International Journal of Pharmacy and Pharmaceutical Sciences

ISSN- 0975-1491 Vol 11, Issue 3, 2019

Case Study

CASE STUDY ON BETA BLOCKERS INDUCED PSORIASIS

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Received: 29 Aug 2018 Revised and Accepted: 26 Jan 2019

ABSTRACT

Drug-induced Psoriasis is one among the common etiological factors of Psoriasis reported worldwide. Familiar drugs known to cause psoriasiform eruptions include Anti-malarials, Beta blockers, NSAIDs, Lithium. etc. Certain antihypertensives like ACE inhibitors, diuretics are also documented to have caused psoriatic episodes.

A 57 y old South-Indian male patient with a history of Hypertension, Diabetes Mellitus, Atrial Fibrillation for 4 y; was on antihypertensive therapy for Hypertension and Atrial Fibrillation with proponolol for past 2 y and metoprolol initially. He was presented to the hospital two weeks after switching on to Metoprolol therapy for chief complaints of erythematous scaly lesions especially over both the extremities and paronydrial appearance of nails. Initially, he was on Propranolol therapy which was then shifted to Metoprolol due to an appearance of oral lesions in the mouth. Metoprolol was now discontinued and switched on to Atenolol. After 1-2 w of therapy with Atenolol, the lesions were found to disappear and no recurrence of psoriatic conditions were found.

Proper reviewing of medical history for any allergic reactions and the optimization of drug therapy through Therapeutic Drug Monitoring could be initiated by Clinical Pharmacist in order to avoid such drug-induced flares.

Keywords: Beta blockers, Clinical Pharmacist, Drug-induced, Erythematous lesions, Hypertension

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DOI: http://dx.doi.org/10.22159/ijpps.2019v11i3.29388

INTRODUCTION

Psoriasis is defined on a clinical basis as chronic, relapsing, remitting papulosquamous eruption with typical localization on the extensor surfaces like elbows and knees involving scalp, genitalia or nails and other sites [1-4]. Etiology includes environmental and genetic factors. Erythematous, scaly skin lesions along with additional manifestations in nails and joints are commonly present. Most common form is plaque psoriasis. Pustular, guttate, inverse and erythrodermic psoriasis are the atypical forms [5-8].

It is clearly known that individual with psoriasis have an increased risk of hypertension and β blockers anti-hypertensive medications, cause psoriasis at its adverse levels. Psoriasis and hypertension are found to be concomitantly related and most medications used in treating the co-morbidities are found to aggravate psoriasis among which β blockers are more common [12-14]. Correlation mechanism of Psoriasis which is induced by β blockers is explained in chart I.

Biological mode of a possible mechanism for drug provoked psoriasis is illustrated in Chart II [18-20].

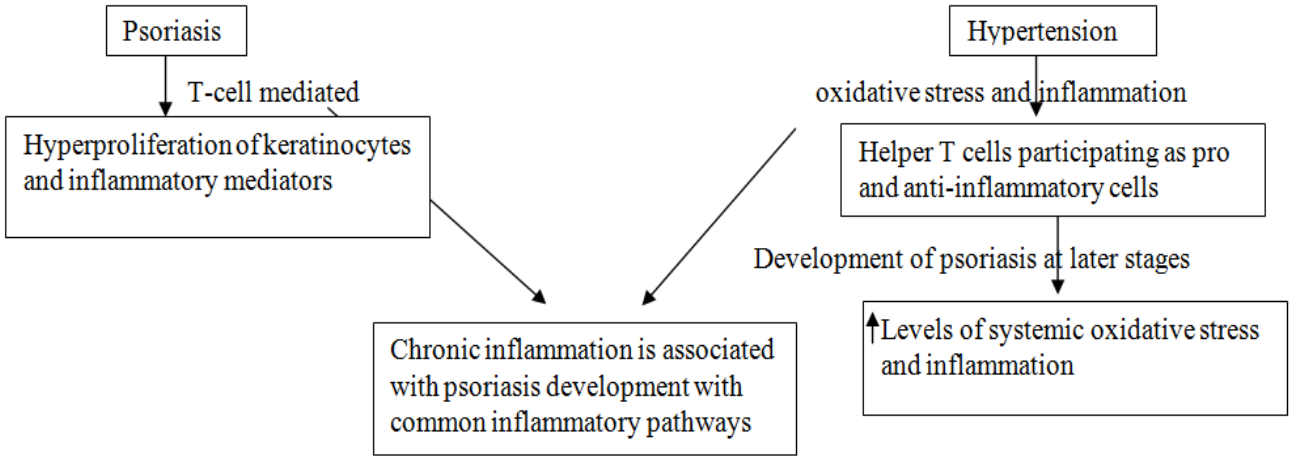

Chart I: Correlation mechanism of psoriasis and hypertension [15-17]

Drug-induced psoriasis is found to act in two ways. One is where the psoriasis is due to an adverse effect of the drug and which subsides on the gradual withdrawal of the drug. Second is an aggravation of the persisting psoriatic condition where withdrawal of drug has no much effect [21-24]. Latency period from ingestion of β blockers to psoriatic flares varies from several days to 12 mo in patients with psoriasis [25].

