E-ISSN 2250-0944
ISSN 2250-1150
doi: 10.31838/ijprt/10.01.05

Case Report

Case report on pemphigus vulgaris

S. PARVEEN[1], DR. C. MADHUSUDHANA CHETTY[2], S. SUNIL KUMAR[3], S. BENJAMEN SAMUEL[4], M. VISHNU VARDHAN[5], C. RENUKA THEJESHWINI
[1,2,3]*Assistant professor, Department of Pharmacology, Santhiram College of Pharmacy, Nandyal, Kurnool, Andhra Pradesh*
[4,5,6] *Pharm.D, Department of Pharmacy Practice, Santhiram College of Pharmacy, Nandyal, Kurnool, Andhra Pradesh*
Received: 22.05.19, Revised: 13.06.19, Accepted: 28.08.19

Corresponding Author Email: parveencology@gmail.com

ABSTRACT
Pemphigus vulgaris is a rare potentially life threatening auto immune disease it may cause severe blistering of the skin and mucous membranes lining the mouth, nose, throat, eye and genital area, blisters develop in the upper layer of the skin surface breaks away easily leaving raw areas erosions that can be extensive and painful. Our immune system wakes antibodies to fight infection Pemphigus vulgaris work against tissue in the body auto antibodies in Pemphigus vulgaris attack proteins called. Desmoglein this proteins present on the cells in the outer layer of the skin (epidermis) this causes skin lesions starts as thin walled fragile blisters a collection of clear fluid within the skin that burst easily leaving raw area known as erosions. This case report described the case of a patient who complained of hypotension and diabetes mellitus since one year the unknown organism/insect bite to the right leg after next day it produces swelling too much inflamed and the scaly appearance who was diagnosed as having Pemphigus vulgaris. Standard treatment for Pemphigus vulgaris is an oral corticosteroid, often with the addition with the adjuvant therapy to improve the disease control.

Keywords: Pemphigus vulgaris, Desmoglein, Fragile blisters, Erosions

INTRODUCTION

Pemphigus vulgaris is a chronic inflammatory autoimmune bullous disease affected around 3 people per 100,000 populations with the highest incidence in the 5th & 6th decade of life with male to female ratio of

1:2 [1, 2]. Pemphigus includes a group of blistering diseases involving auto antibodies that target proteins found in the desmosomes intercellular adhesion protein complexes. Most of the Pemphigus is classified as being a subtype of Pemphigus vulgaris (PV), Pemphigus folia cells (PF), para neoplastic Pemphigus (PNP).

Pemphigus was a highly fatal disease until the introduction of corticosteroids (CS) which have reduced its mortality rate from 75% to less than 10% with the most morbidity and mortality due to iatrogenic causes rather than the disease itself[3,4]. The disease usually occurs in the patients with certain HLA genotypes which generates B-cells responsible for the specific Auto antibodies the activation of these B-cells requires a complex interaction with CD4 + T-helper 2 (Th2) cells and it is the Th2 cell over activation that leads to the auto antibody production that is necessary for PV and PF[1,2,5].

Case report:

A 53 years men went to the field work while doing the work the unknown organism/insect get bitted to the right leg, he thought that it was a simple insect (ant) bite but after next day the leg was got swollen too much and inflammed at the site of the

bite there was a scaly line appearance associated with itching, A round and spherical shaped dots are observed all over the body like elbow, feet, ankles, back side of the body. After 2 months this was fully observed and the next he immediately joined the hospital.

This is a case of Pemphigus vulgaris with hypertension and diabetes mellitus with laboratory data as HbA1C – 6, PBS – 155 mg/dl, PPBS – 267 mg/dl and the department of biochemistry for Renal Function Test revealed Serum Creatinine as 0.95 mg/dl and the department of microbiology ICTC is non reactive; with known Type 2 diabetes mellitus and was treated with Gluconorm-G1, Gabamax gold, Telvas 20mg, Decadron, Taxim 200mg, Ranitidine 150mg, Atarax 10mg, Fourtz-B, Shelcal 500mg, Fusigen-B cream, Condy soaks, Meganearon plus.

