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Drug-Induced Bullous Pemphigoid

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ABSTRACT
Drug-Induced Bullous Pemphigoid is an autoimmune disorder in which they develop blister forms on the skin it mainly interacts with Immunoglobin G contains the Fab region which interacts with proteins activated to attach hemidesmosomes to one of the antigen called Bullous Pemphigoid antigen 1 and antigen 2 and destroys the basal layer of the epidermis of skin leads to Bullous Pemphigoid which triggered by the drugs of chemotherapy like paclitaxel and carboplatin in this condition. It works mainly to reduce apoptosis and reduce cancer. By acting on the cells it may reduce abnormal cell growth and reduces the immune system to produce an overgrowth of cells. Acting on our immune system can lead to Bullous Pemphigoid.

KEYWORDS
Bullous Pemphigoid
Ig G Antibodies
Cell Growth
Immune System

I. Introduction:

It is a rare condition that occurs in older conditions. It is an autoimmune skin disease it causes the skin to form blisters[1]. Skin is divided into 3 layers epidermis, dermis, and hypodermis. Epidermis is made up of keratinocytes which are pan cake-shaped cells containing keratin protein. Stratum basale is a single layer of stem cells that continually divides producing new keratinocytes also contains melanocytes that secrete melanin which is a pigment-protein. Basement membrane containing collagen and laminins and hemidesmosomes which is a protein complex present in the bottom of basal cells. It is mainly triggered by furosemide, captopril, penicillamine, NSAIDS, and antibiotics. Bullous pemphigoid is a type II hypersensitivity reaction in which our immune system produces antibodies[2]. B cells produce Ig G antibodies containing the Fab region and Fc region. Fab region binds with the pathogens and helps other immune cells destroy that pathogen IgG. IgG antibodies also activate the complement cells that destroy the pathogen or induce inflammation. In bullous pemphigoid, the Fab region binds with proteins to produce the hemidesmosome one of the antigens isbullous pemphigoid antigen 1 (BPAG11 or dystonia) and the other protein is the bullous pemphigoid antigen 2 (BAPG2,BP180 or the 17 Collagen)[3]. The Fcregionactivates the complement system the process starts with the C1 protein binds with the Fc region which engages the complement family (c2-c9) some are activated by being cleaved by an enzyme the cleaved enzymes c3a,c4 a, and c5a act as chemotactic factors they attract the mast cells. The mast cells degranulate and releasemolecules that aretumor necrosis factor, leukotrienes, and cytokines like cytokines.also attract the inflammatory cells like neutrophils, eosinophils, macrophages, and T cells these inflammatory cells secrete proteolyticenzymes which the destroys the proteins of hemidesmosomes(BPAG and BPAG2) if hemidesmosomes destroyed the basal cells separated from the basement membrane and split form in between epidermis and dermis. It forms the subepidermal bullae is distinct from the epidermal bullae. In epidermal bulla form in pemphigus vulgaris. In bullous pemphigoid, it also affects the keratinocytesstarting with the inner cells of the dermis. There is no Nikolsky's sign and circulating IgG antibodies. The patient received the chemotherapycontaining sulfhydryl group in cells that changethe antigenic properties of the cell surface leading to antigen and antibody interaction leading to pemphigoid.

II. CASE REPORT

A 57-old-female patient came to the DVL ward of GSL General Hospital, Rajahmundry with a complaint of blisters (fluid-filled) all over the body for 3 days associated with a burning sensation.

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The patient was all right 3 days ago when she developed multiple fluid-filled red color blisters on her hands, back, stomach, and legs associated with a burning sensation. The patent was diagnosed witha calcium cervix 2months ago following which chemotherapy started (Inj paclitaxel, Injcarboplatin)2 cycles of chemotherapy were completed lasted chemotherapy was on 10/04/2023.History of past illness includes similar episodes after 1st chemotherapy lesions healed with hyperpigmentation (1st chemo on 12/3/23). Having known chief complaint of diabetes and hypertension for10 years on telmisartan and metformin.

Used Inj paclitaxel and Inj carboplatin.