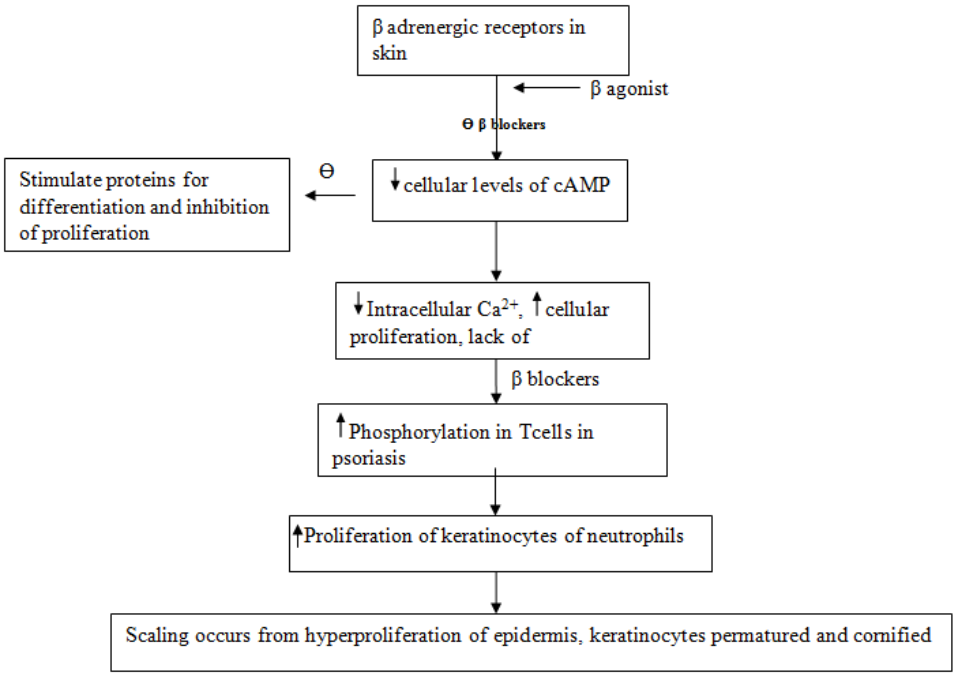

Chart II: Relationship of β blockers and psoriasis in biological and immunological aspects

CASE REPORT

A 57 y old South-Indian Male patient was admitted to the hospital with chief complaints of shredding of scales which are silver, especially those present at his extremities since 3 d. There were generalized erythema scaling with fine scales all over the body. His nails showed paronydrial appearance as depicted in fig. I. There was no history of any such family reports. He had a medical history of Hypertension, Type II Diabetes mellitus and Atrial Fibrillation for which he was on medication for the past 4 y. He was a chain smoker and a functional alcoholic with chewing of Tobacco gums daily. He had a blood pressure of 160/110 mmHg. There was a previous history of oral lesions with propranolol therapy which was discontinued due to poor control in Blood Pressure levels and lesions in the mouth. The patient was on treatment with anti-hypertensive and anti-arrhythmic drugs for the past 4 y and 2 y for his atrial fibrillation. Propranolol and other calcium channel blockers were indicated. Propranolol40 mg orally twice a day was switched on to metaprolol 100 mg orally daily due to the low therapeutic outcome from propranolol after 2 y of therapy. With regards to General, Physical and Systemic examination, the patient was on Erythematous Psoriasis due to Metoprolol. Lab examinations were performed from time to time as shown in table I. Other medications which were taken by the patients included Metfromin (500 mg), Glibenclamide (1 mg), Verapamil (200 mg). Metoprolol was withdrawn immediately on knowing the etiology and topical corticosteroids (1% Hydrocortisone) was prescribed. Methotrexate was prescribed initially with which reducation in scales and eruptions were found. Later on he was prescribed with Atenolol 50 mg for his hypertensive and atrial fibrillation condition.

