCV antibody	Negative	–	Negative	–
Anti-HBc	Negative	–	Negative	–
HBsAg	Negative	–	Negative	–
HSV1/2 IgM	Negative	–	Negative	–
HSV 1/2IgG	Positive	–	Negative	–
Mycoplasma IgG	<10	–	<10	AU/ mL
Mycoplasma IgM	<10	–	<10	Index
Mycoplasma IgA	<8.5	–	<8.5	AU
Anticardiolipin IgG	3.58	–	<15 is negative	GPL Unit
Anticardiolipin IgM	8.85	–	<12.5 is negative	MPL Unit

(Continued)

Table 1 (Continued).

Investigation	Initial Result	Follow-Up	Reference Range	Unit
Anticardiolipin IgA	2.42	–	<12 is negative	APL Unit
Beta-2-glycoprotein –I IGA	7.16	–	<20 is negative	SAU
Beta-2-glycoprotein –I IgG	15.38	–	<20 is negative	SGU
Beta-2-glycoprotein –I IgM	1.87	–	<20 is negative	SMU
Lupus anticoagulant	Undetected	–	Undetected	LAI
Serum creatinine	59	62	50–74	umol/L
Urine protein	166	<68	<68	mg/L
Urine protein-creatinine ratio	37.22	CC	≤20	mg/mmol

Abbreviations: Anti-CCP IgG, anti-cyclic citrullinated peptide antibody; anti-RNP, anti-ribonucleoprotein antibody; AMA, anti-mitochondrial antibody; anti-dsDNA, anti-double stranded DNA; RF, rheumatoid factor; ANA, anti-nuclear antibody; C3, complement 3; C4, complement 4; HCV antibody, hepatitis C virus antibody; anti-HBc, hepatitis B core antibody; HBsAg, hepatitis B surface antigen; HSV1/2 IgM, herpes simplex virus 1 and 2 IgM; HSV 1/2 IgG, herpes simplex virus 1 and 2 IgG; CC, cannot calculate urine protein creatinine ratio due to low urine protein.

performed a skin biopsy from the lesion and the patient was scheduled to receive rituximab. While the patient was waiting to receive rituximab, she spiked fever and rituximab infusion was delayed. Examination showed cheeks redness with honey crusting. The possibility of infectious cause, disease flare or medication related side effect was entertained. She was started on piperacillin/tazobactam and vancomycin for treatment with an increase in the dose of prednisolone to 60mg once daily. Superficial cultures grew *Staphylococcus aureus* that was methicillin sensitive and *Acinetobacter baumannii*. Skin biopsy later on that day showed interface dermatitis consistent with EM or Steven Johnson syndrome. After meeting the criteria, a diagnosis of Rowell syndrome was made. Esomeprazole, azathioprine and HCQ were held. The patient overall condition and rash continued to improve with this dose of prednisolone. Nail shedding was observed at the end of second week of admission. HCQ 200mg twice daily was re-introduced along with mycophenolate mofetil (MMF) 500mg BID before discharge and was tolerated well. During her stay, no evidence of venous thromboembolism was observed. In the



Figure 1 Picture taken during flare of erythema multiforme: showing maculopapular, blanchable, dusky and red in color that was non-itchy rash and involved both hands, forearms, body and lower limbs.



Figure 2 Picture of left forearm two months after discharge: resolution of erythema multiforme rash with residual skin hyperpigmentation.

follow-up visit, her symptoms generally improved, with resolution of skin rash in her extremities (Figures 2 and 3), body and face. Complements level raised to near normal and anti-dsDNA improved significantly (Table 1). MMF was escalated



Figure 3 Picture of right forearm two months after discharge from the hospital; again, resolution of erythema multiforme rash can be appreciated.

to 1 g BID and tapering of prednisolone continued. For glucocorticoid-induced osteoporosis prevention, we started the patient on alendronate 70mg once weekly, along with vitamin D and calcium. However, she suffered from heart burn so it was discontinued. We will start her on denosumab next visit.

Discussion

Erythema multiforme is (an acute immune-mediated skin condition characterized by appearance of target-like lesions on the skin⁵). It has multiple well-known causes in the literature including infectious (as herpes simplex virus infection and mycoplasma pneumonia infection), drug related (as non-steroidal anti-inflammatory drugs (NSAIDs), antiepileptics and antibiotics), malignancy and autoimmune diseases.⁵ A typical course includes resolution within 2 weeks, with possible recurrence in some patients.

In the rare presence of both EM and SLE, when meeting the criteria,² a diagnosis of Rowell syndrome is reached. Rowell syndrome has been described in the literature to be part of CLE spectrum.⁶ Nevertheless, it is also found to be

triggered after starting certain medications. For example, proton pump inhibitors, terbinafine and valproic acid.⁷

Looking back to our patient we can note that her lesions started 3 months after initiating esomeprazole, for her MTX-related erosive esophagitis, and azathioprine. Her symptoms worsened with the increase in the dose of azathioprine to 150 mg despite being on low dose steroids. Azathioprine has been described in the literature to be associated with Rowell syndrome, but this was not a very strong association.⁸ Proton pump inhibitors were also described in the literature after similar period of time.⁷ Never the less her symptoms started to improve when both medications were discontinued and while receiving higher doses of steroids. To our knowledge this is the first case report of overlap syndrome (Rheumatoid arthritis) presenting with Rowell syndrome. Therefore, the clinical and therapeutic response of the patient to various therapies represents an interesting addition to the literature.

Furthermore, when managing EM and the possibility of herpes simplex virus is entertained, usually an antiviral is used. Occasionally prednisolone and other immune suppressant agents, such as azathioprine or MMF, are introduced.⁵ In CLE and Rowell syndrome, HCQ in addition to other immune suppressant agents has been used. Table 2 shows summary of different agents used in 46 open access case reports of Rowell syndrome found in Google scholar and PubMed for the treatment of Rowell syndrome and their frequencies. Mean age for

Table 2 Summary of 46 Open Access Case Reports of 48 Patients with Rowell Syndrome^{7,11–55}

Agent Name	Number of Patients Treated Successfully	Percentage of Success in Treated Patients	Percentage of Patients Treated Out of 48 Patients
Steroids	42/45	93%	93%
HCQ/chloroquine	25/27	92%	55%
Topical agents	13/13	100%	28%
Azathioprine	7/8	87%	16%
MMF	5/5	100%	1%
Cyclosporine	2/3	66%	0.6%
Antibiotics	4/4	100%	0.8%
Dapsone	0/1	0%	0.02%
Thalidomide	0/1	0%	0.02%
NSAIDs	1/1	100%	0.02%
Cyclophosphamide	1/2	50%	0.04%
IVIG	1/1	100%	0.02%

Abbreviations: HCQ, hydroxychloroquine; MMF, mycophenolate mofetil; NSAIDs, non-steroidal anti-inflammatory drugs; IVIG, intravenous immunoglobulin.

patients in these papers was 39 years old and 68% of patients were female. Most authors used prednisolone to induce remission, in addition to HCQ as a backbone therapy. Other immunosuppressant agents, such as azathioprine and MMF, have also been used. These are in concordance with EULAR recommendations of management of CLE.^{9,10}

We conclude that Rowell syndrome can be treated according to the skin section of 2019 update of the EULAR recommendations for the management of SLE.

Institutional Approval

No institutional approval was required for the publication of this manuscript.

Consent

A written informed consent was obtained from the patient for publication of her case details with image of her cutaneous lesions.

Disclosure

All authors declare no conflict of interest.

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