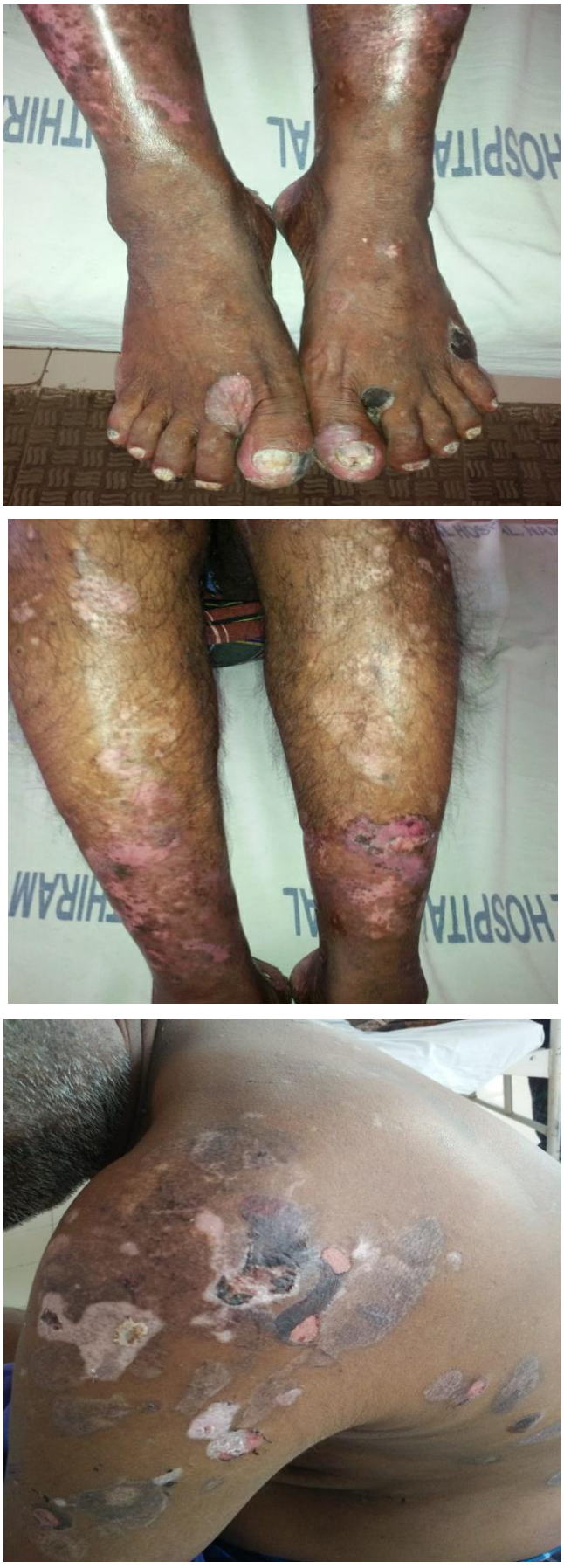

Fig.1: Before treatment

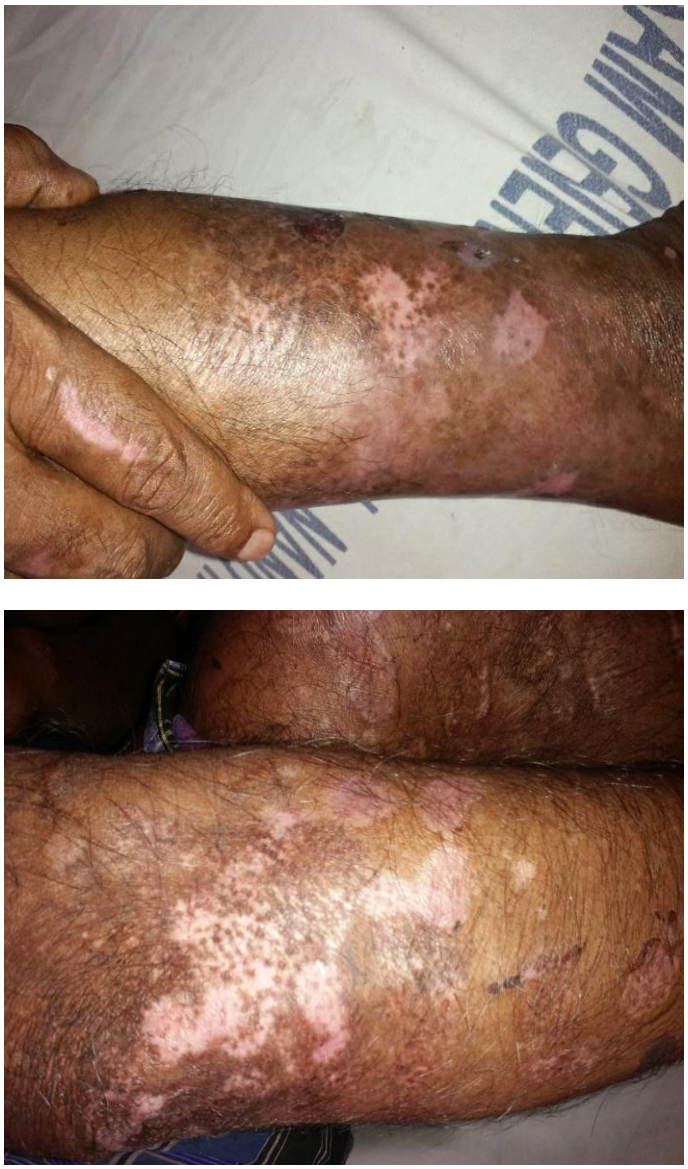

Fig.2: After treatment

Discussion

In Pemphigus vulgaris lesions at first comprise small asymptomatic blisters although these are very thin walled and easily rupture giving rise to painful and haemorrhagic erosions[6]. While the lesions can be located anywhere the oral cavity they are the most often found in the areas subjected to the frictional trauma mainly in the tongue, palate and lower lip. Pemphigus vulgaris should be ruled out one of those pemphigoids the bullous dermatitis of the autoimmune origin that is relatively uncommon. The initial aim of treatment is to reduce disease remission this should be followed by a period of maintenance treatment using the minimum drug doses required for disease control in order to minimize their side effects.

Systemic oral corticosteriods are the treatment of choice in the treatment of pemphigus vulgaris. Topical steroid therapy alone is insufficient for sustained control of the disease because of the systemic autoimmune characteristic of pemphigus vulgaris. Topical corticosteriod rinses and cream including fusigen-B intraledional triamcinolsne may be used for resistance local lesions. The administration of high dose corticosteriods may cause fatal complications.

Conclusion