Laboratory investigations include: total bilirubin: 0.2(0.2-1.2mg/dl), Direct Bilirubin :0.1 (0-0.3), SGOT:19(5-34 IU/L),SGPT :36 (Upto 34U/L), Alkaline phosphatase :159 IU/L (530141 U/L), Total proteins :7.3 (6-8g/dl),albumin:3.9 (3.5-5.2 g/dl), Blood Urea :16 (12.6-42.6 mg/dl), serum creatinine:0.8 (0.5-1.4 mg/dl), serum sodium:144 (135-155meq/L), Serum potassium: 5.1 (3.5-5.5 meq/L) and Random blood sugar : 293 (70-140 mg/dl), Heamoglobin:10.6 (12-15 gm/dl%), PCV : 32(36-46%), RBC:3.95 (3.8-4.8mill/cumm), WBC: 12000 cells/cumm (4000-11000 cells/cumm), neutrophils :75 (40-80%), Lymphocytes: 20(20-40%), Eosinophils: 1(1-6%), Monocytes: 4(2-10%),MCV:80.9 (83-101 fl), MCH: 26.8(27-32 pg), MCHC: 33.1 (31.5-34.5 g/dl), platelets : 3.41(1.50-4.50 lakhs/cumm), pus cells (1-2), Epithelial cells :3-4) and bleeding time: 1:28(1-3 min) and clotting time :3:49(3-7 min). On examination: bilateral asymmetrical, multiple fluid-filled bullae which are sense-binding with upper limb, trunk, and lower limbs approximately 40%BSA involved.

Bullae: a clear fluid filled smallest 1*1cm and largest 3*4 cm on an erythematous base, which does not burst easily associated with erosions present onthe left lower limb with peripheral crushing.

Oral mucosa: bluish discoloration of tongue present

Genitals: desquamation present

Scalp: Anagen effusiveness present

Nikolskysign : negative

Bullae spread sign:positive(round granulated)

Based on the above subjective and objective evidence, the diagnosis was made as Drug-Induced Bullous pemphigoid (Using chemotherapy-induced pemphigoid)

Treatment is given to this patient: T.omnacortil 20mg OD, T. pan 40mg OD, T.Bilastine 20mg OD, T. Dazit 5mg OD, Momate cream OD over lesions, Inj. Actrapid TID, T. Telma 40mg OD, T. Azithral OD and Inj. Avil 2CC Stat.

III. DISCUSSION

Bullous Pemphigoid is an Autoimmune disorder that leads to the blistering type of the disease. Before the patient was diagnosed with calcium cervix the patient used this chemotherapy (Inj. Paclitaxel and Inj. Carboplatin). In cancer condition our own cells will grow continuously and destroy our own body cells. In this patient uses paclitaxel these paclitaxel targets microtubules. Higher doses of paclitaxel cause the stoppage in the mitotic phase of the G2/, phase, and at the Low dose of paclitaxel leads to the destruction of the cell to grow continuously stoppage happens at G0 and G1/S Phase. So, this paclitaxel shows the photosensitivity reactions leads to fluid-filled rashes in Bullous Pemphigoid. Carboplatin activates the platinum complex. It interacted with linkage of DNA molecules and inhibits the DNA synthesis. Carboplatin having to reduce the electrolytes and minerals in our blood leads to skin itching and rashes over the skin.

For early detection based on the symptoms patient approaches very early so the treatment started and patient reached to recovery. Bullous pemphigoid is the blister formation. The patient develops blister formation in various regions of the body like hands, back, stomach and neck.

The treatment was given to reduce itching and redness sensation in various regions and the patient reacted positively to the treatment and all doses and drugs were given at appropriate timing for fast recovery of the patient.

IV. CONCLUSION:

Bullous Pemphigoid is an autoimmune disorder in which our own body cells will react negatively. Caused by chemotherapy received by the patient leads to the development of itchy, rashes, and blisters in various regions of the body. The treatment is given to reduce redness, itchiness and blisters on the body. The patient responded to the treatment and the outcome leads to a positive.

V. ACKNOWLEDGEMENT

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CONFLICT OF INTEREST

The authors declare that there is no conflict of interest.

ABBREVIATIONS

NSAID: Non-SteroidalAnti Inflammatory Drugs; **BPAG:** Bullous Pemphigoid Antigen, **Ig G:** Immunoglobin G; **RBC:** Red Blood Cells; **WBC:** White Blood Cells; **MCV:** Mean Corpuscular Volume; **MCH:** Mean Corpuscular Hemoglobin; **MCHC:** Mean Corpuscular Hemoglobin Concentration; **BID:** Twice a day; **TID:** Thrice a day.

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