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Case Study

A Case report on Pemphigus Vulgaris

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Abstract

Pemphigus Vulgaris (PV) is a chronic auto immune bullous dermatosis. It is the most frequent and severe form of pemphigus occurring universally between 40 and 60 years of age. It is a life-threatening sickness causing blisters and erosions on the oral mucosa followed by lesions on the other mucous membranes and flaccid blisters on the skin, which can be disseminated. The underlying mechanism involves binding of immunoglobulin G auto anti-bodies to desmoglein (Dsg) 1 and 3, a transmembrane glycoprotein adhesion molecule present on desmosomes. Here a 47 years old male patient admitted in the Department of Dermatology with a chief complaint of multiple fluid filled lesions along with erosions present all over the body in the last 3 days. His past medical history revealed that he was suffering from this problem since one and half year and on corticosteroid medication, which provided temporary relief. The patient condition was not improved with the administration of corticosteroids but there may be a chance of improvement, if the treatment strategy was changed.

Keywords: Pemphigus Vulgaris (PV), Lesions and erosions, Immunoglobulin G, Corticosteroids.

INTRODUCTION

The word “Pemphigus,” derived from the Greek word “Pemphix” meaning bubble or blister, was originally used by Wichman in 1791, for describing a chronic blistering disease that corresponds to present-day Pemphigus Vulgaris (PV).

The two major groups are PV and pemphigus foliaceus. Their difference lies in the level of acantholysis, with the former in the suprabasil level and the latter in the sub corneal level. The other forms are vegetans, erythematosus, IgA pemphigus, and Para Neoplastic Pemphigus (PNP).[1]

PV is the most frequent and most severe form of pemphigus, occurring universally, usually between 40 and 60 years of age.

It usually begins with blisters and erosions on the oral mucosa, followed by lesions on other mucous membranes and flaccid blisters on the skin, which can be disseminated.[2] Particularly, the immunoglobulin subclass of these auto antibodies is IgG4 attacks surface components known as desmogleins 1 and 3. The epitope structure of these desmosomes cause damage. Essentially, a type 2 hypersensitivity reaction takes place in which antibodies attach and destroy cell surface receptors.

CASE HISTORY

A 47 years old male patient admitted in the Department of Dermatology with a chief complaint of multiple fluid filled lesions along with erosions present all over the body in the last 3 days. He was apparently normal one and half years back later he noticed multiple fluid filled lesions all over the body. First the lesions are fluid filled which eroded on raw surfaces associated with dozing and burning sensation exacerbated on summer. The lesions first get started over the extremities then it progressed to involve trunk.

He is a Known case of diabetes mellitus and hypertension and using medications for two diseases.

On physical examination the patient was conscious and coherent, pallor(+ve) noticed. Blood Pressure was 160/100 mmHg, Pulse rate -117 bpm. On systemic Examination: CVS-S1S2+, CNS: NAD, Per abdomen- Soft. RS-BAE+.

On dermatological examination multiple erosions present along the trunk, axilla, groins, and extremities. On oral mucosa white membrane present along with few erosions. On scalp, multiple crusted dirty looking scales present over the scalp.

Laboratory Investigations

Table 1: List of investigations performed

S.No.	CLINICAL INVESTIOGATIONS	OBSEREVE VALVE	REFRENCE VALVE
1.	Haemoglobin	8.2 gm	13-15 gm
2.	Total count	6,800cells/mm3	4500-11,000/mm3
3.		Differential count	
4.	Polymorphs	82%	40-65%
5.	Lymphocytes	10%	30-50%
6.	Eosinophils	6%	2-8%
7.	Monocytes	2%	2-8%
8.	Erythrocyte Sedimentation Rate	25mm/Hr	< 15% mm/hr
9.	Platelets	2 lac cells/mm^3	1.5 -4.5 lac cells/mm^3
10.	Random Blood Sugar	329 mgs/dl	140-180 mgs/dl
11.	C-Reactive Protein (CRP)	Positive	
12.		Microscopical Examination	
13.	Albumin	+2	
14.	Pus cells	3 to 5 cells/ hpf	
15.	Epithelial Cells	1 to 2 cells/ hpf	
16.	Urine for Culture Sensitivity	Negative for Bacterial growth	
17.	Blood for Culture Sensitivity	Negative for Bacterial growth	
18.	Direct Nikolsky	Positive	
19.	Indirect Nikolsky	Negative	

Fig 1: Patient with Pemphigus Vulgaris

So based on subjective and objective evaluation patient was diagnosed as Pemphigus Vulgaris (PV) which was shown in fig 1. Patient was admitted and taken treatment in the hospital for 75 days. Clinical evaluation was done and patient was treated symptomatically Inj.Decadron–2cc-IV-OD, T. Mycophenolatemofetil-500mg-PO-QID, T.Cyclophosphomide–100mg- PO-OD, Oint. Fusidic Acid-BD, T.Metformin-500mg- PO- BD, T.Sitagliptine –100 mg -PO- OD, T. Chymoral Forte– PO-TID, Inj. Pantoprazole – 40mg-IV-OD, Inj.Ceftriaxone-1gm-IV-BD, Inj.CPM- IM-0D, T.Quetiapine- 50mg-OD.

DISCUSSION

Pemphigus is defined as a group of life-threatening blistering disorder of skin and mucous membrane.[4] In this case the patient who presented with multiple fluid filled lesions along with erosions present all over the body. The etiology of PV is still unknown Although the disease has raised much concern.[5] Rituximab can induce a prolonged clinical remission in patients with both pemphigus vulgaris and pemphigus foliaceous after a single course of 4 treatments. The preliminary experiences worldwide make rituximab a promising therapeutic option for patients with autoimmune diseases. The high costs and the limited knowledge of long-term adverse effects, however, limit its use to selected patients with treatment-resistant or life-threatening disease.[6] in our case patient was not treated with rituximab which may be the reason for which patient condition was not improved.

CONCLUSION

PV is an auto immune blistering mucocutaneous disease with a genetic predisposition. The severity of disease is variable. Timely diagnosis and prompt treatment are hence necessary for complete cure with reduced morbidity and mortality associated with the disease.

ABBREVIATION USED

Pemphigus Vulgaris (PV), Para Neoplastic Pemphigus (PNP), Erythrocyte Sedimentation Rate (ESR), C-Reactive Protein (CRP).

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