Pemphigus vulgaris is rare reported .In the majority of the patients main mucous membrane erosions are the presenting sign of pemphigus vulgaris and may be the only sign of pemphigus vulgaris for an average of 5months before skin lesions develop. Mortality and morbidity are related to the extent of the disease. Maximum dose of systemic steroids required to induce remission and the presence of other diseases. prognosis is worse in patients with extensive diseases and in old patients.

References

1) Di Zenzo, G., Amber, K.T., Sayar, B.S., Müller, E.J. and Borradori, L., 2016, January. Immune response in pemphigus and beyond: progresses and emerging concepts. In *Seminars in immunopathology* (Vol. 38, No. 1, pp. 57-74). Springer Berlin Heidelberg.
2) Giordano, C.N. and Sinha, A.A., 2012. Cytokine networks in Pemphigus vulgaris: An integrated viewpoint. *Autoimmunity*, *45*(6), pp.427-439.
3) Martin LX, Werth VP, Villaneuva EV, Murrell DF. A systematic review of randomised controlled trials for Pemphigus vulgaris and Pemphigus foliaceus. J Am Acad Dermatol. 2011 May; 64 (5): 903-8. PubMed PMID: 21353333.
4) Atzmony, L., Hodak, E., Leshem, Y.A., Rosenbaum, O., Gdalevich, M., Anhalt, G.J. and Mimouni, D., 2015. The role of adjuvant therapy in pemphigus: a systematic review and meta-analysis. *Journal of the American Academy of Dermatology*, *73*(2), pp.264-271.
5) Tavakolpour, S. and Tavakolpour, V., 2016. Interleukin 4 inhibition as a potential therapeutic in pemphigus. *Cytokine*, *77*, pp.189-195.
6) Manojkumar ST,Raju A,Pradeep R A.Pemphigus Vulgaris: effect on periodontal health Journal Of Oral Science 2010; 52 (3)20-4.