An assertive interventional study on pemphigus vulgaris

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CASE REPORT

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INTRODUCTION

Pemphigus was derived from the Greek word ‘pemphix’ meaning bubble or blister and ‘vulgaris’ a Latin word meaning common [1]. Pemphigus vulgaris (PV) is a rare but potentially life-threatening autoimmune blistering disease affecting the skin and mucous membranes. The disease is caused by circulating antibodies targeted against the desmogleins (desmosomal glycoproteins expressed on the epithelial cells of the mucosa and skin). Disruption of these adhesion protein results in a loss of cohesion between cells leading to acantholysis and blister formation. The clinical result of this process is the easy rupture of the blisters, leaving behind painful sloughing erosions on the mucosa and/or skin. We report a case of severe PV in a 47-year-old woman presenting with widespread, painful, eroded mucocutaneous lesions. The uniqueness of the disease demanded the pharmaceutical care provided by a clinical pharmacist to successfully manage the problem. This report highlights the importance of an early interdisciplinary team approach to improve the outcome of patients suffering from Pemphigus vulgaris.

CASE DESCRIPTION

A 47-year-old female was admitted to the Dermatology department with the chief complaints of oral ulcers, pain in mouth and throat, since 3-4 months, swelling over the nails, lesions over the groin, back neck, labia extended to the anus, per vaginal discharge with itching at perianal region since 2 months. She has taken treatment outside hospitals (Figures 1 and 2). No history of Diabetes and Hypertension. Personal and family histories were not significant. From the complaints, history and clinical examination, a provisional diagnosis of Pustular psoriasis, Secondary syphilis was made. The Final diagnosis of Pemphigus Vulgaris was made based on clinical and histopathological findings. (The consent was taken from the patient before enrolling into the study. Also, the Institutional Ethics Committee of the college has granted the ethical approval for the study.)

Investigations

Perilesional Biopsy shows intra epidermal supra basal acantholytic blistering dermatitis. Several acantholytic cells and neutrophils seen in blisters. The floor of the blister shows “tombstone” pattern with occasional acantholytic cells. Mild spongiosis with neutrophils present at the periphery of the blister.

Impression: Pemphigus vulgaris

Serum Australia antigen – Negative
HIV I and II – Negative
STS (serologic test for Syphilis) - Negative

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Management

In the Inpatient department (IPD), on the first day, the patient was given with inj. Dexamethasone 8 mg BD, inj. cefotaxime 1g BD, inj. Amoxicillin + Clavulanic acid 625 mg BD, inj. Ranitidine 50 mg BD and diluted chlorhexidine mouthwash (Figures 3-5). On the second day, inj. Amoxicillin + Clavulanic acid was withdrawn from the treatment through the clinical pharmacist intervention while the rest of the medications were continued. On the third day of hospitalization, patient was discharged with the following medication with follow up advice after 2 weeks.

Table 1: Medications on discharge.

<table>
<thead>
<tr>
<th>Medication</th>
<th>Dose</th>
<th>Duration</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prednisolone</td>
<td>40 mg/day</td>
<td>2 weeks</td>
</tr>
<tr>
<td>Dapsone</td>
<td>20 mg/day</td>
<td>Next 2 weeks</td>
</tr>
<tr>
<td>Terbinafine</td>
<td>10 mg/day</td>
<td>Next 2 weeks</td>
</tr>
<tr>
<td>Triamcinolone ONT</td>
<td>100 mg/day</td>
<td>4 weeks</td>
</tr>
<tr>
<td>Ranitidine</td>
<td>250 mg/day</td>
<td>2 weeks</td>
</tr>
<tr>
<td></td>
<td>0.1% w/w</td>
<td>2 weeks</td>
</tr>
<tr>
<td></td>
<td>150 mg BD</td>
<td></td>
</tr>
</tbody>
</table>

The patient made a slow but gradual recovery over the next 4 weeks. Two months since discharge, the patient continues to be reviewed on regular basis by the dermatology team (Table 1). She has still prescribed dapsone and is on a tapering dose of prednisolone (currently 10 mg once daily). She has fortunately not suffered a recurrence of the disease till now.

DISCUSSION AND CONCLUSION

PV can be either induced or triggered by certain drugs. (Pemphigus that continues after a patient stops taking a drug is referred to as triggered, whereas lesions that start healing soon after withdrawal are referred to as induced). The clinical, histological and immunofluorescence abnormalities of drug-induced pemphigus vulgaris are similar to those of the idiopathic variety. Drugs associated with PV include Penicillin, captopril, enalapril, penicillamine, thioproline, interleukin-2 (IL-2), nifedipine, piroxicam, and rifampicin [5-7].

As Amoxicillin-Clavulanic acid is found to cause exacerbation of PV and drug-induced PV, it was withdrawn from the prescription through the clinical pharmacist intervention.

The present case study emphasizes the role of a clinical pharmacist in the management of PV through therapeutic interventions, consultation with other health care professionals and counseling of patient. The main objective in the management of the pemphigus vulgaris is to control the disease, prevent relapses and avoid adverse events associated with the prolonged use of steroids and immunosuppressive agents. Therefore the prevention and management of patients with PV requires coordination of care between dermatologist, physician and clinical pharmacist.

ACKNOWLEDGMENT

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CONFLICT OF INTEREST

The author declares there is no conflict of interest.

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