Table 1: Laboratory values of the patient

Diagnostic parameters	Patients value	Normal values	Inference
PCV	43.4%	41-59%	Within limits
WBC	15.60x10^9/l	4.5-11.0x10^9/l	Increased
RBC	5.07x10[12]/l	3.8-4.8 x10[12]/l	Increased
Hemoglobin	11.1 g/dl	12-17 g/dl	Decreased
MCV	95.2fL	76-96fL	Within limits
MCH	31.3pg	27-32 pg	Within limits
MCHC	32.8	31-35	Within limits
Platelets	2.19 x 10^9	1.5-4x 10^9	Within limits
Lymphocytes	21.3%	20-40%	Within limits
ESR	09 min, 26 sec	5-20 mm/hr	Within limits
RBS	288 mg/dl	80-140m/dl	Increased
Urea	26.9 mg/dl	7-18 mg/dl	Increased
Creatinine	0.9 mg/dl	0.6-1.3 mg/Dl	Within limits
Cholestrol	218 mg	0-400 mg	Within limits
Blood group	O+ve		
BMI	27.8		
VDRL	-ve		
SGOT	18 U/l	15-17 U/l	Increased
SGPT	62 U/l	30-65 U/l	Within limits

DISCUSSION

One of the most common side effects of β blockers is psoriasiform drug eruptions. The documented adverse drug reactions of metoprolol include Acute Generalized Exanthematous pustulosis (AGEP), Purpura, Bullous Pemphigoid, Steven-Johnson's syndrome, immune thrombocytopenia. The mechanism involves impairment in lymphocyte transformation with a gradual decrease in cyclic adenosine monophosphate (cAMP) and cell proliferation. Others like ACE inhibitors, frusemide, biguanides etc, could also cause psoriasiform eruptions all over the body [26, 27]. With the discontinuation of metoprolol, the lesions and eruptions were found to be reduced and there was no reappearance of lesions when treated with atenolol in its initial stage. This clearly depicts that the above case is drug-induced erythematous psoriasis with the involvement of nails and extremities. His history of events with beta-blocker therapy is given in table II.

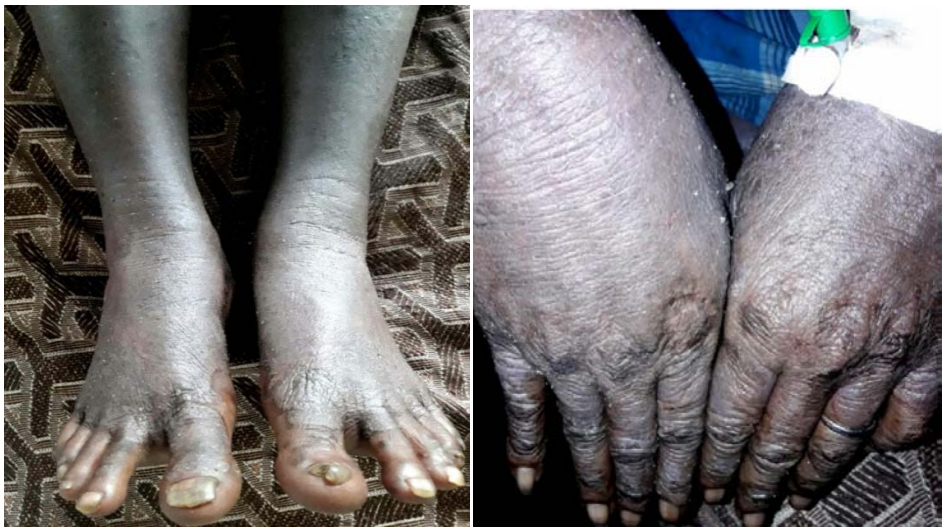

Fig. I: Erythematic lesions on both the extremities of the patient with metaprolol drug therapy

Table II: History of events of psoriasis with beta blocker therapy

S. No.	Duration since initial visit	Shift from one medication to another	Comments
1	0-2 y	Initiation of propranolol	No much therapeutic effect obtained, presence of oral lesions in mouth
2	1 w	Discontinuation of propranolol and initiation of metoprolol	Rash and itching also present in body parts
3	1-2 w	Continuation of metoprolol	Raising of psoriasiform eruptions especially at extremities with scales and paronydrial appearance of nails
4	1-2 w	Discontinuation of metoprolol	Resolving of rashes
5	4 w	Initiation of atenolol	Psoriasis type lesions and scales were resolved

CONCLUSION

Aggravation or exacerbation of drug-induced psoriasis could be reduced by discontinuing the intake of the particular drug. A sound knowledge of the drugs which leads to such conditions could be known in prior to health professionals and individualized therapy could be done in such situations. Timely and propitious detection and withdrawal of all imminent causes is crucial for a positive outcome.

ACKNOWLEDGMENT

The authors are thankful to Dr. C. Sekar, Chief Medical Superintendent, Government hospital, Thiruvallur for his constant support and encouragement throughout the study. We thank the patients who gave their willing consent for the publication of their case by sharing complete information needed for the study.

COMPLIANCE WITH ETHICAL STANDARDS

Written informed consent was obtained from the patient for publication of the case study, the inclusion of the accompanying images. Copies of written consent may be requested for review from the corresponding author.

AUTHORS CONTRIBUTION

Each author has given their full effort in receiving the consent from the patient, preparing the manuscript, aligning it and presenting it to its full form.

CONFLICT OF INTERESTS

The authors declare no conflicts of interest concerning the content of this case report

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